HIGH AND RISING MORTALITY RATES
AMONG WORKING-AGE ADULTS

Kathleen Mullan Harris, Malay K. Majmundar, and Tara Becker, *Editors*

Committee on Rising Midlife Mortality Rates
and Socioeconomic Disparities

Committee on Population
Committee on National Statistics
Division of Behavioral and Social Sciences and Education

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Preface and Acknowledgments

The year 2017 marked the third year in a row that life expectancy in the United States had fallen, the longest sustained decline in life expectancy in a century (since the influenza pandemic of 1918–1919). Already ranked relatively low in life expectancy (26th) in 2015 among the 35 countries that make up the Organisation for Economic Co-operation and Development, the United States would lose even more ground in its global position in national health and well-being. Research had already uncovered some troubling mortality trends and disparities before 2015 and was focused on the search for explanations. Early findings pointed to rising mortality rates among middle-age White adults, although the trends soon revealed that younger adults were also at risk, as were other racial/ethnic groups, such that premature mortality in the working ages of 25–64 was becoming more common in the United States than in prior years and in comparison with its international peers.

In this context, in 2018 the National Institute on Aging and the Robert Wood Johnson Foundation requested that the National Academies of Sciences, Engineering, and Medicine undertake a study on high and rising rates of midlife mortality and concomitant widening social differentials. In response to that request, the National Academies appointed the Committee on Rising Midlife Mortality Rates and Socioeconomic Disparities (under the standing Committee on Population) to carry out the task. Twelve scholars representing a broad array of disciplines—including demography, economics, epidemiology, medicine, public health, sociology,

and biostatistics—were included on the committee, which met six times in person over a 2-year period.

This report presents a considerable body of information. The committee decided to conduct its own analysis of the trends in working-age mortality by age, sex, race and ethnicity, and geography using the most up-to-date data to establish its members' collective understanding of the main drivers of the rising trend and disparities in working-age mortality in the United States. Findings from the committee's analysis are presented in Part I of this report. The committee then conducted a comprehensive review of the research on rising working-age mortality to evaluate evidence on what had changed in American society to bring about the change in mortality rates and how the patterns of change differed for population subgroups. Findings on the explanations for the rise in working-age mortality are presented in Part II of the report. The committee's work was arduous because the amount of data was massive; the problem was complex; and the unique trends by age, sex, race and ethnicity, and geography multiplied that complexity. In this report, the committee attempts to communicate these complexities while at the same time identifying the main drivers of high and rising working-age mortality based on current research and their implications for the future. The committee was also very deliberate and conscientious in its recommendations for further data collection, research, and policy.

This study would not have been possible without the contributions of many people. Special thanks go to the members of the study committee, who dedicated extensive time, thought, and energy to this task. Committee members conducted extensive analysis in generating Part I of the report, often enlisting their students and research assistants to help. Julene Cooney (Syracuse University), Nick Graetz (University of Pennsylvania), Jermaine Heath (Harvard Medical School), Fitore Hyseni (Syracuse University), Jeron Impreso (Harvard Medical School), Sammer Marzouk (Harvard University), Harrison Mintz (Harvard Medical School), Rohan Shah (Harvard Medical School), and Yue Sun (Syracuse University) assisted the committee in analyzing mortality trends and assessing selected research literatures. Thanks are also due to Anna Mueller (Indiana University Bloomington), who provided valuable guidance to the committee on suicide deaths.

The committee received useful information and insights from presentations by outside experts at open sessions of committee meetings. We thank Erika Blacksher (University of Washington), Anne Case (Princeton University), Andrew J. Cherlin (Johns Hopkins University), Kathleen Frydl, Carol Graham (Brookings Institution), Christopher Ruhm (University of Virginia), and Jennifer Silva (Indiana University Bloomington).

Several staff members of the National Academies made significant contributions to the report. Ellie Grimes and Mary Ghitelman made sure

that the committee meetings ran smoothly, assisted in preparing the manuscript, and otherwise provided key administrative and logistical support; Kirsten Sampson Snyder managed the report review process; Yvonne Wise managed the report production process; and Brian Harris-Kojetin, director of the Committee on National Statistics, provided valuable guidance and oversight. We also thank Rona Briere for skillful editing.

This Consensus Study Report was reviewed in draft form by individuals chosen for their diverse perspectives and technical expertise. The purpose of this independent review is to provide candid and critical comments that will assist the National Academies in making each published report as sound as possible and to ensure that it meets the institutional standards for quality, objectivity, evidence, and responsiveness to the study charge. The review comments and draft manuscript remain confidential to protect the integrity of the deliberative process.

We thank the following individuals for their review of this report: Andrew J. Cherlin, Department of Sociology, Johns Hopkins University; Sandro Galea, School of Public Health, Boston University; Mark D. Hayward, Population Research Center, University of Texas at Austin; Ichiro Kawachi, Department of Social and Behavioral Sciences, Harvard School of Public Health; Peter Muennig, Mailman School of Public Health, Columbia University; Samuel H. Preston, Population Studies Center, University of Pennsylvania; Albert L. Siu, Mount Sinai Medical Center; and Frank A. Sloan, Economics Department and Center for Health Policy, Law and Management, Duke University.

Although the reviewers listed above provided many constructive comments and suggestions, they were not asked to endorse the conclusions or recommendations of this report, nor did they see the final draft before its release. The review of this report was overseen by Bradford Gray, Urban Institute, and Eileen Crimmins, University of Southern California. They were responsible for making certain that an independent examination of this report was carried out in accordance with the standards of the National Academies and that all review comments were carefully considered. Responsibility for the final content rests entirely with the authoring committee and the National Academies.

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Committee on Rising Midlife Mortality
Rates and Socioeconomic Disparities

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The past century has witnessed remarkable advances in life expectancy in the United States and throughout the world. In 2010, however, progress in life expectancy in the United States began to stall, despite continuing to increase in other high-income countries. Alarmingly, U.S. life expectancy fell between 2014 and 2015 and continued to decline through 2017, the longest sustained decline in life expectancy in a century (since the influenza pandemic of 1918–1919). The recent decline in U.S. life expectancy appears to have been the product of two trends: (1) an increase in mortality among middle-age and younger adults, defined as those ages 25–64 years (i.e., "working age"), which began in the 1990s for several specific causes of death (e.g., drug- and alcohol-related causes and suicide); and (2) a slowing of declines in working-age mortality due to other causes of death (mainly cardiovascular diseases) after 2010.

STUDY PURPOSE, APPROACH, AND SCOPE

Explaining why mortality has been rising among working-age adults is not straightforward. Mortality is the final result of both acute events and cumulative, long-term processes involving the interaction of social, behavioral, economic, environmental, and biological factors that develop and unfold over the life course. Establishing the complex relationships among these explanatory factors poses methodological challenges that are

[1]This summary does not include reference citations. Citations supporting the content herein are provided in the body of the report.

complicated by issues of data availability and quality, as well as measurement. Studies in the early 2010s indicated that midlife mortality was rising primarily among middle-age non-Hispanic White (White) adults, particularly women, those with a high school degree or less, and those living in rural areas. More recent research, however, has documented this trend among most racial/ethnic groups and in most areas of the country. In some ways, these trends have exacerbated long-standing mortality divides, such as disparities between those of high and low socioeconomic status and metropolitan versus nonmetropolitan populations. These mortality disparities are important in their own right, signaling the effects of deepening inequality across many facets of American life, but they also help elucidate the underlying processes that have generated the recent increases in working-age mortality.

A full understanding of the rise in working-age mortality requires focusing beyond the factors that are most proximate to specific causes of death (e.g., behavior, psychological factors, health care utilization). One must also look upstream to the macrostructural factors (e.g., public policies, macroeconomic trends, social and economic inequality, technology) that may affect the health of Americans in multiple ways and through multiple pathways that flow through local community contexts and intersect with individuals' lives. To that end, the National Institute on Aging and the Robert Wood Johnson Foundation asked the National Academies of Sciences, Engineering, and Medicine to conduct a consensus study to identify the key drivers of increasing midlife mortality and concomitant widening social differentials; elucidate modifiable risk factors that could alleviate poor health in midlife, as well as widening health inequalities; identify key knowledge gaps and make recommendations for future research and data collection to fill those gaps; and explore potential policy implications. In response, the National Academies convened the Committee on High and Rising Midlife Mortality Rates and Socioeconomic Disparities in 2019 to carry out this work.

The committee's first task was to review what is known about trends in working-age mortality in order to establish the contributions of specific causes of death to overall changes in mortality over time, and about disparities in mortality rates by age, sex, race and ethnicity, socioeconomic status, and geography. This review revealed wide variations across studies in the tabulation and presentation of causes of death, as well as in the age and racial/ethnic groups and time periods included in the analyses, which made differences across studies difficult to interpret. Comparisons across studies were also complicated by methodological differences, such as whether mortality rates were adjusted to account for changes in the age distribution of the population over time. To supplement this literature review and obtain a comprehensive and consistent understanding of mortality trends and

disparities, the committee performed its own independent analyses of working-age mortality over the 1990–2017 period based on restricted-access National Vital Statistics death certificate data files. These analyses examined overall trends in mortality and disparities in those trends by age group, sex, race and ethnicity, and geography (metro status, states).

Although the committee also recognized the need to stratify data by socioeconomic status, the data required to do so were found to be lacking. The committee had concerns about the quality of data on educational attainment in vital statistics records and the total absence of data on income in death certificate records. Accordingly, the committee did not examine disparities in working-age mortality by socioeconomic status in its analyses, but instead relied on a thorough review of previous research on mortality differentials by education and income.

TRENDS IN WORKING-AGE MORTALITY

Although recently identified, increasing mortality among U.S. working-age adults is not new. The committee's analyses confirmed that a long-term trend of stagnation and reversal of declining mortality rates that initially was limited to younger White women and men (ages 25–44) living outside of large central metropolitan areas (seen in women in the 1990s and men in the 2000s) subsequently spread to encompass most racial/ethnic groups and most geographic areas of the country. As a result, by the most recent period of the committee's analysis (2012–2017), mortality rates were either flat or increasing among most working-age populations. Although this increase began among Whites, Blacks consistently experienced much higher mortality. These long-standing racial disparities are discussed in greater detail below. Over the 1990–2017 period, disparities in mortality between large central metropolitan and less-populated areas widened (to the detriment of the latter), and geographic disparities became more pronounced. Mortality rates increased across several regions and states, particularly among younger working-age adults, and most glaringly in central Appalachia, New England, the central United States, and parts of the Southwest and Mountain West. Mortality increases among working-age (particularly younger) women were more widespread across the country, while increases among men were more geographically concentrated.

Regarding socioeconomic status, the committee's literature review revealed that a large number of studies using different data sources, measures of socioeconomic status, and analytic methods have convincingly documented a substantial widening of disparities in mortality by socioeconomic status among U.S. working-age Whites, particularly women, since the 1990s. Although fewer studies have examined socioeconomic disparities in working-age mortality among non-White populations, those that have

done so show a stable but persistent gap in mortality among Black adults that favors those of higher socioeconomic status.

As noted earlier, the recent increase in all-cause mortality among U.S. working-age adults is due to both rising mortality from several causes of death and slowing progress in lowering mortality from other leading causes of death. The committee identified three categories of causes of death that were the predominant drivers of trends in working-age mortality over the period: (1) drug poisoning and alcohol-induced causes, a category that also includes mortality due to mental and behavioral disorders, most of which are drug- or alcohol-related; (2) suicide; and (3) cardiometabolic diseases. The first two of these categories comprise causes of death for which mortality increased, while the third encompasses some conditions (e.g., hypertensive disease) for which mortality increased and others (e.g., ischemic heart disease) for which the pace of declining mortality slowed. This report examines most closely the explanations for mortality trends in these three categories of causes of death to identify the key drivers of and modifiable factors in the recent overall rise in working-age mortality.

Other causes of death also contributed to increasing mortality during the 1990–2017 period, although few of these causes made meaningful individual contributions to the alarming mortality increases seen since 2010. Taken together, however, their combined impact was not trivial and therefore should not be ignored, and potential explanations for these increases are addressed briefly in this report. Four of these causes in particular—liver cancer, nervous system diseases, homicides, and transport injuries—merit further attention because their contributions to rising mortality were not negligible. Although the committee of necessity focused its attention on the major drivers of increasing working-age mortality—drugs, alcohol, suicides, and cardiometabolic diseases—it encourages the research community to seek explanations for increases in working-age mortality due to other causes of death identified in this report. Of particular importance is identifying factors contributing to the large and persistent racial/ethnic disparities in working-age mortality trends, such as the rise in homicides and transport injuries among Black and Hispanic men or the delayed reductions in mortality from HIV/AIDS among older Blacks.

Drug- and Alcohol-Related Deaths

Collectively, drugs and alcohol were responsible for more than 1.3 million deaths—approximately 8 percent—among the working-age (ages 25–64 years) population between 1990 and 2017. These substance-related deaths were major contributors to the rise in working-age mortality, and they are not abating. Drug poisoning deaths have been rising for more than three decades and represent the single largest contributor to the rise

in mortality rates among U.S. working-age adults (except among older Hispanic adults ages 45–64). While drug-related mortality rates increased in every U.S. state over the study period, the increases were most pronounced in Appalachia, New England, and the industrial Midwest. The largest increases occurred among Whites (particularly men) and older Black men. Among working-age Whites, increases in mortality due to drug poisoning were largest among younger men (ages 25–44), those with a high school degree or less, and those living in large metropolitan areas. In contrast, among working-age Blacks, mortality increased most among older men (ages 55–64) in large central metropolitan areas. While the committee's review of the literature showed that there was no difference in drug poisoning mortality by educational attainment among Blacks, increasing mortality due to drug poisoning among lower-educated individuals was responsible for most of the growing mortality gap by educational attainment among working-age Whites. The rate of alcohol-induced deaths also increased among Whites during the entire study period, and although the rate of such deaths declined among Blacks and Hispanics throughout the 1990s and early 2000s, that trend ceased in the late 2000s, and alcohol-induced deaths increased in these populations in the 2010s.

The rise in drug poisoning deaths is well studied, and research has yielded several plausible explanations for the trend. Although explanations for rising alcohol-related mortality have been less thoroughly investigated, similar supply-and-demand factors underlie both sets of trends. Sparked by the introduction of OxyContin® in 1996, the country's drug overdose crisis represents a "perfect storm" resulting from the flooding of the market with highly addictive yet deadly prescription and illicit drugs and the underlying and growing demand for and vulnerability to substances that might possibly bring relief, albeit temporary, from physical and/or mental pain.

On the supply side, weak governmental oversight combined with actions in the 1990s and 2000s by the pharmaceutical industry (manufacturers, distributors, pharmacies), pain control advocacy groups (often funded by pharmaceutical companies), and physicians to fuel a massive increase in opioid prescribing, which was followed by a rise in prescription opioid misuse, addiction, and overdose. While opioid-based pain relievers have an appropriate role in treating pain among those suffering from cancer, pharmaceutical companies expanded production and marketing of these drugs throughout the 1990s and 2000s for large populations with noncancer pain and made misleading claims about the drugs' safety and lack of addictiveness. With encouragement from pain control advocacy groups and pharmaceutical companies, physicians and other health care providers significantly increased their opioid prescribing.

Collectively, these forces resulted in saturation of the United States with 76 billion opioid pills just between 2006 and 2012; no other country

approached this level of opioid prescribing. Throughout the 2000s, as policy makers, state health officials, and physicians began to recognize the dangers of opioids and prescribing of the drugs subsequently declined, prescription opioids became less available and grew more expensive. This transition created a "thick market" for heroin, lowering its price and introducing it to a new clientele. Thus emerged a second wave of the opioid crisis, in which the consolidation of the heroin supply chain in Mexico and the much more widespread availability of heroin in the United States led to an increase in heroin overdose deaths. The third wave of the crisis began in the early 2010s, when drug suppliers and dealers began mixing heroin and other drugs (e.g., cocaine) with fentanyl and fentanyl derivatives that were inexpensive but extremely potent opioids with high overdose risk. Fentanyl deaths surpassed those involving heroin in August 2016 and continued to climb even as overall overdose mortality began to level off.

Demand-related explanations for the three-decade surge in drug overdose deaths focus on why certain subpopulations and geographic areas may have been more vulnerable than others to the increased availability of opioids and other drugs. Physical pain may have been one such contributor. Millions of Americans experience chronic pain, and some evidence suggests that the prevalence of physical pain may have increased in recent decades. Although, as noted earlier, adults with non-cancer-related pain were infrequently prescribed opioids before the mid-1990s, high and possibly increasing levels of physical pain may over time have expanded demand for OxyContin and similar products that flooded the market after 1996.

Mental illnesses and substance use disorders are closely intertwined, as are adverse childhood experiences and adult substance use. However, ongoing population surveys addressing adult mental illness and existing research on temporal trends in the prevalence of adverse childhood experiences provide insufficient evidence regarding their potential contribution to the increase in drug overdoses.

"Despair" has been among the more controversial potential explanations for the rise in substance-related deaths. Despair signifies hopelessness, which is a feature of depression and other affective disorders but is not itself a formal mental health diagnosis. The notion that the past 30-year rise in working-age mortality is partly due to increasing psychological distress among working-age adults with lower education is appealing because it accords with long-term economic, family, and social changes that have increased disconnection from the people, activities, and institutions that provide support and give people purpose and meaning. While the committee could find no causal studies on the effects of changing psychological health on U.S. substance use and mortality trends, there is ample empirical support for the hypothesis that psychological health has been worsening among U.S.

working-age adults and that proxies for despair (e.g., hopelessness, sadness, worry) are connected to substance use. Ultimately, measuring despair and determining causality remain key challenges for understanding the true role of despair in contemporary mortality trends. Qualitative research, which provides compelling evidence for the role of increasing despair in substance use and overdose, can offer insights for demographers, economists, and epidemiologists who seek to develop and test strong measures of despair.

Protracted long-term structural changes in and stressors to the U.S. economy, along with acute "shocks" (e.g., the Great Recession of 2007), have had differential effects on population subgroups and geographic areas. These long-term macroeconomic trends may partly explain the geographic patterns observed in drug poisoning mortality, such as the disproportionate impact in rural areas and the industrial Midwest, which have suffered losses in manufacturing and mining jobs. The distribution of industry and occupations is uneven across the country: macro-level economic trends and policy changes have brought prosperity to some places (e.g., high-tech and finance-dominant urban hubs) and decimation to others (e.g., Appalachia, the Rust Belt). The decline and transformation of industries that once provided "good" jobs for adults with only a high school education have eroded the social fabric and economic vitality of communities that once depended on those industries. The decline in opportunities among adults with less than a college education has been especially devastating and may have contributed to the rise in drug poisoning and alcohol-related deaths in this population.

The relationship between economic conditions and mortality, however, is complex, and the evidence is mixed on the causal effect of relatively short-term economic changes on substance-related mortality. Quasi-experimental studies suggest that mortality rates increase in response to specific economic forces—such as job loss, plant closings, and disruption from foreign trade—but there is less evidence about broader economic forces, such as technological advances that replace workers and general economic trends related to productivity. Other studies have found that opioid supply availability has a larger effect on drug-related mortality relative to changes in specific economic factors. The best interpretation of current knowledge about the broader relationship between economic well-being and mortality suggests that economic hardship is associated with higher mortality, especially in the context of widespread availability of potent and life-threatening medications. However, the overall impact of the direct economic shocks that have been examined (i.e., short-term changes in economic circumstances) appears to be modest.

Suicide

Suicide was among the 10 leading causes of death at ages 25–64 in 2015–2017 when life expectancy was declining, and it accounted for 569,099 deaths in the working-age population during the 1990–2017 study period. Historically, suicide mortality has been substantially higher among men than women and among Whites than Blacks and Hispanics. The same was true between 1990 and 2017, when significant increases in suicide rates occurred *mainly* for Whites, and White men in particular. At the beginning of the period, suicide rates differed little by metropolitan status among White adults, but over time, the rates increased more slowly in large central metropolitan areas than in less-populated areas, widening an urban–rural gap in suicide mortality. In line with this differential, suicide rates are higher in Western states, especially those with large rural populations.

Potential causes of rising suicide rates among Whites are complex, involving multiple factors that operate independently and interactively across societal, community, and individual levels. Unfortunately, a paucity of research examines differences by race and ethnicity, estimates causal impacts, or attempts to explain *change* in suicide mortality. As a result, understanding of why suicide rates have increased among working-age Whites during this period is mainly inferential.

Research on suicide trends tends to focus on explanations in four general areas: economic factors; social engagement, religious participation, and social support; access to lethal means; and mental and physical health. Some of the stronger evidence is related to the role of economic conditions. Periods of economic downturn, wage stagnation, weak safety nets, and increasing foreclosure rates are associated with rising suicide mortality in national and state-level studies. In addition, deteriorating economic conditions among those without a college degree may be an important factor explaining rising suicide mortality among Whites, especially White men. There is evidence that social support from embeddedness in formal institutions (e.g., church, school), community organizations, or stable interpersonal relationships that buffer the risks of self-harm has declined in recent decades, and that this decline has been more prominent among lower-educated Whites.

Although access to lethal means of suicide is associated with suicide, *changes* in access do not appear to provide an explanation for *rising* suicide mortality among Whites. Suicide mortality by firearms rose over the period 1990–2017, but its contribution to the rise in overall suicide mortality declined as suicides by other means increased more rapidly. While there is evidence that firearm-related suicide rates are higher in states with looser gun regulations and greater gun ownership and are higher in

nonmetropolitan than in large metropolitan areas, the proportion of all suicide deaths related to firearms declined from 1990 to 2017.

Important predisposing factors related to suicide mortality are life-course traumas and stressors, especially those that occur early in life, such as adverse childhood experiences, and mental illness. Not surprisingly, those with a history of mental illness have a much higher risk of suicide, and Whites tend to report more history with mental illness relative to other racial/ethnic groups. Comorbidities related to physical illnesses, disabilities, and drug and alcohol use also contribute to levels of mental illness and pain, all of which increase the risk of suicide.

Cardiometabolic Diseases

Deaths due to cardiometabolic diseases encompass the following cause-of-death categories: *endocrine, nutritional, and metabolic (ENM) diseases* (e.g., thyroid conditions, diabetes, hyperlipidemia, obesity); *hypertensive heart disease* (e.g., heart disease caused by prolonged exposure to high blood pressure); and *ischemic heart disease and other diseases of the circulatory system* (e.g., reduced blood supply to the heart, including atherosclerosis and coronary heart disease, stroke, and other cardiovascular conditions). Collectively, cardiometabolic diseases were responsible for more than 4.8 million deaths among the U.S. working-age (25–64 years) population between 1990 and 2017. ENM diseases accounted for 703,247 deaths, hypertensive heart disease for 360,309 deaths, and ischemic heart disease and other diseases of the circulatory system for the largest share of 3,782,186 deaths.

The contribution of cardiometabolic mortality to the recent rise in working-age mortality is complex and involves several countervailing trends. Death rates due to ENM diseases and hypertensive heart disease generally increased during 1990–2017, especially starting in 2010, and while there have been significant long-term reductions in mortality from ischemic heart disease and other diseases of the circulatory system, much of that progress appears to have stalled since 2010. The combination of these trends operated to increase all-cause mortality after 2010 because the slowdown in mortality declines from ischemic heart disease and other circulatory diseases no longer offset the rise in mortality from ENM diseases and hypertensive heart disease.

Within the working-age population, certain subgroups experienced greater relative increases in mortality due to ENM diseases and hypertensive heart disease over the study period and slower declines in mortality from ischemic heart disease and other circulatory diseases starting in 2010. These subgroups include younger adults (ages 25–44) of all racial/ethnic groups, White men and women, Black men (in the recent decade), and

those living in rural areas. These troubling changes in cardiometabolic mortality were most pronounced in the South and outside of large central metropolitan areas. Large central metropolitan areas and the Northeast generally experienced the most favorable trends in cardiometabolic disease mortality. As a result, the gap in mortality by metropolitan status grew over time, particularly among White working-age adults.

The literature provides three potential explanations for the trends in cardiometabolic mortality: the obesity epidemic; diminishing returns of medical advances; and social, economic, and cultural changes. The increased prevalence of obesity and its lagged cardiometabolic consequences are the most important. Substantial evidence shows that obesity increases the risks of hypertension, stroke, coronary heart disease, and diabetes, driving up death rates due to ENM diseases and hypertensive heart disease and slowing declines in mortality due to ischemic heart disease and other circulatory diseases. Obesity rates began to rise in the early 1980s and remain high today as a period-based phenomenon that has affected children and adults of all ages. But its cardiometabolic consequences have occurred in a cohort fashion. More recent cohorts—those born in the 1970s, 1980s, and 1990s—have been exposed for their entire lives to "obesogenic environments," defined as the conditions in which people live that encourage sedentary lifestyles and unhealthy diets and discourage or prevent people from adopting and maintaining healthier behaviors. These cohorts have been more affected by the obesity phenomenon because of their earlier life exposure and longer durations at risk relative to prior cohorts.

While the proximate causes of obesity involve health behaviors (diet, physical activity) that produce an imbalance between calories consumed and expended, obesogenic environments are a contributing factor. Substantial evidence documents how physical environments have become increasingly obesogenic—from urban landscapes more conducive to automobiles than pedestrians to the proliferation of fast food restaurants that encourage the consumption of inexpensive, calorie-dense foods. However, further research is needed to disentangle the complex pathways by which changing environments have led to the rise in the prevalence of obesity that, in turn, has fueled the changing trends in cardiometabolic mortality.

Medical advances in drug development and prevention, treatment, and control of chronic diseases, together with major reductions in tobacco use, played a large role in producing the long-term decline in mortality due to ischemic heart disease and other circulatory diseases that took place from 1970 to 2010. Progress may have stalled after 2010 because medical advances reached a point of diminishing returns. Medical advances also may be having less impact because their benefits are being offset by the lagged cardiometabolic consequences of rising obesity that are now affecting rates of diabetes, hypertension, and cardiovascular disease and because

many people who would benefit from cardiovascular treatments, especially those at greatest risk, face barriers to accessing services and adhering to treatment.

The social, economic, and cultural changes that have occurred over the past 50 years represent a natural progression that all advanced societies around the world have experienced. Those changes have increased the pace and efficiency of work and social interactions, but have also necessitated greater education, training, and technological skills to keep up with the faster pace of life, workplace demands, and dwindling opportunities for social mobility. Especially in the United States, these shifts have marginalized those without the necessary education and job skills, limiting not only their socioeconomic status and ability to live in healthy environments but also their access to health care, thereby increasing the daily stresses of life. Chronic stress can itself take a biological and emotional toll, disrupting and damaging endocrine, metabolic, and cardiovascular systems and increasing mortality risks. Yet while research has established links between chronic stress and cardiovascular disease, direct evidence that long-term social, economic, and cultural changes played a *causal* role in recent changes in cardiometabolic mortality is lacking.

RACIAL/ETHNIC, SOCIOECONOMIC, AND GEOGRAPHIC DISPARITIES

The committee's analysis and review of research revealed large, and in some cases widening, racial/ethnic, socioeconomic, and geographic disparities in working-age mortality. While the explanations for these disparities are often specific to certain causes of death, the committee identified common underlying themes that affected population subgroups at different time periods or in different contexts.

The first of these themes is the role of adverse economic trends (e.g., stagnant wages, collapse of job sectors, unemployment) that affected certain geographic areas and population subgroups more so than others. The loss of manufacturing and mining jobs in the industrial Midwest and Appalachia in the 1970s led to a long-term economic decline, often concentrated among the largely White families and communities in these areas. Declining economic conditions tend to weaken societal institutions, community resources, family bonds, social networks, and access to health care—all of which could help explain disparities in working-age mortality according to race and ethnicity, socioeconomic status, and geography.

A second theme is socioeconomic inequality, which could help explain the pace and timing of rising 21st century working-age mortality, as well as the long-standing racial/ethnic disparities in mortality that have persisted throughout U.S. history. Unequal access to societal opportunities and

resources to climb the social ladder create gradients in health and explain mortality disparities both across and within social groups, including racial/ethnic disparities. As a result of the legacy and persistence of structural racism in the United States, Blacks and other minority groups have experienced long-standing socioeconomic inequalities that have compromised their health and produced much higher mortality rates in these groups relative to Whites, a pattern borne out in the data reviewed by the committee. Nonetheless, with the growing importance of education within U.S. society and the need for academic credentials to obtain well-paying technical and professional jobs, socioeconomic inequality has also deepened among Whites, widening socioeconomic disparities in White mortality.

A third theme that emerges in explaining the trends and disparities in working-age mortality is vulnerability, which mediates the degree to which adverse economic conditions and socioeconomic inequality make particular groups more susceptible than others to morbidity and mortality risks. As a result of educational, job, and housing discrimination, for example, Blacks tend to work and live in segregated and often disadvantaged neighborhoods, increasing their exposure to obesogenic, unsafe, and low-resource environments that limit access to medical and behavioral health services and increase mortality risks. Such vulnerability plays a prominent role in today's drug overdose crisis, described earlier as a "perfect storm" in which the flooding of the market with highly addictive and deadly drugs occurred as the population was growing more vulnerable to emotional and physical pain, heightening demand for these products. Declining economic conditions, socioeconomic inequality, and vulnerability are themes that help in understanding how the different and changing social, economic, and geographic contexts of population subgroups may explain recent trends in working-age mortality.

IMPLICATIONS FOR RESEARCH AND POLICY

From a historical perspective, the rise in U.S. working-age mortality and recent resulting declines in life expectancy are relatively new phenomena. As this report documents, because the rise in working-age mortality was specific to certain causes of death but with varying patterns by age, sex, race and ethnicity, socioeconomic status, and geography, existing research into these complex and multilayered patterns is sparse, and research attempting to better understand the explanations for these changing patterns is nascent. Much remains to be learned, therefore, and the committee proposes numerous research efforts to generate better evidence that can serve as a basis for evaluating and refining salient policies. These recommendations span multiple levels and modes of analysis (individual, institutional, societal, and cross-national; quantitative and qualitative); address a variety of disparities

(socioeconomic, racial/ethnic, geographic); encompass a range of causes of death and related factors (drug poisoning; alcohol-related deaths; suicide; cardiometabolic diseases; mental illness; obesity; adverse childhood experiences; psychosocial indicators, such as stress, despair, hopelessness, coping, and resilience; long-term economic changes; social factors, such as family structure, community support, and religiosity); and propose numerous improvements to the data infrastructure that supports this research.

The committee also grappled with how the evidence detailed in this report suggests the need for policy changes with the potential to curb the increase and/or narrow disparities in rates of working-age mortality in the coming years. The committee stresses the immense challenge of predicting policy impacts in this area of science. Studies of mortality trends and patterns, especially at the national level, rely almost exclusively on observational data and federal statistics. As a result, causal evidence in this area is limited, and controlled experiments are difficult if not infeasible. Moreover, as discussed throughout the report, the key hypothesized influences on patterns and trends in working-age mortality are numerous and operate concomitantly at multiple levels. Many of the proposed drivers operate across the life course and/or across decades—in either period or cohort fashion—to influence current patterns and trends. This report therefore focuses on an exceptionally complex set of patterns, trends, and explanations for which clear or simple solutions are lacking.

Nonetheless, despite this complexity and the necessary reliance on observational and administrative data, the committee emphasizes the urgency of policy action in the face of a population health crisis that is claiming the lives of people in the prime of their lives (a crisis that has been exacerbated by the COVID-19 pandemic). Like the phenomena driving the crisis, policy responses need to be multilevel, focusing not only on the immediate causes of these deaths, such as drugs and obesity, but also on the upstream "causes of the causes," such as living conditions that increase the vulnerability of communities, families, and individuals to premature mortality. The committee accordingly offers policy recommendations regarding obesity prevention programs, interventions to target the substance use and overdose crisis at multiple levels on both the supply and demand sides, and the expansion of Medicaid under the Affordable Care Act. The committee also presents broader policy conclusions regarding the need to balance the rights of the food industry, advertisers, grocers, and restaurants to enjoy free market competition against the public health imperative to limit the promotion and consumption of foods and beverages that contribute to obesity; the need to revitalize the communities hit hardest by the overdose crisis by addressing the larger economic and social strains and dislocations that made those communities vulnerable in the first place; and the importance of dismantling structural racism and discriminatory policies of exclusion so as

to reduce and ultimately eliminate inequalities that continue to drive racial/ethnic disparities in health and mortality in the United States.

The United States is losing far too many lives far too early. While it is clear that the research base for understanding the nature of this complex problem needs to be strengthened, the rise in working-age mortality threatens the future of the nation's families, workforce, economy, and national security. It therefore constitutes a crisis that requires action even if the evidence is imperfect or only suggestive of causal effects and solutions. In taking this action, it will be essential to remain cognizant of the potential for unintended consequences—even for policies that are well intended and carefully designed to account for potential risks—and thus to continue to monitor outcomes, generate better evidence, and adjust policies over time.

PART I

1

Introduction

The past century has witnessed remarkable advances in life expectancy, and the United States has shared in that progress. In 2010, however, progress in life expectancy in the United States began to stall despite continuing to increase in other industrialized countries. Alarmingly, U.S. life expectancy fell between 2014 and 2015 (Arias and Xu, 2018) and continued to decrease in the two subsequent years (Arias and Xu, 2018, 2019; Arias, Xu, and Kochanek, 2019).[1] This 3-year period of declining life expectancy represented the longest sustained decline in the United States in a century (since the influenza pandemic of 1918–1919).

The stalling and subsequent decline in life expectancy during the 2010s appears to have been the product of an increase in mortality among middle-age and younger adults, defined as those ages 25–64 ("working age"), which began in the early 2010s. Between 2010 and 2017, the age-adjusted all-cause mortality in this age group increased by 6.0 percent (from 328.5 deaths to 348.2 deaths per 100,000 population) (Centers for Disease Control and Prevention [CDC], 2020b). By contrast, infant mortality decreased by 9.0 percent, and child mortality remained unchanged. Mortality among older adults (ages 65 and over) also continued to decrease over this period (CDC, 2020b).

Explaining why mortality has increased among working-age adults is not straightforward. Mortality is the end result of both acute events and

[1]U.S. life expectancy increased slightly in 2018 (Xu et al., 2020) and 2019 (Andrasfay and Goldman, 2021) ahead of the COVID-19 pandemic of 2020, which is expected to reduce life expectancy once again.

cumulative, long-term processes involving the interaction of biological, behavioral, social, economic, and environmental factors that develop and unfold over the life course. Establishing the complex relationships among these explanatory factors poses methodological challenges that are complicated by issues of data quality and measurement. Research initially indicated that the phenomenon of rising working-age mortality was occurring primarily among middle-age non-Hispanic Whites (Whites) (Case and Deaton, 2015), particularly women (Gelman and Auerbach, 2016) with a high school degree or less (Case and Deaton, 2015, 2017) and in rural America (Erwin, 2017). In fact, mortality appears to have begun rising in the 1990s among both White and non-Hispanic Black (Black) women with a high school degree or less (Montez et al., 2011); however, research using more recent data suggests that the trend now involves men and most racial/ethnic groups (Curtin and Arias, 2019; Woolf et al., 2018) and has expanded beyond rural America to more populated areas (Elo et al., 2019).

In some ways, these trends have exacerbated long-standing disparities in mortality, such as those between individuals of high and low socioeconomic status (Chetty, Hendren, and Katz, 2016; Cutler, Meara, and Richards-Shubik, 2011; Meara, Richards, and Cutler, 2008; Sasson, 2016) and between urban and rural areas (Monnat, 2020b; Singh and Siahpush, 2014; Vierboom, Preston, and Hendi, 2019). These trends also reflect stagnation in and even reversal of the steady progress made over the previous decades in reducing racial disparities in mortality faced by Black adults (Curtin and Arias, 2019). These effects on disparities in mortality are important in their own right, signaling the effects of deepening inequality across many facets of American life, but also because they help elucidate the underlying processes that may have generated the recent increases in working-age mortality.

Initial studies examining the specific underlying cause or causes of death responsible for increases in mortality focused predominantly on older working-age adults, those ages 45–54. These studies highlighted increases in fatal drug overdoses as a primary factor in rising mortality and found that these increases were driven largely by the less educated, particularly Whites with a high school education or less, for whom mortality had risen sharply since 1999 (Case and Deaton, 2015, 2017). However, this research also pointed to increases in mortality due to alcoholic liver disease and suicide among middle-age adults.

The striking rise in deaths from these three causes—drug poisoning, alcohol-related disease, and suicides—led some researchers to label these causes of death collectively as "deaths of despair" (Case and Deaton, 2017). However, more recent research (Dwyer-Lindgren et al., 2017; Geronimus et al., 2019) shows that mortality rates among working-age adults also increased within a broader age range than 45–54 and for a broad spectrum

of diseases involving multiple body systems (e.g., circulatory, digestive, pulmonary, neurologic, endocrine, and cardiovascular). Neither drugs nor "despair" could fully explain so diverse a phenomenon.

The decline in U.S. life expectancy received national news coverage (e.g., Bernstein, 2016, 2018; Rogers, 2016), as did research identifying drugs, alcohol, and suicide as the causes of death driving the decline (e.g., Achenbach and Keating, 2017; Bernstein and Achenbach, 2015). Despite research implicating a broad range of causes of death in rising mortality rates, media reports focused on the opioid epidemic and its impact on specific demographic groups and communities (e.g., Egan, 2018; Robertson and Trent, 2018). These reports were often set in rural or Rust Belt communities and featured stories of largely White families. The accounts often depicted how residents were affected by the job losses and economic instability resulting from the collapse of manufacturing, mining, and other industries that once provided secure and living-wage jobs to those without a college degree. While this narrative was reflective of early trends in drug overdoses due to prescription opioids, which began to increase in the 1990s primarily among working-age Whites with less than a college degree (Alexander, Kiang, and Barbieri, 2018; Ho, 2017), the rise in drug poisoning was not limited to this population. Although communities in Appalachia, New England, New Mexico, and Utah began experiencing rapid increases in opioid-related mortality in the 1990s (Case and Deaton, 2017; Rigg, Monnat, and Chavez, 2018), these increases were geographically heterogeneous, affecting both metropolitan and nonmetropolitan areas in regionally specific ways (Peters et al., 2020; Rigg, Monnat, and Chavez, 2018). Moreover, although opioid overdoses began noticeably increasing earlier among Whites, these increases were also experienced by Blacks, as well as by American Indian and Alaska Native (AI/AN) populations (Alexander, Kiang, and Barbieri, 2018; Tipps, Buzzard, and McDougall, 2018). Because of the popular focus on predominantly working-class Whites, however, the experiences of racial/ethnic minorities during the overdose epidemic have gone largely ignored.

Moreover, although the media focus on communities marked by growing opioid-related mortality has highlighted the important role of the opioid epidemic in increasing mortality among working-age adults, it has also obscured the broader range of causes of death that have contributed to these mortality increases (Geronimus et al., 2019) and the range of populations affected (Curtin and Arias, 2019; Elo et al., 2019; Woolf and Schoomaker, 2019). The affected age groups also are broader than first reported. Studies initially focused on middle-age adults (ages 45–54), but the data show that mortality rates also increased significantly among younger working-age adults (ages 25–44) between 2010 and 2017 (Curtin and Arias, 2019). Among younger adults, cause-specific mortality, particularly for drug

poisoning, has often increased at a faster pace compared with middle-age adults, and suicide rates in young people have increased as well. Indeed, the phenomenon may be extending into adolescence: the data suggest a recent increase in mortality among those ages 15–24, driven by drug overdoses and suicide (Ali et al., 2019). At the same time that mortality rates among White adults began increasing, the rates among middle-age and younger AI/AN adults were also increasing—at even higher rates (Tipps, Buzzard, and McDougall, 2018; Woolf et al., 2018). And between 2010 and 2017, all-cause mortality rates among working-age Blacks and Hispanics increased as well (Curtin and Arias, 2019).

INDIVIDUAL AND SOCIETAL IMPLICATIONS OF RISING MORTALITY AMONG WORKING-AGE ADULTS

The significance of these ominous trends for the country cannot be overstated. Rising mortality among working-age adults is a population health crisis. The premature death of tens of thousands of Americans in the prime of their lives has profound ripple effects on the well-being of families and the social fabric of communities for generations to come. It affects an age span that encompasses the American workforce, impacting the productivity and competitiveness of U.S. businesses, the economy, and national defense. The health conditions driving these mortality increases are adding to escalating health care costs, posing an unsustainable burden not only on government payers (e.g., Medicaid and Medicare) but also on employers, threatening the U.S. position in the global marketplace (Chernew, Hirth, and Cutler, 2009; Kaiser Family Foundation, 2019; Nunn, Parsons, and Shambaugh, 2020; Office of the Assistant Secretary for Planning and Evaluation [ASPE], 2005; Sood, Ghosh, and Escarce, 2009). And military and national security experts also have raised concerns about these trends (Congressional Budget Office, 2017; Keith, 2011; Riley, 2010).

Rising health care costs are an ongoing concern in America that has been exacerbated by mortality increases and their underlying causes. Per capita national health care expenditures increased more than 220 percent between 2000 and 2017.[2] While the direct effect of mortality on health care spending likely stems from the high cost of end-of-life care, the increasing burden of health care spending is associated mainly with chronic conditions that develop earlier in life among working-age younger adults and lead to earlier mortality (Einav et al., 2018; French et al., 2017). The Substance Abuse and Mental Health Services Administration (SAMHSA) projects that by 2020, spending on substance abuse and mental health treatment

[2]According to the Centers for Medicare & Medicaid Services' National Health Expenditures (NHE) data, per capita NHE totaled $4,855 in 2000 and $10,739 in 2017.

will total $280.5 billion, a 63 percent increase relative to 2009 (Substance Abuse and Mental Health Services Administration [SAMHSA], 2014). And an analysis by the Urban Institute found that Medicaid spending on three medications used to treat opioid use disorder and overdose increased 136 percent between 2011 and 2016 (Clemans-Cope, Epstein, and Kenney, 2017). Health care spending for these health issues falls on state budgets, largely through Medicaid, and crowds out other important priorities, such as education (Chernew, Hirth, and Cutler, 2009; Medicaid and CHIP Payment and Access Commission [MACPAC], 2016; Rosewicz, Theal, and Ascanio, 2020).

Higher health care spending associated with the causes of working-age mortality also affects the commercial insurance market. Because health insurance pools risk across the population, the costs associated with early death and associated illness for individuals with employer-sponsored coverage are borne by their coworkers. For example, higher insurance premiums reduce wage growth for all workers (Burtless and Milusheva, 2013; Clemens and Cutler, 2014; Kolstad and Kowalski, 2016). Similarly, as premiums rise, employers reduce the generosity of coverage, and the added financial risk is imposed on all workers (Anand, 2017; Kaiser Family Foundation, 2019). In some cases, higher premiums induce employers to drop insurance coverage altogether.

Deteriorating health among working-age Americans will extend the disability rolls to segments of the adult population that heretofore have been disability free (Chen and Sloan, 2015). Transitions to disability status result in declines in earnings, income, and food and housing consumption, especially impacting those with less education (Cutler, Meara, and Richards-Shubik, 2011; Meyer and Mok, 2019; Prinz et al., 2018). Increases in longer-term opioid prescribing may only exacerbate this trend; some estimates using workers' compensation claims data suggest that such prescriptions roughly triple the duration of temporary disability benefits (Savych, Neumark, and Lea, 2019). Similarly, individual-level survey data on drug use and increases in its intensity are significantly correlated with criminal justice system involvement (Winkelman, Chang, and Binswanger, 2018). Even before the height of the opioid epidemic, cost estimates based on these data suggested that nonmedical use of prescription opioids was responsible for $8.2 billion in criminal justice costs (Hansen et al., 2011). Incarceration also hinders treatment for and recovery from drug use disorders. One study found that only 4.6 percent of justice-referred people received treatment for opioid use, compared with 40.9 percent of other clients (Krawczyk et al., 2017), with significant impacts on individuals and families (see, e.g., National Research Council [NRC], 2014).

Families are especially affected by rising mortality among working-age adults and the conditions leading to those deaths. Children who experience

the suicide of a family member, for example, are four times more likely to commit suicide in their lifetime (Burke et al., 2010). Given that suicide attempts are often related to underlying mental health problems, other studies suggest that children of parents dealing with mental health issues may develop their own issues with depression and suicide ideation later in life (King et al., 2010; Lunde et al., 2018). Similar effects are likely among individuals connected to someone who commits suicide but is outside of the immediate family (Cerel et al., 2016). Beyond suicide, more than one-third of children in foster care were found to have been placed because of parental substance use (Adoption and Foster Care Analysis and Reporting System [AFCARS], 2017), and every 15 minutes a baby is born with exposure to opioids during pregnancy (National Institute on Drug Abuse [NIDA], 2019).

Finally, the economic effects of rising mortality among working-age adults are both direct and indirect. The nation loses valuable workers, their output, and taxes to premature mortality. Moreover, impaired health leads to lower productivity (Currie and Madrian, 1999; Prinz et al., 2018) and increased hospitalizations, which in turn increase unemployment and reduce earnings (Dobkin et al., 2018), and these consequences are amplified for those who lack insurance or lose insurance from their employer. The economic costs due to opioid use are especially illustrative. In 2006, 79 percent of the total cost of nonmedical use of prescription opioids ($53.4 billion) was attributable to lost productivity, primarily through unemployment and subemployment due to opioid abuse (Hansen et al., 2011). Recent data show similar impacts of opioid use and pain management on labor force participation (Harris et al., 2019a), with 40 percent of men ages 25–54 reporting that pain prevented them from working a full-time job (Krueger, 2017). Economic costs will be seen for other causes of premature mortality as well. Indirect costs of lost productivity due to cardiovascular disease are estimated to increase by 55 percent over the coming decades, from $237 billion in 2015 to $368 billion[3] in 2035 (Khavjou, Phelps, and Leib, 2016), as metabolic disorders begin to affect the previously healthy years of working-age adults.

Most troubling is the pervasive nature of the rise in mortality, now affecting a broad range of working-age adults in all racial/ethnic groups and in multiple geographic areas of the United States. The rise in mortality could threaten the well-being of individuals, families, health care, criminal justice, and economic systems and the social fabric of communities. Perhaps the greatest threat is posed by not knowing the underlying causes of these mortality trends. As a result, today's children, whose parents are dying in middle age or as younger adults, may themselves face the insidious causes

[3]Measured in 2015 dollars.

of this phenomenon, carrying the crisis of rising mortality forward into the next generation. Explaining these trends is therefore of paramount importance.

SEARCHING FOR EXPLANATIONS

The increase in mortality rates across multiple conditions among those of working age—but not the very old—precludes easy explanations. A full understanding requires focusing not just on those factors that are most proximate to the specific causes of death (e.g., behavior, psychological factors, health care utilization), but also upstream on the macrostructural causes (e.g., public policies, macroeconomic trends, social and economic inequality) that may affect the health of Americans in multiple ways and through multiple pathways that flow through local community contexts and intersect with the lives of individuals.

Failures in the health care system are conspicuous in the United States and may affect outcomes for multiple conditions. Unlike other industrialized countries, the United States lacks universal access to health care (owing to a lack of health insurance and shortages of providers and facilities in many communities),[4] relying on a fragmented care delivery system characterized by large disparities in the quality of care and the incidence of medical errors. A growing number of studies have demonstrated that acquisition of health insurance coverage can lead to significant reductions in mortality (Borgschulte and Vogler, 2019; Goldin, Lurie, and McCubbin, 2021; Miller et al., 2019). However, studies suggest that only 10–20 percent of premature deaths are attributable to health care (Kaplan and Milstein, 2019) and that only 13 percent of the improvement in life expectancy since 1990 can be attributed to improvements in medical care, excluding new pharmaceuticals (Buxbaum et al., 2020). Deficiencies in health care are therefore unlikely to fully explain the trend.

Health behaviors, such as tobacco use and sedentary lifestyles, are major influences on health and mortality (Choi et al., 2020; Ford et al., 2012; Franzon et al., 2015; Tencza, Stokes, and Preston, 2014). Moreover, caloric intake in the United States exceeds that in other industrialized countries (Bleich et al., 2008). These unhealthy behaviors may explain an increase in tobacco- or obesity-related deaths, and substance use (problem

[4]For example, the United States has fewer physicians, hospitals, and acute care beds per capita than comparable countries (Kamal et al., 2020). The Health Resources and Services Administration maintains a list of U.S. areas that have been designated as Health Provider Shortage Areas and Medically Underserved Areas. See more information here: https://bhw.hrsa.gov/workforce-shortage-areas/shortage-designation#hpsas.

drinking, misuse of analgesics) is a proximate cause of the rise in deaths from alcohol and drugs.

However, extensive research documents that health behaviors (and access to health care) are shaped by people's environments. Healthy eating, for example, requires access to affordable and nutritious foods, while exercise requires a built environment that is conducive to regular physical activity (MacDonald et al., 2010). Whereas other countries have regulations against advertising junk food to children, food companies in the United States are allowed to market highly processed and high-sugar foods to children aggressively, add sugar to everyday products, and lobby policy makers to subsidize sugar production and promote consumption of high-sugar foods (Freudenberg, 2014). Socioeconomic status, such as educational attainment, income, and wealth, in turn determines the ability to live in a healthy neighborhood, maintain healthy behaviors, and afford health care (Pampel, Krueger, and Denney, 2010). In addition, the social environment, from families to communities, affects health: family and social networks can enhance health and promote resilience (Yang et al., 2016), or they can harm health by exposing people to social isolation, poor health behaviors, dysfunction, trauma, violence, racism, or other forms of discrimination.

Individuals' status and behaviors and the families and communities in which they live are shaped by macrostructural factors, including public policies (from federal legislation to local zoning laws); economic trends (e.g., decline of manufacturing, rising income inequality); shifting demographics (e.g., immigration, rural outmigration); and business decisions, from how much to pay workers to the marketing of inexpensive calorie-dense foods or highly addictive prescription opioids. These macrostructural factors, in turn, reflect social and cultural values, such as the proper role and size of government, attitudes about social inequality, beliefs in individualism, structural racism, and other forces that shape public policies and spending priorities.

A full understanding of why mortality rates at working ages have increased since the 1990s therefore requires careful study that gives adequate attention to these complex multilevel influences and how they have changed over time, and to the evidence that such changes may have produced the epidemiologic trends occurring today. It is necessary as well to take a life-course perspective—understanding that health evolves over one's lifespan in a cumulative and interactive fashion, such that events occurring in one life stage shape developmental and health trajectories in subsequent stages. A growing body of research documents the importance of the first 1,000 days of life in shaping growth and development (Crump and Howell, 2019). Exposure to adverse childhood events predicts not only the diseases of young people but also the probability of developing chronic diseases in old age by setting trajectories of risk or resilience in the subsequent life

stages of adolescence, young adulthood, and midlife (Brown et al., 2009; Felitti et al., 1998). This means that the study of rising mortality rates among working-age adults requires consideration of what occurred among the cohorts of Americans born 25–64 years before the trend began and comparing their experiences with those of prior cohorts over time.

CHARGE TO THE STUDY COMMITTEE

In this context, in 2018, the National Institute on Aging and the Robert Wood Johnson Foundation asked the National Academies of Sciences, Engineering, and Medicine to carry out a consensus study on rising rates of midlife mortality and associated socioeconomic disparities. The specific charge to the National Academies is as follows:

> The Committee on Population (CPOP) and the Committee on National Statistics (CNSTAT) of the National Academies of Sciences, Engineering, and Medicine will undertake a study that will: identify the key drivers of increasing mid-life mortality and concomitant widening social differentials; identify modifiable risk factors that might alleviate poor health in mid-life and widening health inequalities; identify key knowledge gaps and make recommendations for future research and data collection; and explore potential policy implications.

To conduct this study, the National Academies appointed the Committee on Rising Midlife Mortality Rates and Socioeconomic Disparities. The committee's membership included 12 prominent scholars representing a broad range of disciplines—demography, economics, epidemiology, medicine, public health, sociology, and statistics. The committee met six times in person over a 2-year period to complete the study and produce this report.

IMPACT OF THE COVID-19 PANDEMIC ON WORKING-AGE MORTALITY

In 2020, as the committee worked to finalize this report, the COVID-19 pandemic began its spread across the world. Between March 2020 and the start of 2021—when this report went into production—COVID-19 grew from a rare disease in the United States to a leading cause of death (CDC, 2021; Woolf, Chapman, and Lee, 2021). Although all countries were affected by the pandemic, no country suffered as many deaths as did the United States (Bilinski and Emanuel, 2020). As of January 19, 2021, more than 400,000 COVID-19–related deaths, about one-fifth of the global total, had occurred in the United States (Johns Hopkins University, 2021), and the daily death toll from COVID-19 had surpassed the toll for heart disease

and cancer, the nation's two leading causes of death (Woolf, Chapman, and Lee, 2021).

Moreover, studies of preliminary vital statistics data indicated that excess deaths—the number of deaths beyond what would have been expected without the COVID-19 pandemic—increased by approximately 20 percent in the United States during this period. However, COVID-19 accounted for only about two-thirds of these excess deaths (Rossen et al., 2020; Weinberger et al., 2020; Woolf et al., 2020). Especially during surges, the nation and individual states experienced sharp increases in deaths from other causes, such as heart disease, Alzheimer's disease, and diabetes (Woolf et al., 2020). Researchers were unable to determine in real time the extent to which excess deaths overall as well as the observed increases in non-COVID-19 deaths occurred among infected patients whose death certificates omitted mention of the virus or uninfected patients who experienced death caused indirectly by disruptions resulting from the pandemic (e.g., inability to access acute emergency services). These deaths also could include deaths due to causes, such as drug overdoses, exacerbated by the pandemic. There is increasing evidence that the pandemic led to increased consumption of alcohol and drugs, including benzodiazepines (antianxiety medications), and in December 2020, the CDC issued a health advisory about the increasing risk of deaths due to these agents during the pandemic (CDC, Emergency Preparedness and Response, Health Advisory, December 17, 2020). While the reasons for this association are not entirely clear, it has been suggested that traumatic stress related to the pandemic in many populations may be partly to blame (Taylor et al., 2021). The pandemic may also have disrupted access to and delivery of substance use and mental health treatment services (Herrera, 2021). And just as the pandemic disrupted the supply chain for many household and food products, it also disrupted the drug supply chain. The result was a decline in the international production and trafficking of heroin in the United States and subsequent increases in fentanyl-adulterated drugs, which pose a greater risk of overdose. Drug shortages have also resulted in increases in the use of injecting drugs and sharing needles, increasing the risk of spreading bloodborne diseases (United Nations Office on Drugs and Crime [UNODC], 2020).

Thus, COVID-19 has reinforced and exacerbated existing mortality disparities within the United States, as well as between the United States and its peer countries. The CDC reported that adults ages 25–44 experienced the largest percentage increases in excess deaths during the pandemic (as of October 2020) (Rossen et al., 2020). COVID-19 mortality was also higher among males, who have long experienced higher mortality than females (Faust et al., 2020). Provisional mortality data from the CDC (2020c) indicated that 54 percent of all COVID-19 deaths had occurred among males. The disparity was even greater for working-age adults, among whom males

represented 65 percent of all COVID-19 deaths reported in the United States.

COVID-19 has disproportionately targeted Hispanic and non-White Americans, particularly Blacks (Ford, Reber, and Reeves, 2020; National Center for Health Statistics [NCHS], 2021). Provisional mortality data (NCHS, 2020) show wide racial disparities in the impact of the disease. Although present and significant at older ages, these disparities are larger among younger Americans, who show the widest racial/ethnic disparities (Ford, Reber, and Reeves, 2020). These disparities are far greater than overall disparities in all-cause mortality among working-age adults. As of early January 9, 2021, 35 percent of COVID-19 deaths among working-age adults had occurred among Hispanic adults and 24 percent among Black adults, even though these groups had experienced only 13 percent and 19 percent of all non-COVID-19 deaths, respectively. In contrast, only 34 percent of COVID-19 deaths among working-age adults had occurred among Whites, who had experienced 63 percent of non-COVID-19–related deaths. Working-age non-Hispanic Asian, AI/AN, and Native Hawaiian and Pacific Islander adults had also experienced disproportionately high mortality from COVID-19 (NCHS, 2021).

These racial disparities are undoubtedly due at least in part to the geographic concentration of the initial waves of the pandemic in large, racially diverse central metropolitan areas, such as New York City, San Francisco, Seattle, and Los Angeles. However, by January 2021, both the COVID-19 case and death rates were higher overall in nonmetropolitan than in metropolitan counties (Ullrich and Mueller, 2021). As the virus spread into less-populated and less racially diverse areas of the country, however, large racial disparities persisted in these areas as well (Cheng, Sun, and Monnat, 2020; Ford, Reber, and Reeves, 2020). Between March 2 and July 25, 2020, for example, average daily increases in COVID-19 death rates were 70 percent higher in rural counties with the largest-percentage Black populations (i.e., those in the top 25th percentile) (Cheng, Sun, and Monnat, 2020). In many ways, the COVID-19 pandemic was ominously poised to exploit and exacerbate existing social and economic inequalities. Because of race-based occupational and residential segregation, Hispanic and Black versus White adults were more likely to be employed in such "essential" occupations as health care, farm work, and food service (Bureau of Labor Statistics, 2019); to live in multigenerational households (Cohen and Casper, 2002); to experience socioeconomic disadvantage; and to lack full access to health care. Moreover, although individual-level data on socioeconomic disparities in COVID-19 mortality are not readily available, individuals of lower socioeconomic status are in general more likely to have comorbidities (Cutler, Meara, and Richards-Shubik, 2011; Pampel, Krueger, and Denney, 2010) that are associated with more severe COVID-19 illness (CDC, 2020a), and

the ability to adhere to social distancing guidelines in daily life depends on financial resources (Weiss and Paasche-Orlow, 2020).

COVID-19, then, will likely magnify the effects of already increasing mortality rates among many subgroups of working-age Americans. These mortality increases are likely to undo years of progress in reducing racial disparities in mortality and to magnify socioeconomic disparities in life expectancy. The important role played by COVID-19 in increasing mortality in the United States in 2020 can already be seen in the percentage of all working-age deaths between January 1, 2020 and January 9, 2021 that were due to COVID-19. Among working-age Whites, 4.3 percent of all deaths involved COVID-19. In contrast, the race-specific contribution of COVID-19 was 10.0 percent among Black adults, 21.4 percent among Hispanic adults, 14.2 percent among AI/AN adults, 13.0 percent among Asian adults, and 16.1 percent among Native Hawaiian and other Pacific Islander adults (NCHS, 2021).

These disparities may continue to shape mortality patterns for decades to come. The burden of COVID-19 is much broader than its direct impact on mortality. As of mid-September 2020, more than 6 million people in the United States had tested positive for the virus (Johns Hopkins University, 2020). Although many people who tested positive experienced no symptoms during their period of infection, growing evidence suggests that many of those who survive COVID-19 infection develop long-term symptoms and health complications that may last far into the future, even when they initially experienced only mild symptoms or were asymptomatic (Pérez-Bermejo et al., 2020; Tenforde et al., 2020). Experience with the COVID-19 pandemic underscores the importance of understanding the underlying causes of current mortality trends, including how racial, socioeconomic, and geographic disparities are consistently produced and reinforced as such novel diseases are introduced into the population.

STUDY METHODS AND LIMITATIONS OF THE EVIDENCE BASE

This report describes and explains the trends in high and rising mortality among working-age adults in the United States and documents the demographic, socioeconomic, and geographic disparities in those trends. To carry out these analyses, the committee developed a multilevel conceptual framework to guide identification of the main drivers of the trends, from upstream macrostructural factors, to local environments in which people live and work, to downstream proximate individual-level factors. Using this framework, the committee reviewed the available evidence on how changes in these factors may have led to increased mortality among working-age adults and how such factors operate across different life stages from

childhood to midlife. This framework also guided the committee's identification of factors that can be modified through policy, education, or other initiatives to reduce mortality risks and disparities among working-age adults. Through a comprehensive review of the research evidence, the committee was able to identify knowledge gaps and offer recommendations for future research and data collection efforts.

As the committee began its initial task of reviewing previous research documenting the demographic, socioeconomic, and geographic disparities in mortality trends in the United States, it found extensive variation across studies that would have limited its ability to draw clear conclusions about the relative contributions of specific causes of death to changes in all-cause mortality within subpopulations. These studies varied in terms of which populations were included in the analyses, the time periods covered, and the tabulation and presentation of causes of death and demographic characteristics.

Moreover, although approval of prescription opioids occurred in the mid-1990s, the committee found that a number of studies evaluating the relative contribution of drug poisoning to overall changes in mortality began with data from 1999[5] (Case and Deaton, 2015; Woolf et al., 2018; Woolf and Schoomaker, 2019), thus missing the initial period of the drug overdose epidemic. In addition, some studies combined deaths from cardiometabolic diseases into a single broad category (Masters, Tilstra, and Simon, 2017), while others highlighted more specific causes within this category, such as heart disease (Case and Deaton, 2017); diabetes (Geronimus et al., 2019; Woolf and Schoomaker, 2019); endocrine, nutritional, and metabolic diseases (Woolf et al., 2018); or hypertensive heart disease (Woolf et al., 2018; Woolf and Schoomaker, 2019). Although these differences across studies indicate that the trends in cause-specific mortality were not uniform within this broad category, they also limit the capacity to compare results across studies.

Still another limitation of the evidence base is the significant differences across racial/ethnic groups in what is known about variations in mortality trends by sex, age, and geography: these variations are documented extensively for working-age Whites and working-age adults more generally, and less information is available for Hispanic and non-White populations. Thus, to clearly assess the relative contributions of different causes of death to trends in all-cause mortality and how these contributions differed by sex, age, and geography for different racial/ethnic groups, the committee decided it was necessary to perform independent analyses. Multiple committee members have extensive experience performing such analyses and,

[5]In 1999, cause of death on U.S. death certificates began to be classified using the International Classification of Diseases, 10th Revision (ICD-10).

through their existing research projects, had access to restricted mortality data files from the National Vital Statistics System that included detailed geographic information on decedent residence. Multiple committee members were involved in conducting the analyses, thus providing internal checks on the accuracy of the analyses. An exception occurred with respect to the examination of mortality rates by educational attainment. After considering the limitations of the information on educational attainment reported on death certificates, particularly during the 1990s, the committee decided to rely on a review of previous literature for estimates of mortality trends by education rather than produce its own estimates. For this reason, the explication in this report of differences in mortality trends by socioeconomic status is more limited than other aspects of the analysis.

ORGANIZATION OF THE REPORT

The remainder of this report is divided into three parts. Part I evaluates trends and disparities in mortality to identify the origins of the recent (since 2010) increases in mortality among working-age adults. This part of the report also evaluates the strengths and limitations of U.S. mortality data. Chapter 2 compares life expectancy and mortality rates in the United States with those in 16 high-income peer countries, beginning in the 1950s, to establish when the United States' relative mortality disadvantage first emerged and the important role played by working-age mortality in contributing to this disadvantage. Chapter 3 examines trends in all-cause mortality within the United States between 1990 and 2017 by sex, age, race and ethnicity, socioeconomic status, and geography to provide greater insight into where and among which populations these increases occurred. Chapter 4 focuses on identifying the specific causes of death that contributed to these mortality trends to illuminate which causes made the greatest contribution and how this differs by sex, age, race and ethnicity, and geography. The final chapter of Part I, Chapter 5, evaluates the quality and limitations of mortality data available in the United States and makes recommendations for improving data capacity for future research on mortality.

Having identified in Part I the causes of death that represent the key drivers of recent changes in all-cause working-age mortality, the report turns in Part II to the committee's evaluation of the quality of current research evidence supporting potential explanations for those changes. Chapter 6 provides an overview of the multilevel conceptual framework the committee applied in evaluating the key contributors to the recent mortality increases. Chapters 7–10 then examine the evidence supporting explanations for the trends in mortality for the key causes of death that drove the increases and provide recommendations for data improvements

and research. Chapters 7–9, respectively, focus on drug poisoning and alcohol-related deaths, suicide, and cardiometabolic diseases. Finally, Chapter 10 evaluates what is known about the broader economic factors that may have contributed to the recent mortality trends.

Part III of the report consists of Chapter 11, which recaps the policy and research implications presented in Part II and the rationale for each and offers new policy and research implications for themes that cut across all of the preceding chapters.

2

U.S. Mortality in an International Context

Life expectancy in the United States is lower than in many countries and has been for an extended period. In 2016, the United States ranked 34th among all countries on life expectancy (World Health Organization [WHO], 2018a) and 40th on "healthy" life expectancy (i.e., years lived without disease or disability) (WHO, 2018b). U.S. life expectancy ranks below that of nearly every other high-income country and below that of many middle-income countries (WHO, 2018a). This underperformance compared with other peer countries is a growing cause for concern and has motivated efforts to understand its contributing factors. The National Academies of Sciences, Engineering, and Medicine conducted two previous consensus studies related to this issue—one focused on U.S. mortality after age 50 (National Research Council [NRC], 2011), and another using a broader set of health metrics to compare health and longevity among Americans and populations in other high-income countries and examine mortality and life expectancy from the 1980s through 2008 Institute of Medicine and National Research Council [IOM and NRC], 2013).

This chapter extends those prior analyses to consider differences in life expectancy between the United States and peer countries over the period 1950–2016. The same 16 peer countries are considered here as in the 2013 report: Australia, Austria, Canada, Denmark, Finland, France, Germany, Italy, Japan, Norway, Portugal, Spain, Sweden, Switzerland, the Netherlands, and the United Kingdom. Trends in the mortality gaps between the United States and these other high-income countries are used to place recent U.S. mortality trends in a larger historical and international context. These comparisons also illustrate how high and rising working-age (ages 25–64)

mortality rates have shaped recent trends in U.S. life expectancy. The chapter presents five sets of international analyses presented in four sections, each intended to help illustrate the growing gap between mortality in the United States and other peer countries and to clarify the rationale for the committee's focus on mortality rates at working ages:

- The first section compares the countries on an overall measure of longevity—life expectancy at birth—spanning the period 1950–2016. This long-term comparison of the United States with other countries helps clarify when the United States began to diverge from its peers, and whether the current gap is consistent with historical trends or marks a new point of divergence.
- The second section focuses on the contributions of specific age groups to the gap in life expectancy between the United States and its peer countries, as well as on how age-specific mortality rates in the United States compare with those of the 16 peer countries. These two sets of analyses clarify the age groups that make the largest contribution to the widening gap in life expectancy and mortality between the United States and its peers and establish the important role played by mortality at working ages since 2010.
- The third section focuses on working-age mortality and examines how trends in age-standardized mortality rates in this age range differed between the United States and the 16 peer countries between 1950 and 2016 after accounting for differences in the age distributions of their working-age populations over time.
- The final section examines how age-standardized mortality rates for several key causes of death differ between the United States and its peers.

A detailed description of the data and analytic methods used to produce these results can be found in the annex at the end of this chapter.

Together, the findings from these analyses paint a bleak picture of U.S. mortality in terms of both its current international standing and historical trends. The current gaps between life expectancy in the United States and peer countries are the largest ever recorded. Moreover, the trends underlying these growing gaps suggest that they would have been likely to increase in the near future even before the effects of the COVID-19 pandemic were realized. The evidence indicates, moreover, that the United States is falling behind its peers as the result of higher mortality at nearly all ages. Although deaths at older ages (i.e., above 65) account disproportionately for the increasing gap in life expectancy between the United States and peer countries, differences in the risk of death are greatest at younger ages. Many causes of death responsible for the increasing U.S. disadvantage are chronic

diseases that are costly and burdensome, require long-term care, and will likely shape U.S. mortality trends for years to come. Deaths related to drug use at younger ages also contribute substantially to the worsening U.S. mortality disadvantage. As discussed in the final chapter of this report, the large and growing U.S. disadvantage in life expectancy and health will likely persist without national-level investments in disease prevention, changes in regulatory policies, and reductions in social and economic inequalities.

LIFE EXPECTANCY AT BIRTH IN THE UNITED STATES VERSUS PEER COUNTRIES

The gap in life expectancy between the United States and peer countries has been decades in the making, with evidence of a possible problem appearing as long ago as the 1950s (Figure 2-1).[1] By 2016, life expectancy for U.S. females (81.1 years) was 3.3 years lower than the average in the 16 peer countries (84.4 years), and life expectancy for U.S. males (76.2 years) was 3.7 years lower than the peer average (79.9 years). Although the gap in life expectancy between the United States and peer countries unfolded over a long period of slower growth in U.S. life expectancy spanning many decades (1950–2010), this trend accelerated rapidly after 2010 as life expectancy stagnated and then declined in the United States alone.

Although U.S. life expectancy increased from 1950 to 2010, it often did so at a slower pace than in the peer countries (Figure 2-1, upper panels). In the first decades of this period, the slower growth in life expectancy within the United States relative to the peer average was heavily influenced by the rapid growth in life expectancy that occurred within those peer countries that were recovering from the effects of World War II. However, U.S. life expectancy also remained below that of the remaining peer countries during this time. By 1970, life expectancy among U.S. males had fallen below the average in the peer countries, and life expectancy among U.S. females had lost the advantage it held during the 1950s. Although increases in U.S. life expectancy in the 1970s were comparable to (and sometimes exceeded) those in the peer countries, year-over-year increases in the United States began diminishing in the late 1970s and failed to keep pace during the 1980s, 1990s, and 2000s. This long-term trend of falling behind peer countries was more pronounced among U.S. females than among U.S. males.

[1]In the 1950s, the countries that had lower life expectancy than the United States for both males and females were Austria, Finland, France, Germany, Italy, Japan, Portugal, and Spain—all of which were recovering from the effects of World War II. U.S. life expectancy already had fallen below that of each of the remaining peer countries—Australia, Canada, Denmark, the Netherlands, Norway, Sweden, and Switzerland—among females and below all but Canada among males.

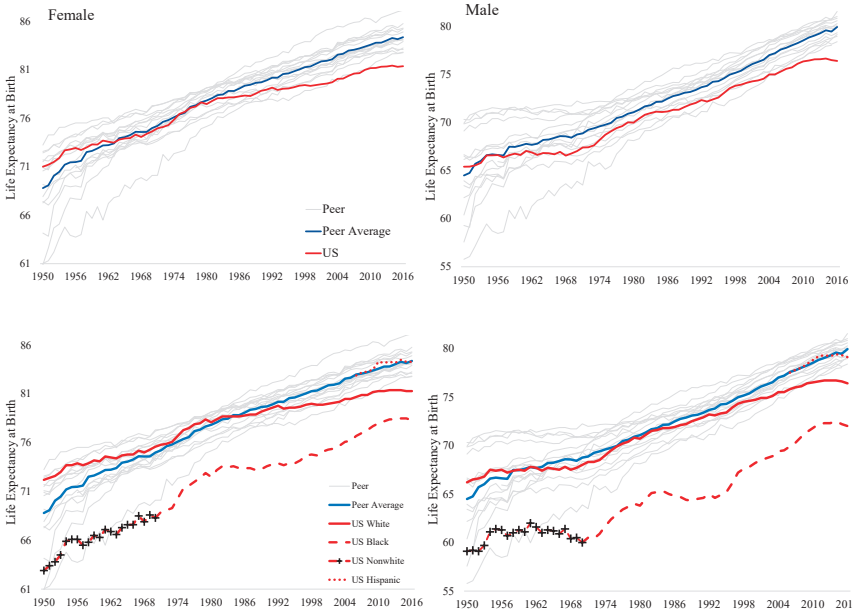


FIGURE 2-1 Female and male life expectancy at birth in the United States and peer countries, 1950–2016.

NOTE: Life expectancy at birth is depicted for females in the two lefthand panels and for males in the two righthand panels. In the top two panels, the red solid lines plot life expectancy at birth for the total U.S. population. In the bottom two panels, the red solid lines plot life expectancy at birth for the U.S. White population, while the red dashed lines plot life expectancy for U.S. non-White populations. In the period before 1970, the red dashed line with the black "+" symbol plots the combined life expectancy at birth for all U.S. non-White populations. Beginning in 1970, the red dashed line represents life expectancy at birth among the U.S. Black population only. The red dotted line that begins in 2006 plots life expectancy at birth for U.S. Hispanics. In all four panels, the gray lines plot the respective life expectancies for each peer country, and the blue solid lines show average female and male life expectancies across the 16 peer countries.

SOURCE: U.S. mortality data are drawn from National Vital Statistics Reports (see the annex at the end of this chapter for detail). Mortality data for the 16 peer countries are drawn from the Human Mortality Database (2019).

In the early-2010s, increases in U.S. life expectancy first stalled and then reversed, with declines occurring for both U.S. males and females between 2014 and 2016. These recent downward trends in U.S. life expectancy were especially pronounced among U.S. males. In contrast, life expectancies for both males and females in peer countries continued their upward trend.

The racial/ethnic diversity of the U.S. population distinguishes it from the populations of its 16 peer countries. Although mortality rates among the U.S. non-White population relative to the non-Hispanic White (White) population are disproportionately high because of a range of historical and current factors (e.g., slavery, immigration, racial discrimination [see Chapter 11]), there is little evidence that racial/ethnic diversity explains the U.S. life expectancy gap. For example, as shown in Figure 2-1 (lower panels), although life expectancy among the U.S. non-Hispanic Black (Black) population was far lower than that of the U.S. White population and of peer countries, life expectancy among the U.S. White population also failed to keep pace with that of peer countries. The stall and decline in U.S. life expectancy after 2010 occurred among the U.S. Black, White, and Hispanic male and female populations.

THE U.S. MORTALITY DISADVANTAGE BY AGE

International Differences in Life Expectancy Decomposed by Age

Which age group is most responsible for the decline in U.S. life expectancy? The gap in U.S. life expectancy was driven by mortality trends among adults roughly ages 18–60 (Figure 2-2). The gap began to take shape early; as early as 1970, the United States had a survival disadvantage compared with its peer countries in all age groups between 10 and 65. The U.S. disadvantage in life expectancy extended over time, gradually encompassing all age groups under 80. Although the United States had a survival advantage among infants and children under age 10 and among older adults (ages 65 and over) in 1970, by 2016 only the very oldest ages (over 80 among males, over 85 among females) retained an advantage in life expectancy over the peer country average.

The overall trends over time in the U.S. disadvantage in life expectancy relative to the peer country average were similar by sex, but the age patterns differed for males and females. Among females in 1970 (Figure 2-2, upper panel), the age-specific contribution to the U.S. life expectancy disadvantage among those ages 10 and over grew steadily with age, peaking at age 55 before reversing and becoming a sizable life expectancy advantage among females over age 65. In contrast, among males in 1970 (Figure 2-2, lower panel), the age-specific contribution to the U.S. disadvantage in life expectancy did not increase steadily after age 10; instead, it increased quickly

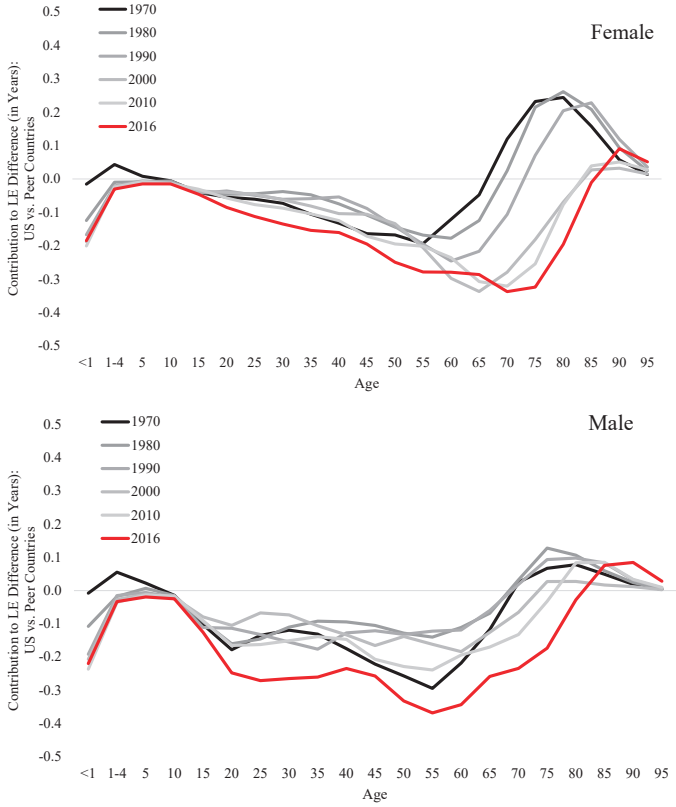


FIGURE 2-2 Contributions of age-specific mortality differences between the United States and peer countries to overall life expectancy (LE) differences.
NOTE: The figure plots the number of years of life expectancy contributed by each age group to the total gap in life expectancy between the United States and the average of the 16 peer countries. Positive values on the vertical axis indicate survival advantages for the United States (i.e., fewer deaths at these ages), whereas negative values indicate survival disadvantages for the United States. The top panel shows the results for females, while the bottom panel shows the results for males. The black and gray-scale lines plot the values for the period 1970–2010 in 10-year increments. The red line plots the values for 2016, the most recent year for which life expectancy data were available for all 16 peer countries. The data presented were estimated using Arriaga decomposition techniques (Arriaga, 1984; Auger et al., 2014). They show the specific contributions of ages <1 year, 1–4 years, and all 5-year age groups from 5–9 through 95–99 (deaths above age 100 do not contribute to the life expectancy differences between the United States and peer countries in all years).
SOURCE: U.S. mortality data are drawn from National Vital Statistics Reports (see the annex at the end of this chapter). Mortality data for the 16 peer countries are drawn from the Human Mortality Database (2019).

between ages 10 and 20 and then remained steady between ages 20 and 35, after which it again began to increase. As was the case among females, the age-specific contribution to the U.S. disadvantage in life expectancy among males was largest at age 55 and then reversed at older ages, so that U.S. males over age 65 held an advantage in life expectancy; however, this age-specific advantage was much smaller than that which occurred among older females.

The relative survival advantage among U.S. females at younger ages in 1970 dissipated over time, and by the 2000s and 2010s had become a growing disadvantage. The disadvantage in life expectancy in midlife that was already present in 1970 continued to increase over time and extended into older ages, erasing the older-age survival advantage by 2016. This steady trend across the years accounted for most of the increase in the U.S. female life expectancy disadvantage. Similar trends occurred among U.S. males, but the magnitude of the changes over time and the contributions of specific age groups to the U.S. life expectancy disadvantage differed from those of females. Between 2010 and 2016, a large younger-age disadvantage, a growing midlife disadvantage, and the loss of an older-age survival advantage emerged among males. Deaths among younger males made a larger contribution to the U.S. male life expectancy disadvantage than was the case among U.S. females. However, the extension of the midlife disadvantage to older ages and the loss of the older-age advantage compared with the peer countries was more pronounced among females.

Although the long-term trend of falling behind the peer countries occurred for both male and female life expectancy in the United States, this trend was more pronounced for female life expectancy between 1980 and 2010 (Figure 2-1). The decomposition of life expectancy by age group (Figure 2-2) shows that during the 1980s and 1990s, while the United States maintained a midlife disadvantage, the differences in female life expectancy between the United States and the peer countries increased, largely as the result of a widening survival disadvantage for U.S. females at older ages; only after 2000 did the widening midlife disadvantage contribute substantially to the widening female survival disadvantage of the United States relative to the peer countries. In contrast to earlier periods, the more recent stalling and decline in U.S. life expectancy since 2010 were more pronounced for U.S. males than for U.S. females, largely because of a widening survival disadvantage for U.S. males at working ages.

U.S. Mortality Relative to International Peers by Age

Comparing mortality rates in the United States and the peer countries highlights how long the United States has experienced higher mortality rates at midlife and younger ages, and this U.S. disadvantage has increased

substantially since 2000 (Figure 2-3). For all age groups between 15 and 65, the probabilities of death have been consistently higher in the United States than in the peer countries since at least the 1970s, and the ratio of mortality rates at these ages in the United States to those within the peer countries grew substantially from 2000 to 2010 and again from 2010 to 2016. After 2000, the relative risk of mortality in the United States among late adolescents and young adults (under age 40) increased more dramatically than among any other age group. In contrast, the relative mortality risk increased more steadily among older age groups over the decades.

The decomposition results shown in Figure 2-2 estimate the *absolute* contributions of each age group to differences in life expectancy between the United States and the peer countries. These data show that because mortality is much higher at older relative to younger ages, the reduction and then reversal over time of the U.S. mortality advantage at older ages had a larger effect on the gap in life expectancy between the United States and the peer countries relative to the consistent U.S. mortality disadvantage at midlife and younger ages over the period. The differences shown in Figure 2-3 are expressed in *relative* terms as ratios of mortality rates and highlight—more vividly than in Figure 2-2—the extremely high mortality risk faced by U.S. infants, children, adolescents, and young adults relative to their peers in other countries since 1990. The contrast is sobering. For example, whereas the risk of female infant death in the United States and the peer countries was comparable in the 1970s, by 2016 female infant mortality rates had increased to be about 75 percent higher in the United States. The risk of U.S. females dying at age 25 was about 40 percent higher in the United States than in the peer countries in 1970 but rose to more than 150 percent higher by 2016.

INTERNATIONAL TRENDS IN WORKING-AGE MORTALITY

The above discussion demonstrates that the United States has long faced higher mortality among working-age adults compared with the peer country average, but the gap has grown dramatically since 2000, contributing to widening differences in life expectancy between the United States and its peers. When the age-standardized probability of death between ages 25 and 64 in the United States is compared with that in each of the 16 peer countries instead of the peer country average (Figure 2-4), it is clear that the United States has consistently been among those countries with the highest working-age mortality since at least 1950, but also has increasingly pulled away from its peers over time. This is especially true for the most recent period, during which working-age mortality declined and became increasingly similar across the 16 peer countries while climbing in the United States.

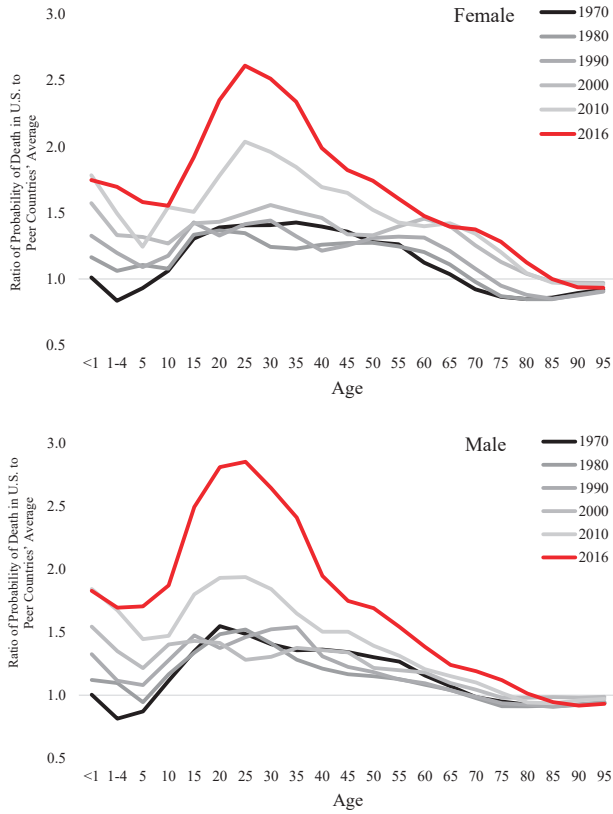


FIGURE 2-3 Ratio of the probability of death in the United States to the average probability of death in peer countries, by age group and year.
NOTE: The top panel shows the relative age-specific mortality risk for females, while the bottom panel shows the relative age-specific mortality risk for males. A value above 1.0 indicates that males and females in the United States experienced a higher probability of dying in that age group relative to the peer country average, while a value below 1.0 indicates that the United States experienced a lower risk of dying in that age group. The black and gray-scale lines plot the values for the period 1970–2010 in 10-year increments. The red line plots the values for 2016, the most recent year for which data on life expectancy were available for all 16 peer countries. The figure shows the specific contributions of ages <1 year, 1–4 years, and all 5-year age groups from 5–9 through 95–99 (deaths above age 100 do not contribute to the life expectancy differences between the United States and peer countries in all years).
SOURCE: U.S. mortality data are drawn from National Vital Statistics Reports (see the annex at the end of this chapter). Mortality data for the 16 peer countries are drawn from the Human Mortality Database (2019).

In 1950, although the United States had high mortality relative to most of its peers, several peer countries experienced higher working-age mortality. Over time, however, the United States increasingly became an outlier. By 2001, it consistently had higher female working-age mortality relative to any peer country, and by 2006, it had the highest male working-age mortality. Beginning in 2010, working-age mortality increased in the United States among both males and females while continuing to decline in peer countries. Whereas in 1950 there was considerable variation in working-age mortality among the 16 peer countries, their mortality rates converged over time, although there remained greater variability across countries in mortality among working-age males versus females.

The status of the United States as an outlier among its peers is highlighted when working-age mortality in the United States is measured in the number of standard deviations above the peer country average (Figure 2-4, bottom panels). Working-age mortality in the United States increased from less than 1 standard deviation above the peer country average in 1950 to more than 4 standard deviations above the average by 2016 among both males and females. Although working-age mortality had been increasing since at least the 1990s, both sexes experienced a steep increase in the most recent period that began in 2010, when working-age mortality started to increase in the United States alone.

Although the position of the United States relative to its peers eroded over time, these changes did not occur steadily over the period. Consistent with the episodic trends described in the previous sections, the United States fell behind the 16 peer countries during the 1950s and 1960s; then improved on both life expectancy and its relative standing in working-age mortality in the 1970s; but increasingly fell behind during the 1980s, 1990s, and 2000s. Finally, in the period following 2010, working-age mortality began to increase in the United States alone, dramatically intensifying the already poor U.S. standing. The results shown in Figure 2-4, which are limited to ages 25–64, show that these trends are not just being driven by a relative slowdown in declines (or by increases) in mortality at increasingly older ages, but are part of a broader pattern of U.S. disadvantage that extends throughout the life course and is increasingly concentrated in working-age adulthood.

U.S. MORTALITY BY CAUSE OF DEATH IN INTERNATIONAL PERSPECTIVE

To provide insight into the underlying causes of death that drive the higher mortality rates in the United States compared with the peer countries, this section compares cause-specific mortality rates—for the United States and peer country averages—for selected causes of death in 2000, 2008, and

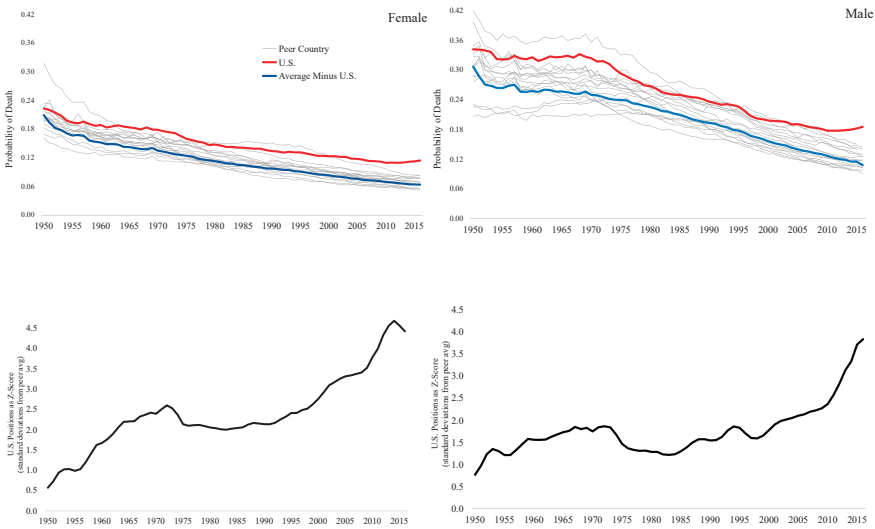


FIGURE 2-4 Age-adjusted probability of dying between ages 25 and 64 in the United States and in peer countries, 1950–2016 (top), and the relative standing of the United States and peer countries with respect to working-age mortality risk (bottom).

NOTE: The top two panels show the probability of dying between ages 25 and 64 in the United States and the 16 peer countries after adjusting these probabilities to account for differences in the age distributions across countries and over time. In these panels, the red line shows the mortality risk in the United States, the gray lines plot the respective mortality risk for each peer country, and the blue solid lines show the average mortality risk across the 16 peer countries. The bottom two panels show the relative positions of the United States and the 16 peer countries. The relative position of the United States is expressed as a z-score—specifically, the number of standard deviations above the peer country average by which the U.S. mortality risk falls.

SOURCE: U.S. mortality data are drawn from National Vital Statistics Reports (see the annex at the end of this chapter). Mortality data for the 16 peer countries are drawn from the Human Mortality Database (2019).

2015 (Table 2-1). These rates are computed for all ages in the populations but are age-adjusted to a standardized age distribution to ensure comparability over time and across countries. Although the United States enjoyed lower mortality from some causes of death during this period, this advantage was more than outweighed by the extensive range of causes for which the United States experienced higher mortality relative to its peers, and this disadvantage increased over time. By 2000, the United States already had higher mortality relative to its peers among both males and females across a wide range of causes of death, including circulatory diseases, endocrine diseases, lung cancer, respiratory diseases, and genitourinary diseases. Over the next 15 years, the mortality gap between the United States and its peers continued to grow for many of these causes of death, and further expanded to include higher mortality among both sexes from digestive diseases and accidental poisonings (including drug overdoses).

The four largest contributors to the growth in the U.S.–peer country mortality gap between 2000 and 2015 were circulatory diseases, diseases of the digestive system, accidental poisonings, and intentional self-harm (i.e., suicide). Mortality due to circulatory diseases, cancers involving organs other than the liver or lung, and respiratory diseases declined in both the United States and the peer countries, but the declines in the United States did not keep pace with those in the peer countries. The same was true for mortality due to diseases of the endocrine system among females. Among males, mortality due to diseases of the digestive system, mental health or alcohol use, and assault (i.e., homicide) stagnated over the period in the United States but continued to decline in the peer countries. Mortality due to accidental poisonings and liver cancer increased in both the United States and the peer countries, but the increases were greater in the United States, contributing to the growing U.S. mortality disadvantage. Finally, mortality for several causes of death—including intentional self-harm and diseases of the endocrine system among males and intentional self-harm, diseases of the digestive system, and mental health and alcohol use among females—increased in the United States but not in the peer countries.

TABLE 2-1 Age-Standardized Mortality Rates (deaths per 100,000 population) for All Causes of Death and Specific Causes of Death in the United States and Peer Countries, 2000, 2008, and 2015

Males	2000		2008		2015		Change in Difference
	U.S.	Peers	U.S.	Peers	U.S.	Peers	
All Causes	1210.4	1163.2	1028.5	975.4	988.4	877.5	63.7
Circulatory Diseases	476.1	446.5	338.1	322.8	310.7	261.0	20.1
Accidental Poisonings	6.5	3.4	13.5	4.7	20.0	4.7	12.2
Digestive Diseases	40.7	46.9	37.4	41.4	37.5	34.9	8.8
Intentional Self-Harm	18.5	21.5	19.8	18.3	21.9	16.7	8.2
Other Cancers	127.3	147.0	115.4	132.9	109.6	123.2	6.1
Respiratory Diseases	123.2	115.5	104.5	91.9	97.9	85.7	4.5
Endocrine Diseases	42.6	29.0	40.3	28.8	43.5	26.6	3.3
Liver Cancer	7.9	10.8	9.7	11.4	11.4	12.0	2.3
Mental Health/Alcohol Use	4.1	5.4	3.3	4.9	4.1	4.4	1.0
Assault	8.9	1.4	9.1	1.2	8.8	0.9	0.4
Genitourinary Diseases	26.7	18.3	25.9	19.2	25.2	17.2	–0.4
Prostate Cancer	35.6	43.5	26.0	36.4	22.0	31.3	–1.4
HIV/AIDS	7.8	2.6	5.0	1.6	3.0	0.9	–3.1
Colorectal Cancer	29.2	35.5	22.7	31.9	19.4	29.0	–3.3
Lung Cancer	89.9	72.2	74.5	64.7	58.1	55.8	–15.4

NOTES: Change in difference = $(\text{U.S.–peer})_{2015}$–$(\text{U.S.–peer})_{2000}$.
Orange highlights indicate U.S. rate-peer rate >3.
Green highlights indicate U.S. rate-peer rate <3.

TABLE 2-1 Continued

Females	2000		2008		2015		Change in Difference
	U.S.	Peers	U.S.	Peers	U.S.	Peers	
All Causes	836.5	732.4	731.8	641.0	713.1	594.6	14.4
Digestive Diseases	34.5	37.2	44.3	31.6	38.5	32.0	9.2
Circulatory Diseases	395.4	363.1	281.5	267.3	257.5	217.0	8.2
Accidental Poisonings	2.5	1.1	6.8	1.6	10.0	1.7	6.9
Intentional Self-Harm	4.1	6.9	4.9	6.1	6.2	5.7	3.3
Respiratory Diseases	96.9	78.8	87.2	68.0	84.4	64.6	1.7
Mental Health/Alcohol Use	2.4	3.2	2.0	3.0	2.6	2.7	0.7
Liver Cancer	3.4	4.0	3.7	4.8	4.6	4.6	0.6
Endocrine Diseases	39.3	26.2	35.1	25.6	36.5	22.9	0.5
Other Cancers	90.6	96.1	80.0	87.5	75.7	81.1	0.1
Genitourinary Diseases	22.9	13.9	22.2	15.2	21.5	14.1	–1.6
Breast Cancer	30.5	30.6	25.6	27.1	23.2	24.2	–0.9
HIV/AIDS	2.5	0.6	1.9	0.5	1.0	0.2	–1.1
Colorectal Cancer	20.4	22.9	16.1	20.0	13.9	17.8	–1.4
Lung Cancer	48.2	22.1	45.4	26.8	39.1	27.6	–14.6

NOTES: Change in difference = $(\text{U.S.–peer})_{2015}$–$(\text{U.S.–peer})_{2000}$.
Orange highlights indicate U.S. rate-peer rate >3.
Green highlights indicate U.S. rate-peer rate <3.
The table presents cause-specific male and female age-standardized mortality rates for the United States and the peer country average for the years 2000, 2008, and 2015. In addition, the table shows how the gap in mortality between the United States and peer countries changed between 2000 and 2015. A positive change in the difference indicates that the United States fared worse than its peers over the period because it experienced either a smaller decrease or a more rapid increase in mortality relative to the peer countries. The table is sorted by the 2000–2015 change in the difference in mortality between the United States and peer countries. "Other cancers" include all cancers excluding prostate, liver, breast, colorectal, and lung cancers.
SOURCE: Data from OECD Stat (https://stats.oecd.org/index.aspx?queryid=30115).

Although the United States became increasingly disadvantaged relative to its 16 peer countries across many causes of death, it also outperformed these countries on lowering mortality due to HIV/AIDS and several types of cancer. Compared with the peer countries, the United States experienced a faster decrease in mortality from HIV/AIDS and colorectal cancer; female mortality due to breast cancer; and male mortality from lung cancer, prostate cancer, and diseases of the genitourinary system. Female mortality from lung cancer and diseases of the genitourinary system decreased in the United States but rose in peer countries. However, these reductions in cancer mortality were offset by increases in mortality from other causes, and the overall mortality gap increased.

Because of reporting differences across countries and over time, the cause-specific mortality rates discussed here could not be restricted to working-age adults, nor could these analyses be extended to the period before 2000. Despite these limitations, however, these analyses provide important clues about trends in causes of death that are most likely responsible for the U.S. disadvantage in life expectancy.

SUMMARY

The current U.S. disadvantage in life expectancy relative to peer countries is part of a decades-long trend in which the United States fell behind its peers in both life expectancy and mortality outcomes. For decades, the increase in U.S. life expectancy failed to keep pace with the increase in peer countries, and the gap widened after 2010 because of the stagnation and decline in U.S. life expectancy, which reversed years of progress. This stagnation and subsequent decline cannot be explained by the greater racial/ethnic diversity of the United States compared with its peers because it occurred among multiple racial/ethnic groups, including Whites.

U.S. working-age mortality has been among the highest of all peer countries since the 1950s, and the recent increase that occurred after 2010 did not occur in the peer countries, where mortality rates continued to decline. In the 1970s, although the United States experienced higher mortality among working-age adults, mortality was lower than in peer countries among young children and those over age 65. Much of the growing gap in life expectancy that occurred in the latter half of the 20th century was due to the expansion of the U.S. mortality disadvantage into both older and younger ages, where the United States had previously held an advantage. This long-term trend was driven by an increasing mortality disadvantage among adults over age 65, particularly among females. In the period since 2000, however, large and growing increases in working-age mortality that occurred only in the United States have played a larger role in the changes

in mortality rates, magnifying and expanding the disparity between the United States and its peers, particularly among males.

Although the United States saw relative improvements over peer countries in mortality from some causes of death (e.g., HIV/AIDS, certain cancers), these gains were offset by the growing U.S. disadvantage for other causes of death, particularly circulatory and digestive system diseases, accidental poisonings, and intentional self-harm. The U.S. disadvantage in mortality relative to its peers extends across a wide range of causes of death, including many chronic diseases that are costly and burdensome to treat, suggesting that eliminating the disparity between the United States and its peers will not be a simple task.

The U.S. disadvantage may be further complicated by the effects of the COVID-19 pandemic. The committee completed its work before detailed mortality data for 2020 were available. Although firm conclusions must therefore await future research, preliminary mortality data suggest that the U.S. mortality disadvantage may have worsened because the United States failed to contain the virus as effectively as did the 16 peer countries (Johns Hopkins University, 2020). The United States experienced higher mortality from COVID-19 relative to most of its peers (Bilinski and Emanuel, 2020). Although the results presented in this chapter show that the United States continued to maintain a mortality advantage at ages 75 and over, the high COVID-19 mortality at these ages is likely to have further eroded that advantage and potentially to have reversed it. In addition, the increase in mortality at working ages due to the virus will further widen the long-standing U.S. mortality disadvantage at these ages. Although the direct effects of COVID-19 on mortality will likely be temporary, some preliminary evidence indicates that those who recover from the disease experience long-term health problems.

ANNEX 2-1

International Trends Methodology

SOURCE OF INTERNATIONAL TREND COMPARISON DATA

Data for the Sixteen Peer Countries

All-cause mortality data for the 16 peer countries included in the analysis in this chapter were downloaded from the Human Mortality Database (HMD) on July 24, 2019 (www.mortality.org). The HMD is a collaborative international effort to provide open, international access to detailed mortality and population data for 41 countries and geographic areas throughout

the world. It is organized by research teams in the United States at the University of California, Berkeley and the Max Planck Institute for Demographic Research in Germany, with scientific contributions from researchers from many countries, and is supported by research grants and financial contributions from governmental and private sources. The HMD data include calculated mortality rates and life tables, as well as the original data underlying these calculations. All the HMD data can be accessed publicly and downloaded for free.

The analysis in this chapter uses calculated data from period life tables based on mortality from all causes of death for every year between 1950 and 2016 for which these data were available for the 16 peer countries. Data covering the full period were available for most of the countries; for a small number of countries, however, the available data did not begin until after 1950 or ended prior to 2016. Data availability for the 16 peer countries can be found in Annex Table 2-1.

Cause-specific mortality rates were drawn from the Organisation for Economic Co-operation and Development (OECD) statistics generator, which in turn draws its data from the World Health Organization's (WHO's) Mortality Database (2019). Data included in the WHO Mortality Database come from national vital statistics registration systems in the respective countries and focus on underlying causes of death coded using the International Classification of Diseases. WHO defines underlying cause of death as "the disease or injury which initiated the train of morbid events leading directly to death, or the circumstances of the accident or violence which produced the fatal injury" (WHO, 2019, p. 1).

Data for the United States

Data on life expectancy at birth for U.S. males and females and for White and Black males and females for the period 1950–2016 were drawn from Table 19 of the National Vital Statistics Reports (NVSRs) published by the National Center for Health Statistics (Arias, Heron, and Xu, 2017; Arias, Xu, and Kochanek, 2019), which includes estimated life expectancy at birth, in years, by race, Hispanic origin, and sex. Detailed sex- and race-specific life table data for 1970, 1980, 1990, 2000, 2010, and 2016 were also drawn from NVSRs from the relevant years (Arias, 2014; Arias, Rostron, and Tejada-Vera, 2010; Arias, Xu, and Kochanek, 2019; National Center for Health Statistics [NCHS], 1964, 1974, 1984, 1994). Although it was possible to download the detailed sex- and race-specific life table data for 1990 and later years directly, data from the 1970 and 1980 NVSRs were copied by hand into digital form. The hand-entered data and subsequent calculations were then checked for accuracy by National Academies' staff.

ANNEX TABLE 2-1 Availability of Mortality Data for the 16 Peer Countries

Country	Available Years	Exclusions/Exceptions
Australia	1950–2016	
Austria	1950–2016	
Canada	1950–2016	
Denmark	1950–2016	
Finland	1950–2016	
France	1950–2016	
Germany	1956–2016	As East and West Germany before 1990
Italy	1950–2014	
Japan	1950–2016	
Norway	1950–2016	
Portugal	1950–2015	
Spain	1950–2016	
Sweden	1950–2016	
Switzerland	1950–2016	
The Netherlands	1950–2016	
United Kingdom	1950–2016	

The reporting of race-specific life expectancy and mortality data in the NVSRs changed over the 1950–2016 period covered in these analyses. Prior to 1970, these reports included life expectancy and mortality estimates for only two racial categories: Whites and non-Whites. From 1970 to 2005, the NVSRs reported life expectancy and mortality estimates for only the White and Black populations. In 2006, the National Center for Health Statistics began to incorporate ethnicity into its NVSRs. Beginning in that year, the NVSRs reported separate life expectancy and mortality estimates for the non-Hispanic White, non-Hispanic Black, and Hispanic populations. These changes mean that the racial/ethnic categories used for the time series in Figure 2-1 are not perfectly comparable over time. For example, the estimates for Whites include Hispanic Whites during the 1950–2005 period but exclude Hispanic Whites during the 2006–2016 period.

ANALYTIC STRATEGY

This chapter presents the results of five sets of analyses, presented in four sections. The first section compares life expectancy at birth by sex in the United States and the 16 peer countries. Life expectancy in the United

States is presented by sex and by sex and race or race and ethnicity. The second set of analyses (second section) presents the age-specific contributions to overall differences in life expectancy at birth between the United States and the peer countries derived using Arriaga decomposition techniques. The third set of analyses, also presented in the second section, shows the ratio of age-specific mortality rates in the United States to the peer country average. The fourth set of analyses, presented in the third section, shows the age-adjusted probability of dying between ages 25 and 64 in the United States and the peer countries, as well as the relative U.S. position expressed as a z-score. The final set of analyses, presented in the fourth section, compares cause-specific mortality rates in the United States with the peer country averages for selected causes of death. These final analytic results were drawn directly from OECD Stat and were calculated by the WHO Mortality Database (2019). The methods used to calculate them are detailed at www.mortality.org.

Life Expectancy at Birth

Life expectancy at birth for each country was not directly calculated but drawn from outside data sources. Life expectancy for the 16 peer countries was calculated by the HMD using standard life table construction methods detailed more extensively in the online documentation located on the HMD website.[2] Life expectancy at birth for the United States was drawn directly from Table 19 of the NVSRs.

Arriaga Age Decomposition of Differences in Life Expectancy

As noted, the committee used Arriaga decomposition techniques (Arriaga, 1984; Auger et al., 2014) to estimate how deaths at specific ages contributed to differences in life expectancy between the United States and the peer countries in 1970, 1980, 1990, 2000, 2010, and 2016. Using this method, it was possible to show the specific contributions of ages <1 year, ages 1–4 years, and all 5-year age groups from 5–9 through 95–99. (The contribution for ages 100 and older was not calculated because deaths above age 100 are unusual and do not contribute substantively to the differences in life expectancy between the United States and peer countries.)

Using this method, the age-specific contributions to differences in life expectancy have two components. First is the direct effect of higher mortality rates for each age group: people who die within that age range relative to those who do not contribute fewer years lived to life expectancy during that range. For example, an individual who dies at age 27.5 in a given year

[2]See http://www.mortality.org.

contributes 2.5 years lived (from age 25 until age 27.5) during the 25–29 age interval, while an individual in this age group who does not die contributes 5 years lived (from age 25.0 to the end of the interval, age 30.0).

The second component of the age-specific contribution to life expectancy is the indirect effect: individuals who die within a given age group will not add any years lived to older age groups. For example, if a woman would have survived to age 64.0 had she been subject to the mortality rates in population A but instead died at age 60.0 because she was subject to the higher mortality rates in population B, then part of the difference in life expectancy for ages 60–64 in populations A and B is due to the fact that this individual did not survive to contribute years lived to this age group in population B but would have done so in population A.

The Arriaga decomposition technique decomposes the total difference in life expectancy into the differences in the contributions to life expectancy of mortality within each age group. These age-specific contributions are calculated as the sum of the direct and indirect effects of differences in mortality between two populations in each age group. For example, the difference between the life expectancy for females in the United States and in peer countries in 1980 was –.33 years; that is, U.S. female life expectancy was .33 years lower than the average female life expectancy in peer countries. This overall .33-year life expectancy difference can be decomposed into age-specific differences between the United States and peer countries:

- a .23-year disadvantage for U.S. females below age 25,
- a .31-year disadvantage for U.S. females between ages 25 and 49,
- a .59-year disadvantage for U.S. females between ages 50 and 74, and
- a .80-year advantage for U.S. females above age 75.

Taken together, these age-specific differences generate the overall .33-year disadvantage:

$$-.23 + -.31 + -.59 + .80 = -.33 \text{ years}$$

Because of the indirect effect of higher mortality, the same differences in mortality can have a larger impact on life expectancy at birth if they occur at younger versus older ages. However, this effect is countered by the fact that age-specific mortality rates are much lower at younger than at older ages, so absolute differences in mortality rates tend to be smaller.

Ratio of Age-Specific Mortality in the United States to That in the Peer Countries

To isolate the effect of international differences in age-specific mortality rates, the committee calculated the ratio of the probability of dying within a given age group in the United States to the peer country average for 1970, 1980, 1990, 2000, 2010, and 2016. As with the Arriaga decompositions, the analysis focused on the specific contributions of ages <1 year, ages 1–4 years, and all 5-year age groups from 5–9 through 95–99. When possible, these age-specific probabilities were drawn directly from the NVSRs (United States) or the HMD (the 16 peer countries). When the probabilities included in these sources were reported in single-year age increments, the 1-year probabilities were used to calculate the 5-year probabilities using standard life table calculation methods (Preston, Heuveline, and Guillot, 2000).

The Probability of Death Between Ages 25 and 64

Figure 2-4 shows the probability of death occurring between ages 25 and 64, based on calculating the probability of dying for each 1 year of age using standard life table methods (Preston, Heuveline, and Guillot, 2000), and then applying these probabilities to a standardized population to create a synthetic cohort whose members were exposed to these death probabilities throughout their working ages. Assume, for example, that the probability of dying between an individual's 25th and 26th birthdays is 0.00057, and the probability between the 26th and 27th birthdays is 0.00063. Based on these probabilities, in a population of 100,000 people who survive to age 25, the probability of dying between ages 25 and 27 would be calculated as

100,000 x 0.00057 = 57 deaths 99,943.00 survivors to age 26
99,943 x 0.00063 = 61.96 deaths 99,881.04 survivors to age 27
probability of death between ages 25 and 27:
(57 + 61.96)/100,000 = 0.0011896

Note that the probability of dying between ages 25 and 27 is less than the sum of the probabilities of dying at ages 25 and 26, because those who do not survive to age 26 are not eligible to also die at age 26. By beginning with a standardized population size and iteratively applying the probability of death at each age to only the population that survives to that age, the final probabilities of death between ages 25 and 64 are standardized and are no longer dependent on the underlying population age distribution. In standard life table notation this estimate refers to $(l_{25} - l_{65})/l_{25}$, where l_{25} is the number of people that survive to age 25, and l_{65} is the number of people that survive to age 65.

3

U.S. Trends in All-Cause Mortality Among Working-Age Adults

The United States has experienced higher mortality among working-age adults (ages 25–64) relative to other high-income countries since at least the mid-20th century (see Chapter 2). Beyond this long-standing difference, something changed in the 21st century to rapidly expand the gap in working-age mortality between the United States and its peers. Since 2010, this change in working-age mortality has been large enough to stall and then reverse the decades-long trend of increasing life expectancy at birth within the United States (Arias and Xu, 2019). To aid in understanding this phenomenon, this chapter presents trends in all-cause mortality (i.e., all deaths from any cause) that occurred in this age group over the study period (1990–2017) in the United States, stratified by individual and geographic characteristics. Examining differences in trends by such individual characteristics as age, sex, race and ethnicity, and socioeconomic status, as well as by geographic characteristics, can provide insights into among whom and where mortality rates have increased.

The chapter begins with an overview of trends in all-cause mortality rates among working-age adults by age and sex over the study period. The second section focuses on racial/ethnic disparities in these rates over the same period, which are shown by the international comparisons in Chapter 2 to have persisted for a long time within the United States. Next is a review of existing literature on trends in socioeconomic disparities in mortality. Finally, geographic differences in trends in mortality rates are examined, including differences by metropolitan area size, region, state, and county.

In the analyses presented in this chapter, deaths were pooled across 3-year periods from 1990 to 2017, with the exception of the first period

(1990–1993), which includes 4 years. Chapter 5 provides an overview of the data and analytic methods on which the discussion in this chapter is based.

The results of these analyses reveal a troubling stagnation and increase in mortality that affected both males and females starting in 2010, regardless of age and race and ethnicity. Among non-Hispanic (NH) White (White) females and males, this marked a continuation of trends that began in the 1990s and 2000s, respectively, and were heavily concentrated among those with a high school degree or less. In contrast, among NH Black (Black) and Hispanic males and females, this trend marked the end of a long period of decreasing mortality and narrowing racial/ethnic disparities in mortality.

The changes in mortality were the result of growing geographic disparities in mortality that favored large central metropolitan areas. Dramatic mortality decreases within large metropolitan areas were often the driving force behind large county and statewide improvements in mortality. These large central metropolitan areas experienced large declines in mortality between 1990–1992 and 2015–2017, while mortality increases were more heavily concentrated in nonmetropolitan areas. The growing disparities in mortality between large central metropolitan areas and less populated areas were more striking and consistent among Whites. These troubling reversals in long-term improvements in mortality rates across multiple demographic groups raise concerns about the outlook for future U.S. mortality trends.

TRENDS IN ALL-CAUSE MORTALITY BY SEX AND AGE

Starting in the early-2010s,[1] all-cause mortality rates stagnated or increased among working-age adults in the United States, regardless of sex or age decile (Figure 3-1).[2] Between 1990 and 2011, mortality rates did not decrease steadily over time, nor did they decline uniformly across sex and age groups. Although mortality among males ages 35–64 declined throughout the 1990s and 2000s, mortality among males ages 25–34 declined in the 1990s, stagnated in the 2000s, and increased after 2011. Females experienced little progress, with their mortality rates remaining mostly flat[3] between the 1994–1996 and 2006–2008 periods. Particularly concerning

[1]To simplify the discussion throughout the chapter, the text refers to the decadal periods of the 1990s (1990–1993 vs. 2000–2002), the 2000s (2000–2002 vs. 2009–2011), and the 2010s (2009–2011 vs. 2015–2017) where relevant.

[2]As shown in Figure 3-1, mortality rates are reported here for four age deciles: 25–34, 35–44, 45–54, and 55–64.

[3]An exception occurred for females ages 55–64, among whom mortality declined steadily until the late 2000s. Among females ages 25–34, mortality rates were slightly higher in 2015–2017 than they were in 1990–1993, and among females ages 35–44, the rates were nearly identical in 1990–1993 and 2015–2017.

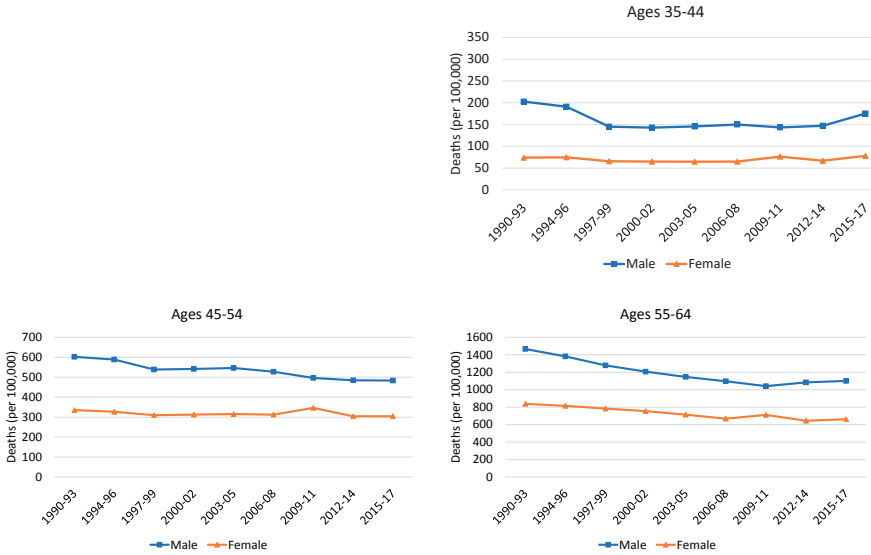


FIGURE 3-1 All-cause mortality rates by sex and 10-year age group, 1990–2017.
NOTE: All-cause mortality rates (number of deaths per 100,000 population) are shown for males and females and for four working-age deciles (ages 25–34, 35–44, 45–54, and 55–64). The blue lines show mortality rates for males, while the orange lines show mortality rates for females. To ensure comparability over time and across subpopulations, rates are age-adjusted by single year of age and standardized to reflect the age distribution of the U.S. population in 2000. Deaths were pooled across 3-year periods, with the exception of the first period (1990–1993), to smooth large fluctuations in mortality trends that sometimes occur when smaller populations have relatively low death counts.
SOURCE: Data from https://www.cdc.gov/nchs/nvss/nvss-restricted-data.htm.

is the reversal and upward tick in mortality rates among both males and females between 2012–2014 and 2015–2017. With the exception of mortality among those ages 45–54, mortality rates among both sexes in the other three age deciles were higher in 2015–2017 than they were in 2012–2014.

Although mortality declined overall over the study period among working-age males and older females, these trends meant that mortality rates changed little among younger working-age females between 1990–1993 and 2015–2017. Among females ages 35–44, mortality rates were nearly unchanged, while among females ages 25–34, the rates were higher in 2015–2017 than in 1990–1993. In contrast, because working-age males saw declines in mortality during the 1990s and 2000s that exceeded the recent increases, their mortality rates remained lower in 2015–2017 than in 1990–1993, despite the increases in mortality they experienced in the 2010s.

TRENDS IN ALL-CAUSE MORTALITY BY SEX, AGE, AND RACE AND ETHNICITY

As noted earlier, the United States has a long history of racial/ethnic disparities in mortality, and these disparities remained sizeable in 2015–2017 (Figure 3-2). Although working-age NH Black (Black) adults experienced large declines in mortality rates[4]—outpacing the decline among working-age NH White (White) adults and narrowing the Black–White mortality gap—their rates remained substantially higher than those among White adults at the end of the study period. Working-age Hispanic adults maintained the lowest mortality rates throughout the period. Although the committee calculated mortality rates and examined trends for working-age NH Asians/Pacific Islanders and NH American Indians/Alaska Natives (AI/ANs), data for these groups are not presented because of concerns about the accuracy of death certificate coding of race and ethnicity for these groups (Arias, Heron, and Hakes, 2016). Summaries of existing research (based on alternative data sources) of mortality trends in these groups are presented in Boxes 3-1 and 3-2, respectively.

Among both Black and Hispanic working-age adults, all-cause mortality rates decreased throughout the 1990s and 2000s, but this was not the case among all working-age White adults. For both males and females, mortality rates among White adults ages 25–54[5] stagnated or increased during most of the 1990s and 2000s. Only those ages 55–64 experienced decreases in mortality during this period. After 2009–2011, all-cause mortality rates stagnated or increased among working-age adults regardless of age, sex, or race and ethnicity. Most concerning, in the most recent period of this analysis (2012–2014 to 2015–2017), mortality rates ticked upward among most of these groups, including those ages 25–44 of both sexes and all three races/ethnicities, White males and females ages 45–64, Black males ages 45–54, and Black females ages 55–64. In fact, among White males and females ages 25–44 and White females ages 45–54, mortality rates in 2015–2017 were already higher than they were in 1990–1993. These troubling reversals across multiple demographic groups erased years of progress in lowering mortality rates and raise concerns about the future outlook for U.S. mortality trends.

[4]As a percentage, rate declines were especially pronounced among Black and Hispanic males ages 25–44 and 45–54.

[5]In this section, the age groups 25–34 and 35–44 were combined to simplify presentation of the results because trends in mortality rates among these two groups were virtually identical (although the absolute rates were higher in the 35–44 group).

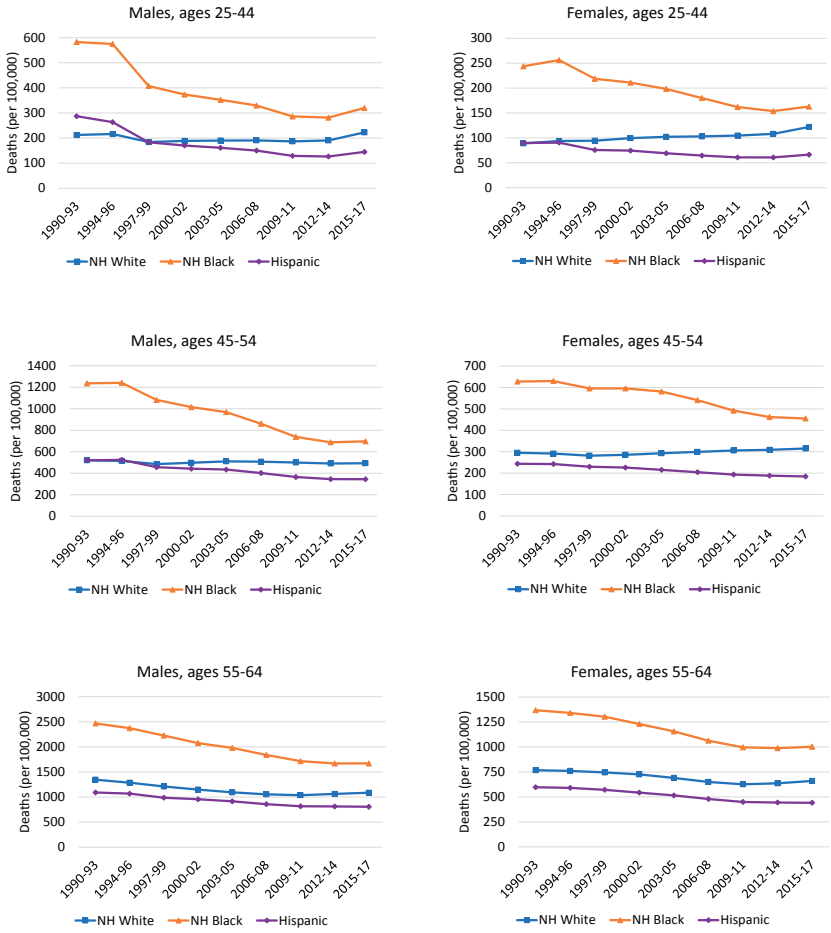


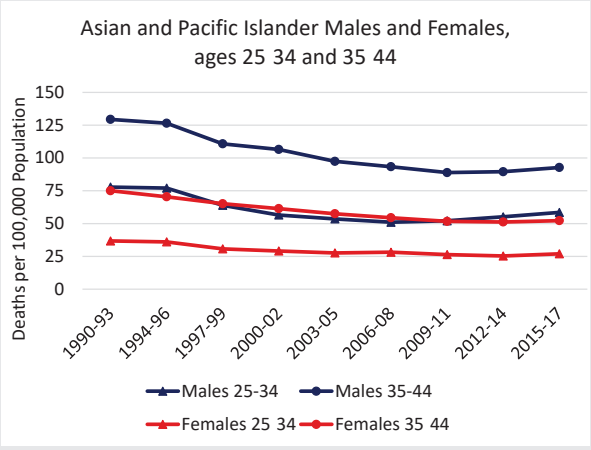
FIGURE 3-2 All-cause mortality rates (deaths per 100,000 population) by sex, age group, and race and ethnicity, 1990–2017.
NOTE: Age-adjusted all-cause mortality rates are shown for non-Hispanic (NH) White (blue lines), NH Black (orange lines), and Hispanic (purple lines) males (left panels) and females (right panels) by age group. The rates are presented for three age groups: 25–44 (top panels), 45–54 (middle panels), and 55–64 (bottom panels). Rates are age-adjusted by single year of age to a standard population age distribution.
SOURCE: Data from https://www.cdc.gov/nchs/nvss/nvss-restricted-data.htm.

BOX 3-1
Trends in Mortality Among Asians and Pacific Islanders

Asians and Pacific Islanders (APIs) are one of the fastest-growing racial/ethnic populations in the United States, which makes tracking changes in mortality within this population an important health surveillance tool. However, changes in immigration patterns and the ethnic composition of this population, as well as improvements in the quality of the classification of race on some death certificates over time—issues that are discussed in greater detail in the annex at the end of this chapter—mean that trends in mortality for this population may reflect changes in composition or data quality rather than changes in mortality. Using methods for estimating API mortality similar to those used for Whites, Blacks, and Hispanics, the committee calculated trends in API all-cause mortality for working-age adults (ages 25–64) from 1990 to 2017. These estimates were not adjusted to account for the reporting errors discussed above; therefore, readers should exercise caution in interpreting these trends given the improvement in data quality over time.

The figure below presents all-cause mortality for four 10-year age groups. Below age 45, mortality rates among both males and females declined until 2009–2011, but subsequently increased slightly, mirroring recent trends among other racial/ethnic groups. At older ages, mortality also declined until 2009–2011 but plateaued thereafter. At all ages, however, API male and female mortality rates remained lower than the rates among other racial/ethnic groups. Among males, API mortality rates were about half as high as the rates among Whites, with the gaps growing over time. API females had rates that were about 40 percent lower than those for Whites in 1990–1993, and the female API advantage grew to about 50 percent lower by the end of the period.

a.



b.

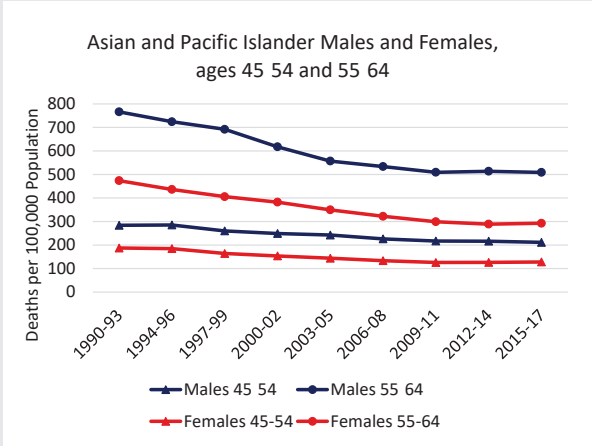


FIGURE B3-1 All-cause mortality rates for Asian/Pacific Islander males and females ages 25–64, 1990–2017.

NOTE: The figure presents age-adjusted all-cause mortality rates (in deaths per 100,000 population) for non-Hispanic Asian/Pacific Islanders (APIs) by age group and sex for 1990–2017. The blue lines show the rates for males, while the red lines show the rates for females. Younger working-age adults ages 25–34 (triangle markers) and 35–44 (round markers) are shown in panel a. Older working-age adults ages 45–54 (triangle markers) and ages 55–64 (round markers) are shown in panel b. Rates are age-adjusted by single year of age to the year 2000 population age distribution.
SOURCE: Data from https://www.cdc.gov/nchs/nvss/nvss-restricted-data.htm.

BOX 3-2
Trends in Mortality Among American Indians and Alaska Natives

Estimating mortality among American Indians and Alaska Natives (AI/ANs) is hampered by known data quality issues stemming from misclassification of the race of AI/ANs on death certificates. These issues are discussed in greater detail in the annex at the end of this chapter and in Chapter 5. Although the reporting of race has improved over time, misclassification of AI/ANs remains extensive. High-quality mortality and population data are collected for tribal areas, allowing researchers to construct mortality and life expectancy estimates for AI/ANs living in 637 Contract Health Service Delivery Area (CHSDA) counties that contain tribal lands or are adjacent to such lands (Arias, Xu, and Jim, 2014; Espey et al., 2014). These estimates are restricted to the 64 percent of non-Hispanic (NH) AI/ANs who live in or near tribal lands. Much less is known about the 36 percent of the AI/AN population that is not covered by these data.

The available evidence reveals that life expectancy among the AI/AN population is much lower than that among NH Whites, NH Blacks, and Hispanics (see Annex Figure 3-3). Within CHSDA counties, working-age AI/AN mortality remains high relative to Whites, with some variation in the magnitude of the disadvantage by CHSDA region (see the figure below). The relative disadvantage is highest at ages 25–44 and is particularly high in the Northern Plains, Alaska, and the Southwest. In all regions, the relative disadvantage at ages 45–64 is also higher than the overall disadvantage and is most pronounced in the Northern Plains and Alaska. The lowest relative disadvantage in all comparisons is found in the East.

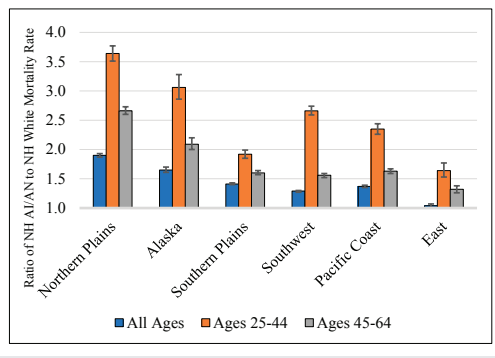


FIGURE B3-2 Ratio of all-cause mortality rates among NH AI/AN to those among NH Whites, Contract Health Service Delivery Area counties, 1999–2009.
NOTE: Rates are age-adjusted; error bars represent 95 percent confidence intervals.
SOURCE: Data from Espey et al. (2014).

It is clear that the AI/AN population suffers much higher mortality relative to other racial/ethnic groups in the United States. Additional efforts are needed to better document the health and mortality of all AI/AN individuals and to address the pressing needs of this population subgroup using more recent data.

TRENDS IN MORTALITY BY SOCIOECONOMIC STATUS

This section summarizes the existing research on trends in all-cause mortality by socioeconomic status (SES). The committee elected to present this summary instead of conducting original analyses because of limitations of the SES data in the death certificate files in the National Vital Statistics System (NVSS) used to generate the original analyses documented in this report, some of which differentially affect mortality estimates for low- and high-SES individuals. One important limitation of these data is that the NVSS files include only a single measure of SES—educational attainment—and this measure, when drawn from death certificates, suffers from well-established issues of accuracy, availability, and quality (Rostron, Boies, and Arias, 2010). A more detailed discussion of this issue is provided in Chapter 5.

The committee therefore carried out a comprehensive review of research that has used NVSS data, as well as alternative data and methods, to assess disparities in mortality by SES. This review considered studies using data that linked survey data to death records to improve the quality of education reports,[6] as well as studies that accounted for changes in the distribution of education in the population over time. As educational attainment increases within the United States, the meaning and benefit of each level of education, as well as the relative socioeconomic position of those who attain that level of education, also change (Dowd and Hamoudi, 2014). The committee reviewed the findings of studies that account for this by using education rank within the educational distribution rather than educational attainment (e.g., Geronimus et al., 2019). The committee also reviewed research using other measures of SES, such as income (e.g., Chetty, Hendren, and Katz, 2016), but there were few studies that relied on measures other than education or income. This review found that a large set of studies—using different data sources, measures of SES, and analytic methods—convincingly documents widening disparities in mortality by SES among working-age Whites in the United States since the 1990s but not among working-age Blacks. Too few studies examine disparities in mortality by SES among Hispanic adults or other racial/ethnic groups for that information to be reported here. The highest-quality and/or most influential studies are discussed below.

A classic study by Kitagawa and Hauser (1973) was among the first to provide comprehensive documentation of socioeconomic disparities in U.S. adult mortality, showing lower mortality among individuals with

[6]The strengths and limitations of these linked survey data are also discussed in Chapter 5.

higher versus those with lower levels of educational attainment and family income. Using 1960 U.S. Census data linked to death certificates for those dying in the 4 months after the U.S. Census was conducted, the authors demonstrated that relative disparities in mortality by education and family income were wider for working-age (25–64) than for older (65+) adults and for Whites than for non-Whites. Relative mortality disparities by educational attainment were generally wider among women, while relative disparities by family income were generally wider among men. However, Kitagawa and Hauser (1973) cautioned that income disparities in mortality are subject to bias because family incomes may decline in the years preceding deaths among family members as a result of health-related job losses and retirements that are themselves associated with both lower income and higher mortality risk. They argued that educational attainment may outperform income as a socioeconomic predictor of mortality risk, a view recently echoed in major literature reviews on the topic (Elo, 2009; Hummer and Hernandez, 2013).

A large body of research on the topic followed the Kitagawa and Hauser (1973) study, with most studies finding that socioeconomic disparities in working-age mortality widened between 1960 and 1990 because of steeper mortality declines among those with higher versus lower educational attainment and income over that period, particularly among White men (Duleep, 1989, 1998; Elo and Preston, 1996; Lauderdale, 2001; Preston and Elo, 1995). For example, Crimmins and Saito (2001) estimated that the disparity in life expectancy among White men at age 30 between those with 0–8 years of education and those with 13 or more years grew from 4.1 to 6.7 years between 1970 and 1990 (Crimmins and Saito, 2001). Similarly, Cutler, Meara, and Richards-Shubik (2011) showed that educational disparities in mortality for White adults ages 25–74 widened between the 1970s and 1990s. By 1990, then, it was clear that there were wide socioeconomic disparities in U.S. working-age mortality by both educational attainment and family income, with especially large disparities for White men and women (Pappas et al., 1993).

Since 1990, major data innovations have provided even stronger evidence of growing socioeconomic disparities in mortality (Hummer and Lariscy, 2011). First, educational attainment was added to the Standard U.S. Certificate of Death in 1989. While states differed in the timing of their adoption of this new item, some studies now trace educational disparities in working-age mortality on a yearly basis back to 1990; such data also facilitate documentation of changes in detailed cause-specific mortality by educational attainment for most of the United States. Second, several large, nationally representative surveys, such as the National Health Interview Survey and the Current Population Survey, began systematically linking their respondents to the National Death Index. Such linked datasets

provide analysts with self-reported data on educational attainment and family/household income that are statistically linked to follow-up mortality records. Third, researchers more recently have linked more than 1 billion individual-level income records from tax data compiled by the Internal Revenue Service to follow-up mortality data from the Social Security Administration to examine detailed trends in adult mortality by income (Chetty, Meara, and Richards-Shubik, 2016).

Using these new sources of data and measures of SES, a new set of studies has convincingly documented widening disparities in mortality among working-age adults in the United States since the 1990s. The most prominent study using income as the primary measure of SES is that of Chetty, Meara, and Richards-Shubik (2016), who used income tax data and follow-up mortality records to examine trends in all-cause mortality for adults ages 40–76 between 2001 and 2014 but did not evaluate disparities for different racial/ethnic groups. They report very little change in mortality rates for those in the bottom 5 percent of the income distribution but a marked decrease in mortality among those in the top 5 percent. As a result, the disparity in mortality by individual-level income increased starting at the beginning of the 21st century, with estimated life expectancy differences of 10.1 years among women and 14.6 years among men for those in the highest and lowest income percentiles in the country, respectively.

The literature on trends in working-age mortality by educational attainment is much more extensive than that on trends by income. Several prominent studies (Jemal et al., 2008; Meara, Richards, and Cutler, 2008; Montez et al., 2011) report widening mortality differences by educational attainment among working-age Whites between the late 1980s/early 1990s and the late 1990s/early 2000s that were more pronounced among women than men. Moreover, those studies also document increasing working-age mortality among White women with less than a high school education during the 1990s alongside continued declines in working-age mortality among White women with a college degree or higher. While Jemal and colleagues (2008) and Montez and colleagues (2011) found no evidence of widening educational disparities in working-age mortality among Black adults, Meara, Richards, and Cutler (2008) and Masters, Hummer, and Powers (2012) each report a modest widening of educational differences in working-age mortality in this population.

Most striking, Sasson (2016) used vital statistics data to demonstrate substantially widening educational disparities in working-age mortality for Whites, with rates among White women and men with less than a high school degree exhibiting substantial increases between 1990 and 2010. White women and men with a high school degree also exhibited modestly increasing working-age mortality rates between 1990 and 2010. At the same time, Black women and men of all educational levels exhibited

mortality decreases across the 20-year period (Sasson, 2016). As a result, there was little change in educational disparities in life expectancy for Black women and men during this period, but there were steep increases in educational disparities in life expectancy for White women and men. Similar to the disparities by income, life expectancy for women and men varies by 10–12 years, respectively, among those with the highest and lowest levels of educational attainment.

Most recently, Geronimus and colleagues (2019) document changes in educational disparities among Black and White women and men of working age (and older) between 1990 and 2015. Previous research on educational disparities in mortality has been criticized for not taking into account changes that occur in the characteristics of people within education categories as educational attainment increases in the population over time (Dowd and Hamoudi, 2014). Therefore, Geronimus and colleagues measured educational attainment in quartiles, comparing changes in mortality disparities between 1990 and 2015 among those in the bottom 25 percent of the educational attainment distribution versus those in the top 25 percent. Their results, consistent with those of the above studies, showed that educational disparities in mortality among those ages 25–64 had widened for White men and women since 1990.

GEOGRAPHIC DIFFERENCES IN MORTALITY TRENDS

The national trends reported thus far in this chapter mask considerable geographic variation in mortality that occurred across the nation during the study period. This section presents trends in all-cause mortality across several measures of U.S. geography: metropolitan status, region, state, and county.[7] In these analyses, metropolitan status is classified into four groups[8]: (1) *large central metropolitan areas* (counties in metropolitan statistical areas [MSAs] of more than 10 million population, including counties that contain all or part of the area's inner cities, hereafter referred to as "large central metros"); (2) *large fringe metropolitan areas* (surrounding counties of the large central metros, hereafter referred to as "large fringe metros"), corresponding to suburbs; (3) *small and medium metropolitan areas* (counties in MSAs of 50,000–999,999 population, hereafter referred to as "small/medium metros"); and (4) *nonmetropolitan areas* (counties outside of metropolitan areas, hereafter referred to as "nonmetros"), corresponding

[7]To simplify the analyses and improve the quality of estimates for smaller geographic levels, only two age groups were used for these analyses: 25–44 and 45–64 years.

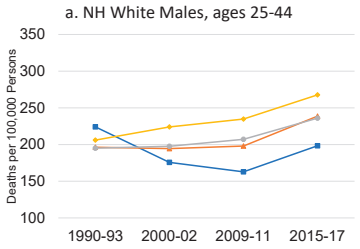
[8]To maintain consistency over time, the counties' metropolitan categories were assigned based on the 2013 classification scheme of the U.S. Department of Agriculture Economic Research Service. For more information, see Chapter 5.

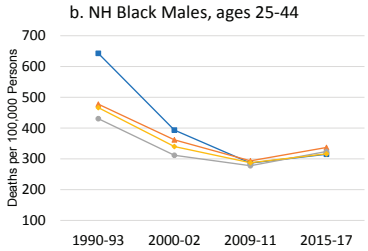
to rural areas. Region of the United States is classified into the four broad areas of the United States defined by the U.S. Census Bureau: Northeast, Midwest, South, and West.

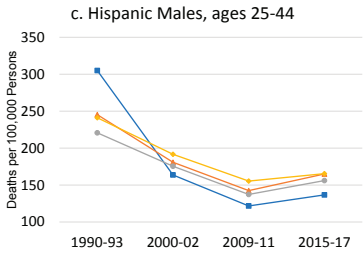
Trends in All-Cause Mortality by Metropolitan Area Type

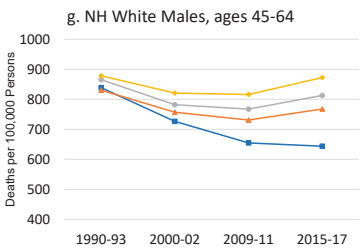
Two clear trends emerged over the 1990–2017 period to produce growing disparities in mortality among metropolitan area types, disparities that were particularly large among working-age Whites (Figure 3-3). First, working-age adults in large central metros experienced the most favorable trends in mortality over the period, driven by large improvements in mortality during the 1990s and 2000s that far exceeded those occurring in less-populated areas. When mortality increased in large central metros during these two decades, as it did among younger working-age White females (ages 25–44), these increases were smaller than those in other areas. Among younger White males, decreasing mortality in large central metros was large enough to mask the emergence of increasing mortality elsewhere. Working-age adults in large central metros continued to experience a mortality advantage in the 2010s compared with those in less-populated areas, but this advantage was smaller than in the two previous decades as improvements in mortality stalled or reversed in most areas. Although older Black males and females continued to see decreases in mortality in the 2010s, these improvements were much smaller than those of previous decades.

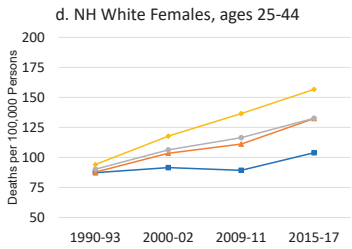
The second clear trend that emerged is a growing working-age mortality disadvantage outside of large central metros, particularly within nonmetros. Females (White, Black, and Hispanic) and White males in nonmetros experienced either the smallest improvements or the largest increases in mortality throughout the 1990s and 2000s. Younger working-age Whites experienced increasing mortality in all areas outside of large central metros, with the largest increases occurring in nonmetros. Among older White females, only those in nonmetros saw increasing mortality; mortality decreased in more populated areas. Older Black males experienced the smallest improvements in mortality in large fringe metros, while Hispanic males and younger Black males in small/medium metros did so. The one exception to these trends was older working-age Hispanic females, among whom mortality declined in parallel across most metro areas; only those living in large fringe metros experienced slower decreases in mortality relative to those in other areas. In the 2010s, mortality increases expanded to most working-age adults living outside of large central metros. Nonmetros continued to experience the least favorable trends among most older working-age adults, except older Hispanic males. Among younger working-age adults and older Hispanic males, however, mortality increased the

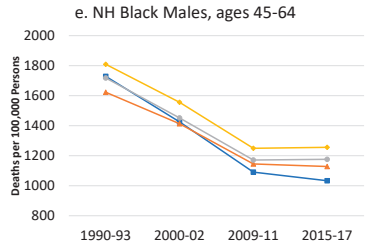

a. NH White Males, ages 25-44
Deaths per 100,000 Persons
350
300
250
200
150
100
1990-93
2000-02
2009-11
2015-17

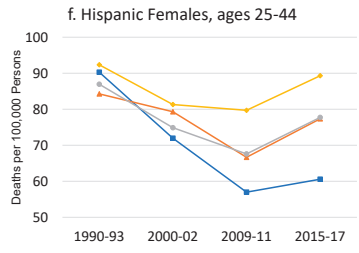

b. NH Black Males, ages 25-44
Deaths per 100,000 Persons
700
600
500
400
300
200
100
1990-93
2000-02
2009-11
2015-17

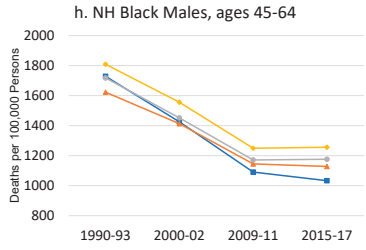

c. Hispanic Males, ages 25-44
Deaths per 100,000 Persons
350
300
250
200
150
100
1990-93
2000-02
2009-11
2015-17


g. NH White Males, ages 45-64
Deaths per 100,000 Persons
1000
900
800
700
600
500
400
1990-93
2000-02
2009-11
2015-17


d. NH White Females, ages 25-44
Deaths per 100,000 Persons
200
175
150
125
100
75
50
1990-93
2000-02
2009-11
2015-17


e. NH Black Males, ages 45-64
Deaths per 100,000 Persons
2000
1800
1600
1400
1200
1000
800
1990-93
2000-02
2009-11
2015-17


f. Hispanic Females, ages 25-44
Deaths per 100,000 Persons
100
90
80
70
60
50
1990-93
2000-02
2009-11
2015-17


h. NH Black Males, ages 45-64
Deaths per 100,000 Persons
2000
1800
1600
1400
1200
1000
800
1990-93
2000-02
2009-11
2015-17

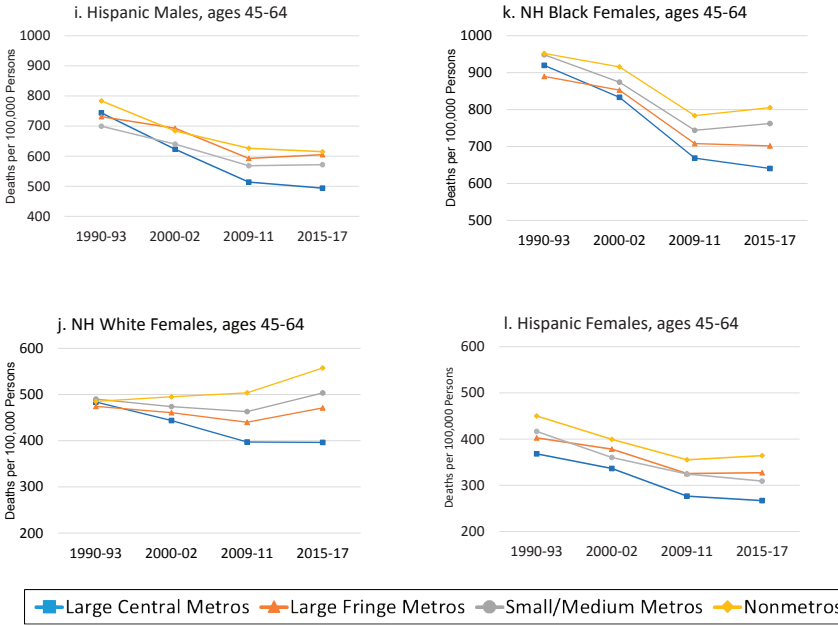


FIGURE 3-3 All-cause mortality rates by race and ethnicity, sex, age group, and metropolitan status, 1990–2017.
NOTE: All-cause mortality rates are shown for ages 25–44 (panels a-f) and 45–64 (panels g-l) across four levels of metropolitan status: (1) large central metropolitan areas (blue lines), (2) large fringe metropolitan areas (orange lines), (3) small or medium metropolitan areas (gray lines), and (4) nonmetropolitan areas (yellow lines). The rates in these four types of areas are presented separately by sex (males in panels a-c and g-i; females in panels d-f and j-l) and for non-Hispanic (NH) Whites (panels a, d, g, j), NH Blacks (panels b, e, h, k), and Hispanics (panels c, f, i, l). Rates are age-adjusted by 10-year age group.
SOURCE: Data from https://www.cdc.gov/nchs/nvss/nvss-restricted-data.htm.

most in large fringe metros, except among younger Black males, who saw slightly larger mortality increases in small/medium metros.

Taken together, these two trends led to a growing mortality gap between large central metros and nonmetros, except among younger Black and Hispanic males. Among younger Black and Hispanic males, extraordinarily high mortality within large central metros in 1990–1993 combined with the dramatic improvements in these areas over the subsequent two decades meant that the differences among metropolitan area types were smaller in 2015–2017 than in 1990–1993. In fact, younger working-age adults in large central metros generally had higher mortality rates in 1990–1993, but mortality rates in these areas were below those in less-populated areas

by the end of the period. At the same time, the unfavorable trends within nonmetros among working-age females (except older Hispanic females) and White males created a large and growing nonmetro mortality "penalty" that was particularly large among working-age Whites. The large relative improvements in mortality among Black adults, combined with increasing mortality in nonmetro areas among younger White adults, led to a significant narrowing of racial disparities in mortality between working-age Blacks and Whites over the period within all metropolitan area types.

Trends in All-Cause Mortality Across U.S. Regions and States

Figures 3-4a to 3-4d show trends in all-cause mortality between 1990–1992 and 2015–2017 for the 50 states,[9] organized by the four U.S. Census regions. There was considerable variation in mortality rates across the regions and across states within regions, as well as within states, at both time points.

The Southern region of the United States maintained the highest mortality throughout the period; however, mortality in the Northeast among younger working-age adults, particularly younger males, was nearly as high as in the South in 1990–1992 because of very high mortality rates in New York and New Jersey. And while the Southern region had the highest mortality in 1990–1992, the Northeast experienced the largest improvement between those years and 2015–2017. Among younger adults, these improvements were driven entirely by large decreases in mortality in New York and New Jersey, but among older working-age adults, all-cause mortality rates improved substantially in all Northeastern states.

Mortality rates were lowest in the Western region of the United States in 1990–1992, except among younger working-age males. High mortality among younger males in California pushed the region's mortality rate above that of the Midwest. By 2015–2017, however, mortality rates for younger males had decreased substantially in California while increasing slightly in the Midwest, leaving the Western region with the lowest all-cause mortality across the four regions. The increases in all-cause mortality among younger females between 1990–1992 and 2015–2017 were driven by increases in mortality in both the Midwest and the South; mortality rates among younger females decreased in the Northeast and West over this period. Although the Northeast experienced the most consistent improvements in mortality over the period, these gains were not large enough to offset that

[9]The District of Columbia (DC) is excluded from the figures because mortality rates for DC in 1990–1992 were about twice those of other states, and its inclusion dramatically skewed the charts. The rates for DC are discussed in the text where relevant.

region's higher mortality rates in 1990–1992, and the Western region continued to have the lowest mortality in 2015–2017.

Within U.S. Census regions, there was variation across states in how these regional trends were produced. In some regions, mortality trends were driven by changes within in a single populous state, such as among younger males in the Northeast and West. In other cases, trends were consistent across states within the region, such as among older adults in the Northeast. Consistent with the trends by metropolitan area type, the largest and most consistent improvements in mortality occurred in DC and in states with large populations that are anchored by large central cities, including New York, California, New Jersey, and Illinois. In contrast, the states that experienced the largest increases in mortality (or the smallest decreases among older working-age adults) were often characterized by larger proportions of the population in small/medium metros and nonmetros, as was the case, for example, in West Virginia, New Hampshire, Oklahoma, and New Mexico. In some regions, the differences among states within the region were larger than the differences across regions.

Northeast and West

The Northeast and West exhibited the most favorable trends in mortality rates between 1990–1992 and 2015–2017, with comparatively large mortality declines occurring across both sexes and age groups.[10] These favorable trends were not uniform across the regions in both age groups, however. Younger working-age adults in upper New England, for example, had a different experience from that of their peers in states in the southern tier of the Northeast. Thus, while mortality for the Northeast region declined by 28.9 percent over the period among males ages 25–44, it decreased little in Massachusetts and Rhode Island and increased greatly in New Hampshire (64.7%) and Maine (38.5%). The region's mortality reduction was driven mainly by very large declines in New York (56.6%) and New Jersey (33.0%). The decrease in New York was striking, with that state beginning the period with the nation's highest mortality rate and ending it with among the lowest. New York consistently experienced the largest absolute reductions in mortality among all 50 states. Among older working-age adults, mortality improved in all Northeastern states, but the smallest increases occurred within Pennsylvania and Maine.

[10]In the Northeast and West, respectively, mortality rates decreased by 28.9 percent and 33.1 percent among males ages 25–44, by 32.5 percent and 25.1 percent among males ages 45–64, by 17.7 percent and 8.4 percent among females ages 25–44, and by 29.0 percent and 22.8 percent among females ages 45–64. These declines exceeded those in the other two regions.

In the West, mortality among males ages 25–44 decreased even more (33.1%) than in the Northeast, but this reduction was driven by large decreases in California (46.5%), along with smaller decreases in nearby Nevada (26.3%) and Arizona (16.8%). Mortality increased moderately among males ages 25–44 in New Mexico and more substantially (47.2%) among females in that age group, and that state began and ended the period with the highest rates in the region. New Mexico was also the only state in the West to see increasing mortality among older working-age adults (ages 45–64). Rates among younger adults also increased in Montana, Wyoming, and Alaska, where older adults experienced only marginal improvements.

South

Relative to the other three regions, the South experienced the highest mortality—in both 1990–1992 and 2015–2017[11]—and many of the largest increases in mortality throughout the period, particularly among females. Compared with their counterparts in the Northeast and the West, working-age males in the South experienced smaller declines in mortality, and while all-cause mortality decreased in the Northwest and West for females ages 25–44, it increased by 6.1 percent in the South. Although mortality declined by 10.2 percent within the region overall among females ages 45–64, the South accounted for all but one of the eight U.S. states in which mortality increased.[12]

West Virginia, Kentucky, Tennessee, Oklahoma, Arkansas, Alabama, and Mississippi had especially unfavorable mortality trends among working-age adults. West Virginia was particularly affected: Of all 50 states, it experienced the largest absolute increase in mortality rates among young adults (ages 25–44), the third largest increase among older females, and the smallest decrease in mortality among older males, and it ended the period with either the highest (ages 25–44) or second highest (ages 45–64) mortality rates in the nation.[13] The adjacent state of Kentucky experienced

[11]As of 2015–2017, states in the South had four of the five highest mortality rates among males ages 25–44 (West Virginia, New Mexico, Mississippi, Kentucky, and Alabama), four of the five highest mortality rates among females ages 25–44 (West Virginia, Kentucky, Alabama, New Mexico, and Mississippi), the five highest mortality rates among males ages 45–64 (Mississippi, West Virginia, Alabama, Kentucky, and DC), and the ten highest rates among females ages 45–64. DC, which experienced among the largest mortality rate declines (28.8%), still had the sixth highest mortality rate in 2015–2017.

[12]The mortality rate increased in Mississippi, West Virginia, Oklahoma, Arkansas, Kentucky, Alabama, Tennessee, and New Mexico.

[13]Mississippi experienced modest changes in mortality but ended the period with the nation's highest mortality rates among adults ages 45–64.

the second highest absolute increase in mortality among young adults (ages 25–44).

The South was also the location of some of the states with the largest improvements in mortality, which occurred most notably in DC, Maryland, Virginia, North Carolina, Georgia, Florida, and Texas. Working-age adults in DC experienced the largest absolute decreases in mortality in the United States, and younger adults and older females experienced the largest percentage decreases.[14] Aside from DC, several states in the South, including Texas, Georgia, and Florida, saw decreases in mortality of more than 20 percent. In many ways, the South represents a tale of two regions, with high and rising mortality rates in the east south central region and much of southern and central Appalachia, and major improvements in states along the Atlantic coast. Among females ages 45–64, for example, mortality increased in Oklahoma[15] and Arkansas and a chain of states along the Appalachian trail (Mississippi, Alabama, Tennessee, Kentucky, and West Virginia), but decreased in some Gulf Coast states (Texas, Louisiana) and states stretching north along the Atlantic seaboard (Florida, Georgia, South Carolina, North Carolina, Virginia, Maryland, and Delaware).

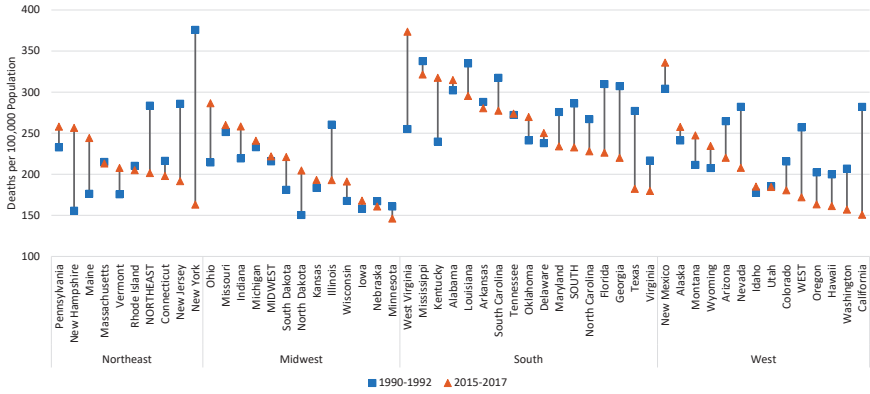
Midwest

Between 1990–1992 and 2015–2017, young adults and older females in the Midwest experienced unfavorable mortality trends, with increasing mortality among the former group and only marginal increases among the latter. The industrial Midwest and the Dakotas saw the largest increases in mortality among younger working-age adults, although those in Illinois fared better. Whereas other regions of the nation saw mortality rates decrease for males ages 25–44 during the period, the Midwest—which began the period with comparatively low mortality—saw rates increase by 2.8 percent. Large increases in such states as Ohio (33.6%) were offset by a large improvement in Illinois (26.0%). Trends among females ages 25–44 were even less favorable; their mortality rates increased by 20.2 percent overall, with large increases in Ohio (55.0%), Indiana (41.2%), North Dakota (57.5%), and South Dakota (47.7%). Illinois was the only state in the region to experience a decrease in mortality among both

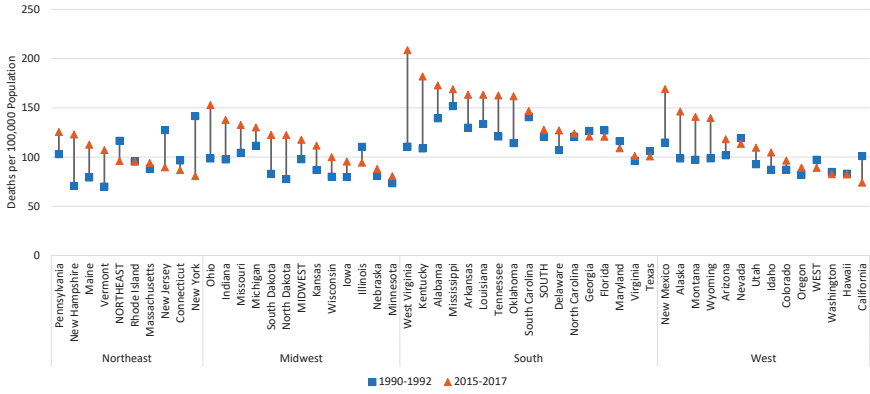
[14]In DC, mortality rates decreased by 633.4 deaths per 100,000 (77.0%) among males ages 25–44, 164.2 deaths per 100,000 (64.0%) among females ages 25–44, 779.1 deaths per 100,000 (42.9%) among males ages 45–64, and 265.3 deaths per 100,000 (28.8%) among females ages 45–64.

[15]The increase in Oklahoma was modest, but the state ended the period with the third highest mortality rates in the nation.

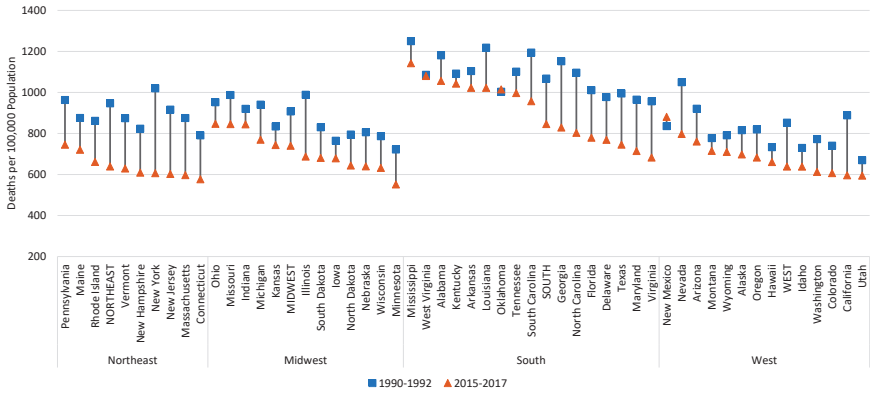
(a) Males ages 25-44



(b) Females ages 25-44



(c) Males ages 45-64



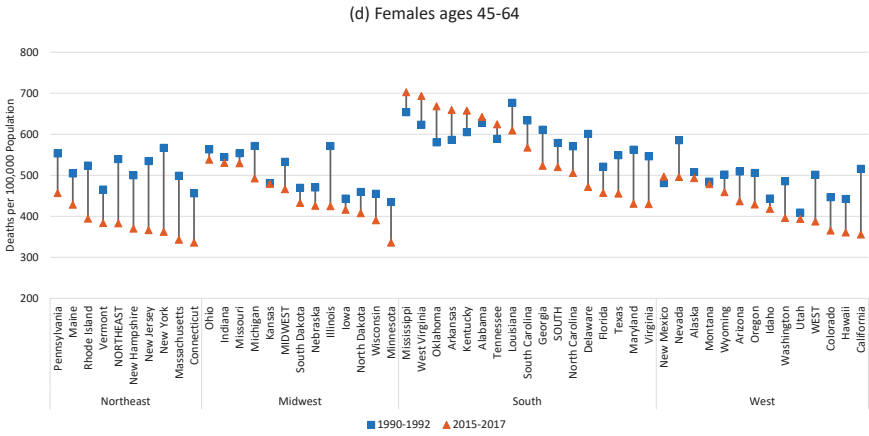


FIGURE 3-4 All-cause mortality rates (deaths per 100,000 population) by U.S. Census region and state, 1990–1992 and 2015–2017.
NOTE: Working-age mortality rates are shown for 1990–1992 (blue squares) and 2015–2017 (orange triangles). The length of the connecting lines between the 1990–1992 and 2015–2017 values indicates the absolute changes in the mortality rates. Rates are age-adjusted by 10-year age band. DC is excluded because of excessively high mortality in 1990–1992 that would have skewed the chart. States are sorted by highest to lowest mortality in 2015–2017 within U.S. Census region.
SOURCE: Data from https://www.cdc.gov/nchs/nvss/nvss-restricted-data.htm.

younger and older working-age adults. Although mortality decreased in the Midwest among males and females ages 45–64—by 18.4 percent and 12.5 percent, respectively—this decrease was smaller than that in any of the other three regions. Ohio, with its borders overlapping the Appalachian region, ended the period with the highest mortality rates in the region. Although Minnesota experienced only modest changes in mortality during the period, its mortality rates as of 2015–2017 were among the lowest in the nation.

Trends in All-Cause Mortality Across U.S. Counties

County-level mortality rates highlight the fact that just as mortality trends often varied dramatically across states within a region, they varied considerably across counties, including those within the same state (Figures 3-5 to 3-8). Indeed, these county-level within-state disparities can often exceed the disparities among states. These trends show that decreasing mortality within large central metros within a state can mask increasing

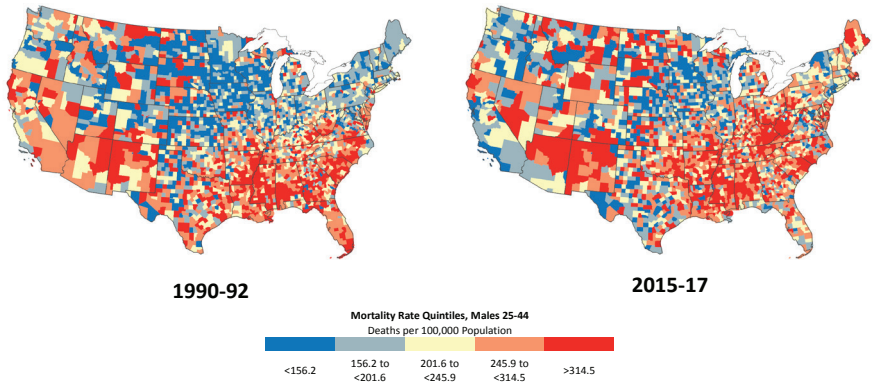


FIGURE 3-5a County-level all-cause mortality rates (deaths per 100,000 population), 1990–1992 and 2015–2017: Males ages 25–44.
NOTE: Mortality rates are shown for 1990–1992 (left) and 2015–2017 (right). Counties are classified into quintiles based on sex- and age-specific mortality rates in 1990–1992. Quintiles with the most favorable mortality rates are dark blue, while those with the least favorable mortality rates are dark red. Consistent quintiles were used for 1990–1992 and 2015–2017 to show changes in the share and distribution of counties in a specific mortality range for each sex and age group (i.e., to show both the share of counties where mortality rates were higher in 2015–2017 versus 1990–1992 and where the rates increased). Rates are age-adjusted by 10-year age group. Small population sizes and death counts resulted in extremely high mortality rates for some counties (e.g., 1 death in a population of 59 results in an age-adjusted mortality rate of 2,272 per 100,000 population). Accordingly, Winsorized binning was used to calculate the rate quintiles, so that counties with mortality rates in the top and bottom 1 percent were removed. On the maps, those counties in the bottom and top 1 percent of mortality are represented in the bottom and top quintiles, respectively. All counties with mortality rates of 0 are included in the bottom quintile. For ease of presentation, figures are limited to the contiguous United States; Alaska and Hawaii are excluded.
SOURCE: Data from https://www.cdc.gov/nchs/nvss/nvss-restricted-data.htm.

mortality within that state's less-populated areas. The county-level mortality rates also identify areas of increasing mortality that cross state boundaries, such as the growing mortality disadvantage in the Appalachian region that includes areas of Ohio, Kentucky, West Virginia, and Pennsylvania.

At the county level, among males ages 25–44 in 1990–1992, the highest mortality rates were concentrated in counties in the Southeast and parts of the desert Southwest and Mountain states (Figure 3-5a). Counties with low

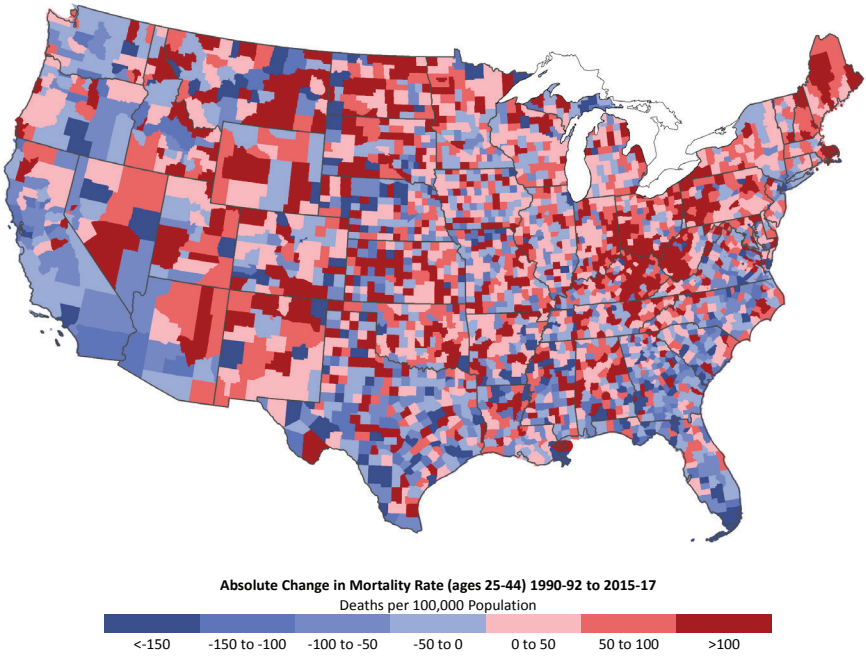


FIGURE 3-5b Absolute change in county-level all-cause mortality rates (deaths per 100,000 population), 1990–1992 to 2015–2017, by U.S. county: Males ages 25–44.
NOTE: Counties with the largest decreases in mortality are dark blue, while those with the largest increases in mortality are dark red. Rates are age-adjusted by 10-year age group. Small population sizes and death counts resulted in extremely high mortality rates for some counties (e.g., 1 death in a population of 59 results in an age-adjusted mortality rate of 2,272 per 100,000 population). For ease of presentation, figures are limited to the contiguous United States; Alaska and Hawaii are excluded.
SOURCE: Data from National Vital Statistics System Detailed Mortality Files.

mortality rates were concentrated in New England, the Northeast, and the Midwest. By 2015–2017, a new pocket of counties with high mortality had emerged throughout Appalachia, including a band stretching from eastern Indiana to southern Ohio, Kentucky, and western New York and Pennsylvania. Counties in the Mountain West and the New England states of Maine, New Hampshire, Vermont, and Massachusetts also experienced large increases in mortality among younger working-age males (Figure 3-5b). The

counties that saw the largest declines in mortality among males in this age group were along the Pacific Coast and the Southeastern Atlantic seaboard.[16]

Among older working-age males, mortality was more likely to decrease over the period, leading to a greater diffusion of areas with mortality improvements. Although counties with decreasing mortality among younger males were concentrated in a small number of regional pockets within the United States, males ages 45–64 experienced decreases in mortality between 1990–1992 and 2015–2017 across a large number of counties in most regions of the country (Figures 3-6a and 3-6b).

Southern counties experienced the largest improvements in mortality, although they remained the most disadvantaged overall. Large increases in mortality occurred in a small pocket of counties in central Appalachia (comprising West Virginia, Eastern Kentucky, and Southern Ohio), while other counties with large increases were scattered throughout the central and Western United States. Although mortality rates increased among younger males in New England, the rates for males ages 45–64 declined throughout the region. Improvements in mortality were also apparent in California, in the Northern Midwest, and along the Southeastern seaboard.

County patterns among younger females (ages 25–44) mirrored those of younger males (Figure 3-7a), but their mortality increases were far more pronounced and widespread across the United States (Figure 3-7b). County-level mortality rates show a startling increase in the share of U.S counties with high mortality among young females.[17] As was true among younger males, the counties that experienced large increases in mortality among young females were located in the Appalachian region, extending down into Northeastern Tennessee, but also in New Mexico, most of Oklahoma, and to a lesser extent across Arkansas and Arizona. The counties that experienced improvements in mortality among young females were less consolidated by region but were often located along the coasts and parts of the central United States, particularly in large central metros.

Among females ages 45–64, the counties that experienced large increases in mortality stretched across the Appalachian region and through Tennessee, Arkansas, and Oklahoma, just as occurred among younger females (Figures 3-8a and 3-8b). Compared with the trends among younger

[16] County-level mortality estimates are not calculated separately by racial/ethnic group because the resulting small cell counts for many counties would produce unstable estimates for Black and Hispanic populations. Because county-level estimates are not adjusted for differences in racial composition, some differences in mortality among counties may be attributable to the racial/ethnic composition of those counties. For example, mortality declines in the Southeast were driven largely by massive declines in mortality rates among Black adults.

[17] Based on the national trends reported earlier (increases in mortality among White females and declines among Black and Hispanic females), one can conclude that these rate increases were driven by mortality increases among White females.

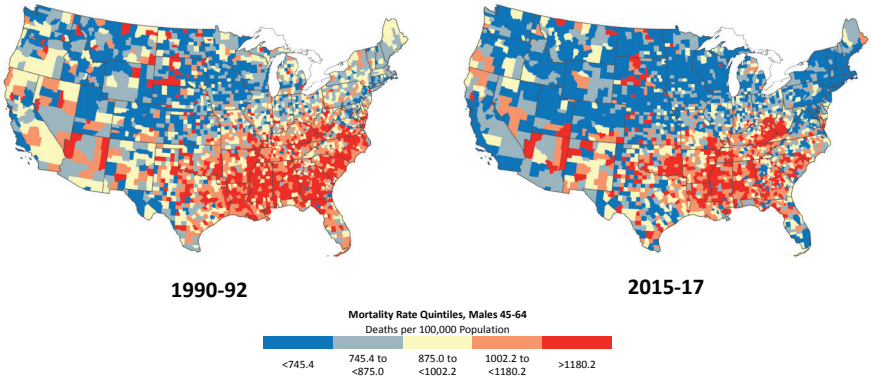


FIGURE 3-6a County-level all-cause mortality rates (deaths per 100,000 population), 1990–1992 and 2015–2017: Males ages 45–64.
NOTE: Mortality rates are shown for 1990–1992 (left) and 2015–2017 (right). Counties are classified into quintiles based on sex- and age-specific mortality rates in 1990–1992. Quintiles with the most favorable mortality rates are dark blue, while those with the least favorable mortality rates are dark red. Consistent quintiles were used for 1990–1992 and 2015–2017 to show changes in the share and distribution of counties in a specific mortality range for each sex and age group (i.e., to show both the share of counties where mortality rates were higher in 2015–2017 versus 1990–1992 and where the rates increased). Rates are age-adjusted by 10-year age group. Small population sizes and death counts resulted in extremely high mortality rates for some counties (e.g., 1 death in a population of 59 results in an age-adjusted mortality rate of 2,272 per 100,000 population). Accordingly, Winsorized binning was used to calculate the rate quintiles, so that counties with mortality rates in the top and bottom 1 percent were removed. On the maps, those counties in the bottom and top 1 percent of mortality are represented in the bottom and top quintiles, respectively. All counties with mortality rates of 0 are included in the bottom quintile. For ease of presentation, figures are limited to the contiguous United States; Alaska and Hawaii are excluded.
SOURCE: Data from https://www.cdc.gov/nchs/nvss/nvss-restricted-data.htm.

females, however, the mortality increases for females ages 45–64 were smaller and less geographically widespread. In contrast, areas with counties showing increasing mortality among older females were more pronounced and more widespread throughout the United States relative to counties with increasing mortality among males in this older age group. Despite the large increases in mortality within the Appalachian region, high mortality rates among older females became much more concentrated in the South over the period. This trend stood in contrast to the pattern among males in the same age group, for whom mortality rates in the South improved dramatically.

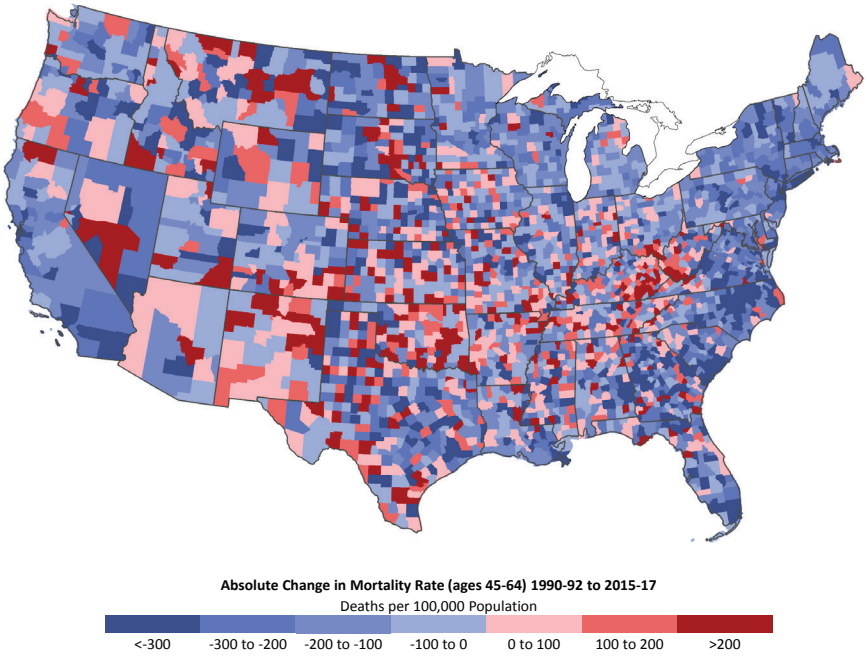


FIGURE 3-6b Absolute change in county-level all-cause mortality rates (deaths per 100,000 population), 1990–1992 to 2015–2017, by U.S. county: Males ages 45–64.
NOTE: Counties with the largest decreases in mortality are dark blue, while those with the largest increases in mortality are dark red. Rates are age-adjusted by 10-year age group. Small population sizes and death counts resulted in extremely high mortality rates for some counties (e.g., 1 death in a population of 59 results in an age-adjusted mortality rate of 2,272 per 100,000 population). For ease of presentation, figures are limited to the contiguous United States; Alaska and Hawaii are excluded.
SOURCE: Data from https://www.cdc.gov/nchs/nvss/nvss-restricted-data.htm.

A much smaller number of counties experienced substantial decreases in mortality among females ages 45–64. The counties in which mortality decreased substantially among older females were concentrated along the East and West Coasts and throughout the northern Great Lakes region and New England.

From these county-level maps, it is clear that many of the counties with the largest declines in mortality are home to the nation's largest cities. To clarify whether the observed county-level patterns are related to metropolitan status, the committee examined how the percentage of counties with the highest and lowest mortality rates changed between 1990–1993 and

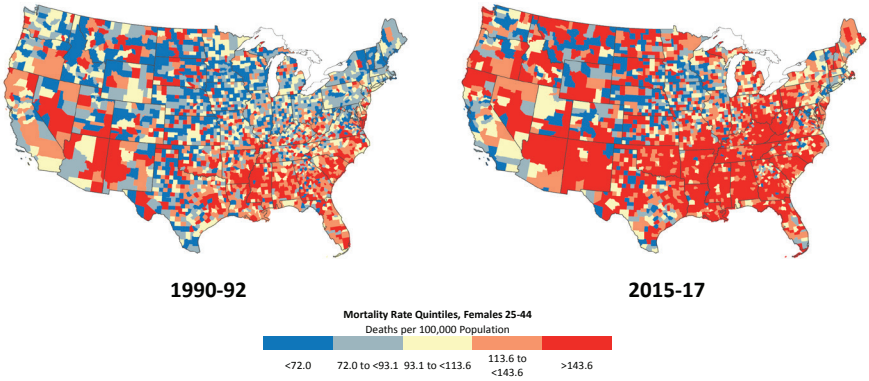


FIGURE 3-7a County-level all-cause mortality rates (deaths per 100,000 population), 1990–1992 and 2015–2017: Females ages 25–44.
NOTE: Mortality rates are shown for 1990–1992 (left) and 2015–2017 (right). Counties are classified into quintiles based on sex- and age-specific mortality rates in 1990–1992. Quintiles with the most favorable mortality rates are dark blue, while those with the least favorable mortality rates are dark red. Consistent quintiles were used for 1990–1992 and 2015–2017 to show changes in the share and distribution of counties in a specific mortality range for each sex and age group (i.e., to show both the share of counties where mortality rates were higher in 2015–2017 versus 1990–1992 and where the rates increased). Rates are age-adjusted by 10-year age group. Small population sizes and death counts resulted in extremely high mortality rates for some counties (e.g., 1 death in a population of 59 results in an age-adjusted mortality rate of 2,272 per 100,000 population). Accordingly, Winsorized binning was used to calculate the rate quintiles, so that counties with mortality rates in the top and bottom 1 percent were removed. On the maps, those counties in the bottom and top 1 percent of mortality are represented in the bottom and top quintiles, respectively. All counties with mortality rates of 0 are included in the bottom quintile. For ease of presentation, figures are limited to the contiguous United States; Alaska and Hawaii are excluded.
SOURCE: Data from https://www.cdc.gov/nchs/nvss/nvss-restricted-data.htm.

2015–2017 across the four levels of metropolitan status: (1) large central metro, (2) large fringe metro, (3) medium/small metro, and (4) nonmetro (see Annex Figure 3-3 in the annex at the end of this chapter). These results confirm that mortality for both younger (ages 25–44) and older (ages 45–64) males and females improved dramatically in large central metros over this period. At the same time, the counties with the highest mortality rates became increasingly concentrated within nonmetros.

Large central metro counties are home to a greater share of the U.S. population relative to less-populated areas; therefore, changes in mortality rates within large central metros are likely to affect a larger share of the

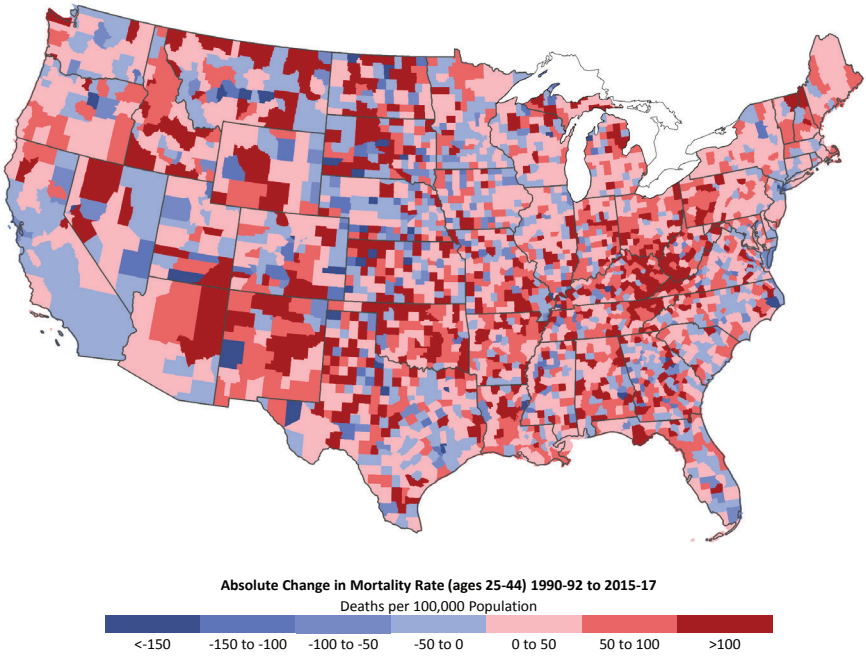


FIGURE 3-7b Absolute change in county-level all-cause mortality rates (deaths per 100,000 population), 1990–1992 to 2015–2017, by U.S. county: Females ages 25–44.
NOTE: Counties with the largest decreases in mortality are dark blue, while those with the largest increases in mortality are dark red. Rates are age-adjusted by 10-year age group. Small population sizes and death counts resulted in extremely high mortality rates for some counties (e.g., 1 death in a population of 59 results in an age-adjusted mortality rate of 2,272 per 100,000 population). For ease of presentation, figures are limited to the contiguous United States; Alaska and Hawaii are excluded.
SOURCE: Data from https://www.cdc.gov/nchs/nvss/nvss-restricted-data.htm.

population compared with changes in small/medium metros or nonmetros. For this reason, the committee also examined the share of the population, by sex and age group, that was living in counties with the highest and lowest mortality rates (based on quintiles) in 1990–1992 and 2015–2017 (Annex Figure 3-4). As a result of the improvements in mortality within large central metros, the share of the population living in low-mortality counties increased over this period, while the share living in high-mortality counties declined. That is, more working-age adults were living in healthier

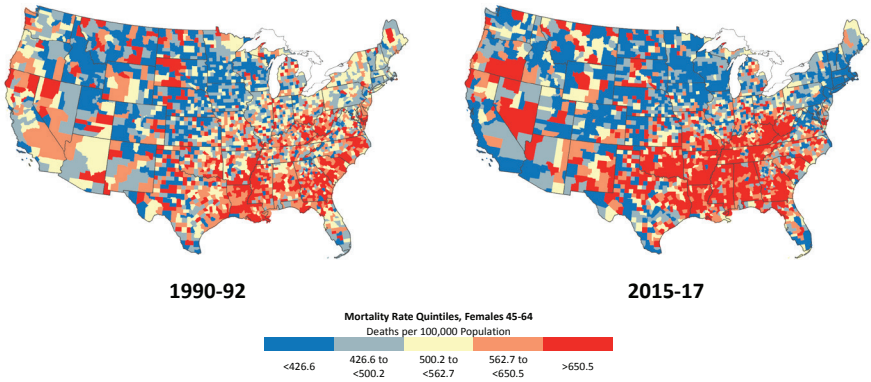


FIGURE 3-8a County-level all-cause mortality rates (deaths per 100,000 population), 1990–1992 and 2015–2017: Females ages 45–64.
NOTE: Mortality rates are shown for 1990–1992 (left) and 2015–2017 (right). Counties are classified into quintiles based on sex- and age-specific mortality rates in 1990–1992. Quintiles with the most favorable mortality rates are dark blue, while those with the least favorable mortality rates are dark red. Consistent quintiles were used for 1990–1992 and 2015–2017 to show changes in the share and distribution of counties in a specific mortality range for each sex and age group (i.e., to show both the share of counties where mortality rates were higher in 2015–2017 versus 1990–1992 and where the rates increased). Rates are age-adjusted by 10-year age group. Small population sizes and death counts resulted in extremely high mortality rates for some counties (e.g., 1 death in a population of 59 results in an age-adjusted mortality rate of 2,272 per 100,000 population). Accordingly, Winsorized binning was used to calculate the rate quintiles, so that counties with mortality rates in the top and bottom 1 percent were removed. On the maps, those counties in the bottom and top 1 percent of mortality are represented in the bottom and top quintiles, respectively. All counties with mortality rates of 0 are included in the bottom quintile. For ease of presentation, figures are limited to the contiguous United States; Alaska and Hawaii are excluded.
SOURCE: Data from https://www.cdc.gov/nchs/nvss/nvss-restricted-data.htm.

counties in 2015–2017 than was the case in the early 1990s. This was especially true for males, reflecting the larger mortality reductions they experienced over the period. Only younger (ages 25–44) females deviated from this pattern; although they experienced a similar increase in the share of the population living within low-mortality counties among these adults, the share living in the counties with the highest mortality also increased, suggesting the emergence of a major geographic divergence (rising inequality) in mortality trends.

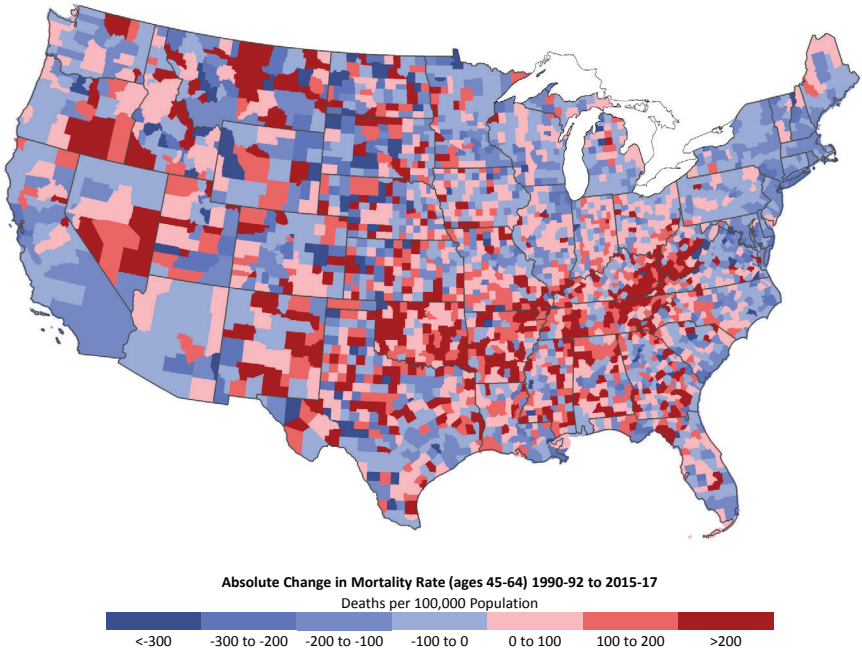


FIGURE 3-8b Absolute change in county-level all-cause mortality rates (deaths per 100,000 population), 1990–1992 to 2015–2017, by U.S. county: Females ages 45–64.
NOTE: Counties with the largest decreases in mortality are dark blue, while those with the largest increases in mortality are dark red. Rates are age-adjusted by 10-year age group. Small population sizes and death counts resulted in extremely high mortality rates for some counties (e.g., 1 death in a population of 59 results in an age-adjusted mortality rate of 2,272 per 100,000 population). For ease of presentation, figures are limited to the contiguous United States; Alaska and Hawaii are excluded.
SOURCE: Data from https://www.cdc.gov/nchs/nvss/nvss-restricted-data.htm.

Summary of Geographic Trends in Mortality

Geographic disparities in working-age mortality rates are large and grew substantially between 1990–1993 and 2015–2017. Mortality rates increased across several regions and states, particularly within the younger age group (25–44), and most glaringly in central Appalachia, New England, the central United States, and parts of the Southwest and Mountain West. The Northeast and West exhibited the most favorable trends, with comparatively large declines in mortality rates among both males and females. Although the South saw some mortality declines, its absolute rates were the

highest in the nation in both 1990–1992 and 2015–2017, and the reductions occurred in only some Southern states. Some of the largest declines in mortality occurred in DC, Maryland, Virginia, North Carolina, Georgia, Florida, and Texas. In contrast, West Virginia, Kentucky, Tennessee, Oklahoma, Arkansas, Alabama, and Mississippi had particularly unfavorable mortality trends. Apart from DC, the largest and most consistent improvements in mortality among working-age adults occurred in states with large populations that are anchored by large central metros, including New York, California, New Jersey, and Illinois.

Mortality trends were not uniform within regions or states, and within-state disparities were often as pronounced as those among states. High- and low-mortality counties were distributed unevenly across different levels of metro status, with greater reductions in mortality seen in large central metro counties and the least favorable trends seen in nonmetro (i.e., rural) counties. These differences were large enough to shift the overall distribution of mortality across metropolitan and nonmetropolitan areas. By 2015–2017, large central metro counties were most likely to have the lowest mortality rates (and least likely to have high mortality rates), while nonmetro counties were most likely to have the highest mortality rates in the United States.

A trend of increasing female mortality spread across the nation, affecting a growing share of U.S. counties. These increases were more widespread than those among males in both age groups, but particularly among those ages 25–44. This was the case by metro status, state, and county. Among adults ages 45–64, large declines in male mortality rates occurred across much of the country, whereas females in this same age group experienced increases in mortality across a large share of U.S. counties. In other words, mortality outcomes among males and females in middle age showed a pattern of increasing divergence, with males experiencing widespread decreases in mortality and more geographically isolated increases, while females, especially those in the younger age group, experienced widespread increases, with geographically concentrated areas of disadvantage, especially in Appalachia and the Southeast and in nonmetros.

SUMMARY

The findings in this chapter reveal that an alarming trend began in the 2010s: mortality rates stagnated or increased among working-age adults in the United States. Particularly concerning is the increase in mortality that occurred among both males and females in the latest period considered here (between 2012–2014 and 2015–2017). This increase marked a reversal from previous trends among working-age Black and Hispanic adults and White males, mortality rates having decreased throughout the 1990s and 2000s

except among males ages 25–34, among whom mortality decreased in the 1990s but stagnated in the 2000s. Among working-age White females, by contrast, this increase followed a long period during the 1990s and 2000s in which mortality rates remained largely unchanged. Only females ages 55–64 experienced decreasing mortality during this period.

These racial/ethnic differences in mortality trends during the period had a significant impact on racial/ethnic disparities in mortality among working-age adults. Black adults had the highest mortality throughout the 1990–2017 period and experienced the largest decreases in mortality, narrowing the White–Black mortality gap over time. Since 2010, however, the increases in mortality among the Black population have arrested this progress. In contrast, working-age Hispanics and AI/ANs generally had lower mortality relative to working-age White adults in 1990; therefore, the large decreases in mortality they experienced during the 1990s and 2000s led to a growing mortality disadvantage for working-age White adults. The committee was unable to examine trends in mortality among working-age AI/ANs; however, data from tribal areas in 1999–2009 demonstrate that AI/ANs living in or near these areas experienced higher mortality than any other racial/ethnic group. Although similar data were not available for the period since 2010, recent research using NVSS death record data suggests that AI/ANs also experienced increasing mortality between 2010 and 2017 (Woolf et al., 2018); however, the authors of this study did not account for race reporting errors, which means these trends should be interpreted with caution.

The findings in this chapter point to a growing mortality disadvantage among less-educated working-age Whites and among working-age Whites, Blacks, and Hispanics living outside of large central metros, particularly those living in nonmetros. By some of these estimates, the gap in life expectancy between White Americans with the highest and lowest levels of education or income in the most recent period grew to as large as 10–12 years. Although less-educated working-age Blacks also experienced higher mortality relative to working-age Blacks with more education, this disadvantage remained largely stable between 1990 and 2017, and little is known about these disparities within other racial/ethnic populations.

In contrast, the growing mortality advantage in large central metros held across working-age Whites, Blacks, and Hispanics, although Whites experienced the largest and most consistent expansion of mortality disparities by metropolitan status over the period. Although working-age mortality was generally higher in large central metros than in less-populated areas in 1990–1993, these areas experienced the largest declines and as a result, had the lowest mortality by 2015–2017. The states that experienced the largest decreases in mortality between 1990–1993 and 2015–2017 were

Southern states with large Black populations and those that were anchored by large central metros that experienced dramatic improvements in mortality over time. Mortality rates increased across several regions and states, particularly within the younger working-age group and most glaringly in central Appalachia, New England, the central United States, and parts of the Southwest and Mountain West. These increases occurred more broadly throughout the country among working-age females, while increases among males were more geographically concentrated. By 2015–2017, the U.S. counties with the highest mortality rates were increasingly concentrated in nonmetros.

It is important to note that these findings are descriptive in nature and cannot establish causal relationships. Based on these analyses, one cannot rule out the possibility that at least some of these trends reflect not changes in mortality per se but other forces that affect the composition of the population, such as immigration and internal migration. The annex to this chapter notes that the changing ethnic composition of the U.S. Asian population could affect trends in mortality within this population over time. Similarly, Black immigrants to the United States from the Caribbean and sub-Saharan Africa have, on average, more education and higher SES relative to the native Black population. Large increases in immigration from these locations have increased the average SES level within the U.S. Black population, leading to more favorable improvements in outcomes for the Black population as a whole than would have occurred if this selective migration had not taken place (Hamilton and Massey, 2019). This means that immigration patterns explain at least some portion of the mortality decreases within the working-age Black population, although the size of their contribution to these changes has not yet been established. A similar phenomenon likely contributes to the growing mortality advantage in large central metros. For example, Preston and Elo (2014) found that most of the relative improvements in mortality that occurred between 2000 and 2010 in New York City relative to the rest of the United states could be explained by the high proportion of immigrants residing in the city compared with the rest of the nation. Thus, although the relative improvements in mortality within large central metros and among non-White working-age adults—particularly the large decreases in mortality among Black adults—are promising, they should be interpreted with caution.

Chapter 4 examines cause-specific mortality to determine the key causes of death that drive these trends in all-cause mortality. The underlying explanations for these key drivers are explored in Part II of this report.

ANNEX 3-1

Mortality Trends Among U.S. Asians/Pacific Islanders and American Indians/Alaska Natives

MORTALITY TRENDS AMONG U.S. ASIANS AND PACIFIC ISLANDERS

The U.S. Asian and Pacific Islander (API) population has experienced rapid growth since the 1965 Immigration and Nationality Act. The Pew Research Center estimated, for example, that between 2000 and 2015, the Asian American population grew by 72 percent, faster than any other racial/ethnic group (Lopez, Ruiz, and Patten, 2017). While there have long been sizable populations of Native Hawaiians, Samoans, and other Pacific Islanders dating back to the U.S. occupation of Hawaii and other Pacific Islands in the late 1800s and concentrated populations of Chinese, Japanese, and Filipino Americans in California and a few other Western states dating back to the 1800s, the past half-century has witnessed greater immigration from an array of Asian countries, leading to both the rapid growth and the diversification of the API population. As of mid-2018, the U.S. Census Bureau estimated that more than 20 million single-race APIs resided in the United States, accounting for more than 6 percent of the total U.S. population (U.S. Census Bureau, 2019). In addition, another 3 million multiracial individuals claim at least one Asian American or Pacific Islander racial category (U.S. Census Bureau, 2019).

Despite the growing size of the API population, valid estimates of age-specific mortality rates and life expectancy for this population have been challenging because of misclassification of race on some death certificates. The construction of U.S. mortality rates is based on race counts from death certificates as the numerator and on population counts by race from U.S. Census data as estimates of the denominator. In the API population, differential recording of race in these two data sources has resulted in underestimates of actual mortality rates. However, in a thorough recent investigation of this problem, Arias, Heron, and Hakes (2016) found that API death rates in the United States grew more accurate between the 1980s–1990s and the 2000s–2010s. Their estimates suggest that recent calculations of API age-specific mortality rates are only 3 percent too low, on average, with greater accuracy documented in states with larger API populations (e.g., California) relative to states with lower percentages of APIs. These authors, who provide corrected age-specific mortality rates for the API population (including the 25–64 age range that is the focus of this report), report that API mortality rates were 50–60 percent lower than those of non-Hispanic

Whites in 2009–2011. This estimate is consistent with scattered published age-specific mortality and life expectancy estimates for the API population, according to which API life expectancy is the highest among all U.S. racial/ethnic groups, exceeding that of non-Hispanic Whites life expectancy by 6–8 years for both women and men (Acciai, Noah, and Firebaugh, 2015; Singh and Hiatt, 2006).

Despite the overall favorable patterns and trends in working-age mortality among APIs, aggregate data for the entire API population mask considerable heterogeneity within this population. First, most of the API population is foreign-born; as of 2015, 73 percent of the Asian American adult population was foreign-born (Lopez, Ruiz, and Patten, 2017). Foreign-born populations from nearly all countries of origin, including those in Asia, have been shown to have lower mortality and higher life expectancy by about 3 years relative to their U.S.-born coethnics for a variety of reasons, but perhaps most important, because of the healthy selection of new immigrants (Dupre, Gu, and Vaupel, 2012; Mehta et al., 2016; Singh and Hiatt, 2006). However, some Asian immigrants came to the United States as refugees or as undocumented persons and are not necessarily selected in terms of favorable health. Thus, estimates of API mortality that do not specify nativity not only mask the higher overall mortality of U.S.-born APIs relative to their foreign-born counterparts but also conflate immigrant groups that arrived under very different conditions.

Second, the API population includes a diverse mix of people who trace their origins to the Pacific Islands, East Asia, Southeast Asia, and the Indian Subcontinent. Among all the different groups included under the API umbrella, the largest subgroups are Chinese, Indian, Filipino, Vietnamese, Korean, and Japanese American, in that order (Lopez, Ruiz, and Patten, 2017). However, the API category also includes dozens of other subgroups, each with its own distinct culture of origin and experience (Iceland, 2017), including those with family origins in U.S. states and territories (e.g., Hawaiians, Samoans, Chamorros) and others from a diverse array of nations and cultures (e.g., Pakistanis, Nepalese, Hmong, Indonesians). Moreover, while some API groups are nearly all or predominantly U.S.-born (e.g., Hawaiians, Japanese Americans), others are 80–95 percent foreign-born (e.g., Burmese, Bhutanese, Nepalese) (Lopez, Ruiz, and Patten, 2017). Accordingly, mortality patterns and trends documented for the overall API population tend to reflect the experiences of the largest subgroups and not necessarily the diverse histories and experiences of dozens of smaller subgroups.

Finally, the API population in the United States is geographically concentrated. Some states (e.g., Montana, Maine) have very small API populations, whereas California is home to more than 30 percent of the nation's API population (Lopez, Ruiz, and Patten, 2017). Other large API

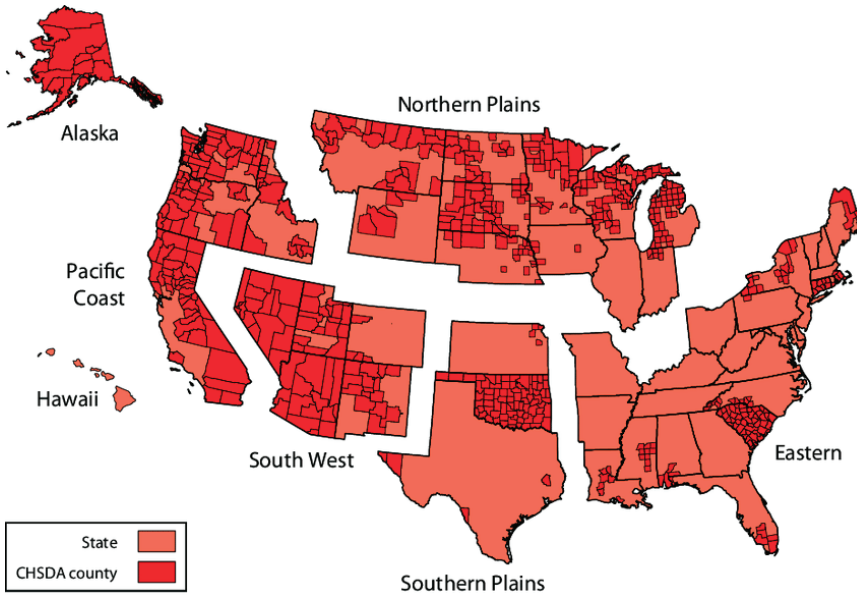
populations live in New York, Texas, New Jersey, Illinois, Washington, Florida, Virginia, Hawaii, and Massachusetts. Thus, the mortality patterns of the API population tend to reflect the health profiles of those states more so than the profiles of states in which this population is poorly represented.

MORTALITY TRENDS AMONG AMERICAN INDIANS/ALASKA NATIVES

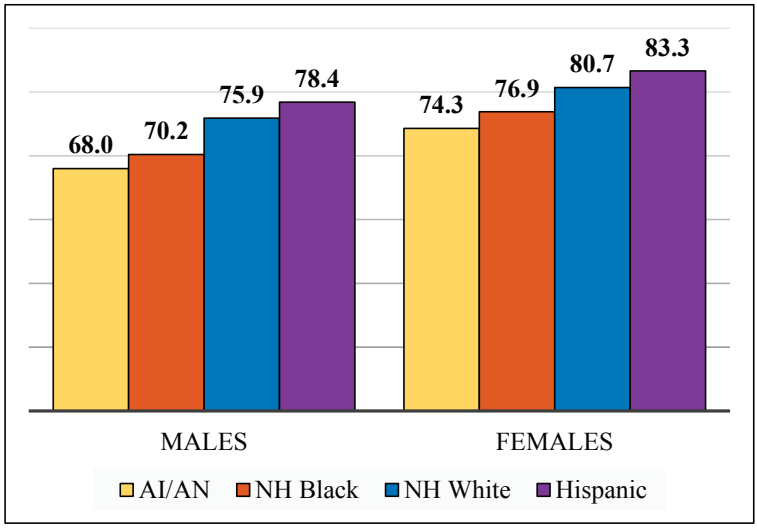
As with APIs, the construction of life tables and other mortality estimates for the American Indian/Alaska Native (AI/AN) population is hindered by the misclassification of AI/AN identity on U.S. death certificates (Anderson, Copeland, and Hayes, 2014). A linkage of Current Population Survey records to the death certificates of the same individuals, for example, revealed that for about 30 percent of self-identified AI/AN individuals, another race was recorded on the death certificate (Arias, Xu, and Jim, 2014). In addition, sample sizes for the AI/AN population in surveys, such as the National Health Interview Survey, that are linked to the National Death Index are too small for the estimation of AI/AN age-specific mortality and life expectancy. Attempts to minimize misclassification biases have restricted mortality estimates to the records of the Indian Health Service registration system that permit the identification of all AI/AN deaths, including those that have been misclassified. Life tables have also been estimated for populations residing in 637 Contract Health Service Delivery Area (CHSDA) counties that contain or are adjacent to tribal lands (Arias, Xu, and Jim, 2014; Espey et al., 2014). The map of CHSDA counties in Annex Figure 3-1 shows their locations in the United States, with the greatest concentrations seen in Alaska, the Southwest, the Pacific Coast, and the Northern Plains. These counties represented 20 percent of all U.S. counties and accounted for 64 percent of the non-Hispanic AI/AN population (Anderson, Copeland, and Hayes, 2014).

The available evidence reveals that mortality among the AI/AN population is much higher than that among non-Hispanic Whites, non-Hispanic Blacks, and Hispanics. In 2007–2009, AI/AN male life expectancy in the CHSDA counties was 7.9 years lower than the national estimate for non-Hispanic White males, 2.2 years lower than for non-Hispanic Black males, and 10.4 years lower than for Hispanic males. The respective figures for females were 6.4 years, 2.6 years, and 9.0 years (Annex Figure 3-2).

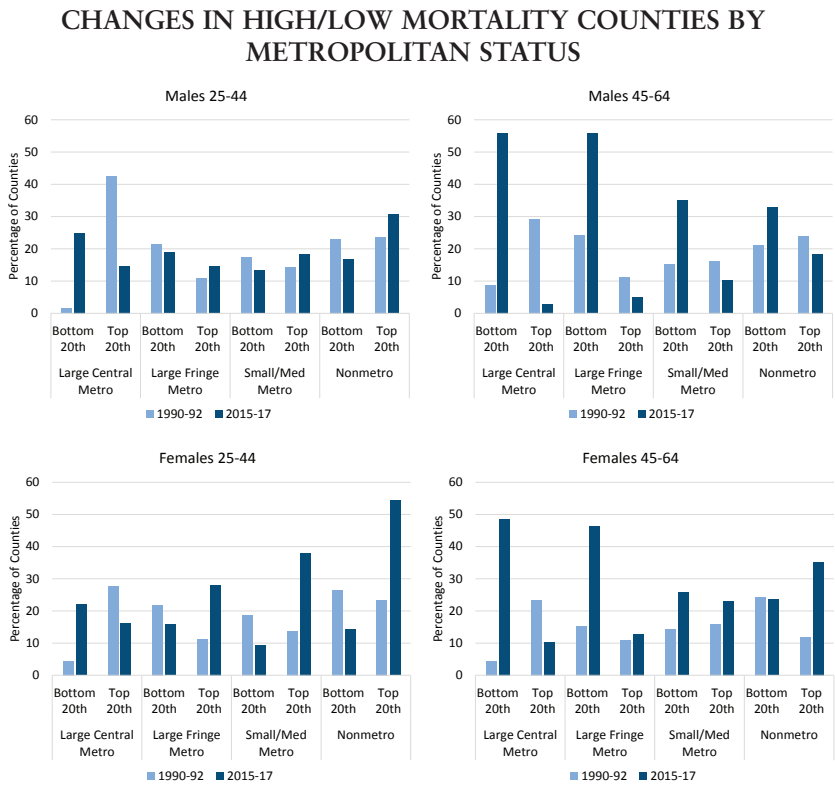
It is important to note that the data presented here cover only AI/ANs living on or near tribal lands or in counties adjacent to those lands. Much less is known about the 36 percent of the AI/AN population that is not covered by these data.



ANNEX FIGURE 3-1 Locations of Contract Health Service Delivery Area (CHSDA) counties across the United States.
SOURCE: Perdue et al. (2014).



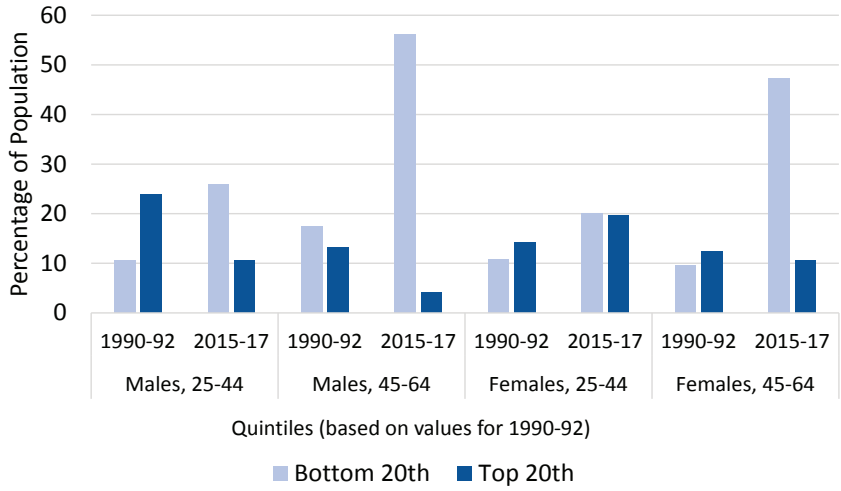
ANNEX FIGURE 3-2 Life expectancy at birth for males and females by race and ethnicity, 2007–2009.
NOTE: American Indian/Alaska Native life expectancy in Contract Health Service Delivery Area (CHSDA) counties in 2007–2009. National non-Hispanic Black, non-Hispanic White, and Hispanic life expectancy in 2008. Rates are age-adjusted.
SOURCE: Data from Arias (2012); Arias, Xu, and Jim (2014).



ANNEX FIGURE 3-3 Percentage of U.S. counties in top and bottom sex- and age-specific county mortality rate quintiles, 1990–1992 and 2015–2017, by metropolitan status.

NOTE: The share of counties in the top and bottom mortality quintiles is shown for 1990–1992 (light blue bars) and 2015–2017 (dark blue bars) by metropolitan area type (large central metro, large fringe metro, small/medium metro, and nonmetro). The data are shown separately for males ages 25–44 (upper left panel), males ages 45–64 (upper right panel), females ages 25–44 (lower left panel), and females ages 45–64 (lower right panel). Counties are classified into quintiles based on sex- and age-specific mortality rates in 1990–1992. Consistent quintiles were used for 1990–1992 and 2015–2017 to show changes in the share and distribution of counties in a specific mortality range for each sex and age group. Rates are age-adjusted by 10-year age group. Small population sizes and death counts resulted in extremely high mortality rates for some counties (e.g., 1 death in a population of 59 results in an age-adjusted mortality rate of 2,272 per 100,000 population). Accordingly, Winsorized binning was used to calculate the rate quintiles, so that counties with mortality rates in the top and bottom 1 percent were removed. Counties in the bottom and top 1 percent of mortality are represented in the bottom and top quintiles, respectively. All counties with mortality rates of 0 are included in the bottom quintile.

SOURCE: Calculations by the committee based on restricted death certificate files from the National Vital Statistics System.



ANNEX FIGURE 3-4 Distribution of U.S. population in counties with highest and lowest mortality rates, 1990–1992 and 2015–2017, based on 1990–1992 county mortality rate quintiles, by sex and age.

NOTE: The light blue bars show the percentage of the population living in counties that fall in the bottom (most favorable) quintile, while the dark blue bars show the percentage living in counties that fall in the top (least favorable) quintile. Counties are classified into quintiles based on sex- and age-specific mortality rates in 1990–1992. Consistent quintiles were used for 1990–1992 and 2015–2017 to show changes in the share and distribution of counties in a specific mortality range for each sex and age group. Rates are age-adjusted by 10-year age group. Small population sizes and death counts resulted in extremely high mortality rates for some counties (e.g., 1 death in a population of 59 results in an age-adjusted mortality rate of 2,272 per 100,000 population). Accordingly, Winsorized binning was used to calculate the rate quintiles, so that counties with mortality rates in the top and bottom 1 percent were removed. Counties in the bottom and top 1 percent of mortality are represented in the bottom and top quintiles, respectively. All counties with mortality rates of 0 are included in the bottom quintile.

SOURCE: Calculations by the committee based on restricted death certificate files from the National Vital Statistics System.

4

U.S. Trends in Cause-Specific Mortality Among Working-Age Adults

The previous chapter establishes that among most working-age adults (ages 25–64), the recent increase in all-cause mortality rates in the United States was due less to a break from earlier trends than to the accumulation and acceleration of long-term slowdowns and reversals of progress in mortality. These all-cause mortality trends demonstrate that there were important differences in where and among whom mortality rates increased between 1990 and 2017; however, they cannot establish which factors contributed to these mortality trends over time. In this chapter, the committee begins to examine how these trends were produced by presenting them by cause of death (cause-specific mortality) to determine which causes contributed most to the overall trends reviewed in Chapter 3.

The comparison of working-age mortality rates in the United States and 16 peer countries presented in Chapter 2 indicates that the U.S. rates exceeded those of the peer countries across a wide range of causes of death. However, the United States also performed better than its peers on some causes of death, such as lung cancer and HIV/AIDS. International disparities in cause-specific mortality are the result of complex systemic interactions among historical economic, demographic, and policy contexts that differ across countries. Although they may point to areas in which policy changes could lead to improvements within the United States, long-standing differences across countries in mortality from some causes of death may not explain recent within-country mortality trends. Knowing whether recent rises in U.S. working-age mortality were due to increases in a small number of specific causes of death or a broad range of causes can provide insight

into the underlying explanations for the recent troubling trends and may help inform policy strategies for combating and reversing these trends.

This chapter presents U.S. cause-specific mortality trends in five sections. The first reviews the leading causes of death in the United States in the first and last periods of the time span addressed by this study (1990–1993 and 2015–2017) and the changes in cause-specific mortality rates over this period. This review identifies which causes contributed most to the changes in all-cause mortality over time. The second section examines the findings from previous research on disparities in cause-specific mortality by socioeconomic status to highlight what is known about the causes of death that have contributed most to the growing socioeconomic disparities in mortality. The third section explores how the changes in each cause of death contributed to changes in all-cause mortality by metropolitan status. The fourth section decomposes the overall changes that occurred between 1990 and 2017 into three time periods (roughly representing decades) to show whether the causes of death that drove overall increases or decreases in mortality during this period represent long-term trends or are a more recent phenomenon. The final section of the chapter summarizes these findings, identifying the specific causes of death that have been the most important drivers of the changes in mortality in the United States since 1990.

An overview of the data and analytical methods used in these analyses is presented in Chapter 5. Causes of death are based on the underlying causes of death identified on death certificates. The underlying causes of death were classified into one of 20 nonoverlapping categories, which are exhaustive of all possible causes. Causes of death were coded according to the International Classification of Diseases, 9th Revision (ICD-9) for 1990–1998 and 10th Revision (ICD-10) for 1999–2017. More information regarding the specific ICD codes included in each of the 20 cause-of-death categories, as well as the process for coding underlying cause of death, is provided in Chapter 5. Two of these categories, noted in the tables that follow, could not be made comparable over the 1990–2017 period because of a change in ICD coding.

Mortality rates are presented separately by sex and age group for non-Hispanic (NH) White (White), NH Black (Black), and Hispanic adults. Although concerns about the quality of race reports on death certificates for the American Indian and Alaska Native (AI/AN) and Asian and Pacific Islander (API) populations prevented the presentation of similar comparisons for these groups, results are included for these groups where possible. When available, this information also was drawn from published research using alternative data sources and therefore may not be directly comparable to the findings presented for the White, Black, and Hispanic populations. As was done for Chapter 3, deaths were pooled across 3-year periods

(1990–1993, 2000–2002, 2009–2011, and 2015–2017), with the exception that the first period (1990–1993) includes 4 years.

The findings presented in this chapter demonstrate that the recent trends in all-cause mortality among working-age adults were the result of the confluence of two important trends: (1) rising mortality from drug poisoning and other causes of death, such as nervous system diseases; hypertensive heart disease; endocrine, nutritional, and metabolic (ENM) diseases; and, among Whites, alcohol use and suicide; and (2) slower progress in lowering mortality from heart diseases and other leading causes of death that drove improvements in all-cause mortality rates before 2010. Mortality due to drug poisoning increased throughout the 1990–2017 period among working-age White, Black, and Hispanic adults of both sexes, with the largest increases occurring among younger (ages 25–44) White adults and older (ages 55–64) Black adults, and was the largest contributor to increases in mortality among all but older Hispanics.

Despite their early onset and alarming magnitude, the large increases in mortality from drug poisoning did not lead to corresponding increases in all-cause mortality until the 2010s among most working-age adults because prior to this period, working-age adults experienced large reductions in mortality from ischemic heart disease and other circulatory diseases and most cancers. The dramatic decreases in mortality from these causes of death more than offset the large increases in mortality from drug poisoning and smaller increases in other causes of death during the 1990s and 2000s. The largest reductions occurred among working-age Blacks, leading to dramatic declines in Black–White mortality disparities during this period. In the 2010s, mortality from ischemic heart disease and other circulatory diseases continued to decrease among working-age Blacks, though at a slower pace, but stalled among Whites and Hispanics.

TRENDS IN U.S. WORKING-AGE MORTALITY BY CAUSE OF DEATH

To identify the key underlying causes of death responsible for the changes in all-cause mortality over the 1990–2017 period, this section presents mortality rates (in deaths per 100,000 population) at the beginning (1990–1993) and end (2015–2017) of the period and the changes in cause-specific mortality rates over the period for working-age White, Black, and Hispanic adults in three age groups—24–44, 45–54, and 55–64. The changes over time are presented in terms of both absolute change in mortality rates (in deaths per 100,000 population) and the percentage contribution of each cause-specific change in mortality to the total increase or decrease in all-cause mortality. The latter percentages were calculated by dividing the increase (or decrease) in the cause-specific mortality rate by

the total increase (or decrease) across all causes of death that increased (or decreased) between 1990–1993 and 2015–2017.

The results presented in this section reveal dramatic reductions in mortality from the most common (leading) causes of death in 1990–1993, including ischemic heart disease and other circulatory diseases,[1] cancers (excluding liver cancer), and HIV/AIDS. These improvements occurred among both sexes and each of the three racial/ethnic groups the committee examined. However, mortality also increased across a wide range of causes of death, offsetting some, and in some cases all, of these gains. Although there were racial/ethnic differences in which causes of death increased over the period, mortality due to drug poisoning and diseases of the nervous system[2] increased across all working-age adults, regardless of sex, age group, or race and ethnicity.

Although working-age Black adults maintained the highest mortality throughout the period, they also experienced the largest decreases in mortality across the widest range of causes of death, narrowing the racial gap in mortality between the 1990–1993 and 2009–2011 periods. Working-age Hispanic adults also experienced (comparatively) large reductions in mortality across many causes of death. In contrast, working-age White adults experienced increases in mortality across the widest range of causes of death, and for this reason had higher mortality than working-age Hispanic adults at the end of the period.

Although similarly detailed cause-specific trends in mortality for APIs and AI/ANs could not be included here because of concerns about the quality of racial data on death certificates, Box 4-1 (Asians and Pacific Islanders) and Box 4-2 (American Indians and Alaska Natives) briefly review cause-specific mortality for these populations.

[1] "Other diseases of the circulatory system" include all circulatory diseases besides ischemic heart disease and hypertensive heart disease. Major contributors to working-age deaths in this category include stroke, cardiomyopathy, congestive heart failure, intracerebral hemorrhage, cardiac arrest, and pulmonary embolism. Each of these causes contributed to at least 5 percent of working-age deaths in this category, 1999–2017.

[2] Diseases of the nervous system include, but are not limited to, meningitis and other inflammatory diseases, encephalitis, myelitis, and encephalomyelitis, Huntington's disease, spinal muscular atrophy and related syndromes, Parkinson's disease, movement disorders, Alzheimer's disease and other degenerative diseases of the nervous system, multiple sclerosis and other demyelinating diseases of the central nervous system, epilepsy and other episodic and paroxysmal disorders, sleep disorders, cerebral palsy and other paralytic syndromes, and other disorders of the brain. In 2017, the most common causes of death within the working-age population were anoxic brain damage, not elsewhere classified; motor neuron disease; multiple sclerosis; infantile cerebral palsy; and Alzheimer's disease (CDC 2020b).

BOX 4-1
Trends in Cause-Specific Mortality Among Asians and Pacific Islanders (APIs)

As noted in Chapter 3 and discussed in greater detail in Chapter 5, the development of valid estimates of age-specific mortality rates has been challenging for the API population because of misclassification of race on some death certificates. As a result, the committee's detailed original analyses of cause-specific mortality trends for APIs are not presented in this report. Instead, the trends we observed for this group are briefly summarized here.

As was the case with Whites, Blacks, and Hispanics, drug poisoning was the largest contributor to increases in all-cause mortality among APIs during 1990–2017 for males and females in all age groups except males ages 55–64, for whom increased mortality from endocrine and metabolic diseases surpassed the increase in deaths from drug poisoning. These increases were small, however, particularly when compared with those experienced by Whites. Besides drug poisoning, only a handful of other causes increased by more than 1 death per 100,000 between 1990 and 2017, and these increases were very small. Among males, they included endocrine, nutritional, and metabolic diseases; hypertensive heart disease; mental and behavioral disorders (only among those ages 45–64); diseases of the nervous system (only among those ages 45–64); diseases of the genitourinary system (only among those ages 45–64); alcohol-induced causes (only among those ages 45–64); and suicide (only among those ages 24–54). Among females, the only cause of death besides drug poisoning to increase by more than 1 death per 100,000 over the period was diseases of the nervous system (only among those ages 45–64). At all ages, any increases observed over the study period were more than offset by mortality declines from other causes of death, yielding declines in all-cause mortality between 1990 and 2017 for both males and females in all age groups.

For more detail on the challenges with mortality data for APIs and important considerations for readers in interpreting trends, see the annex to Chapter 3 and Chapter 5.

Non-Hispanic White Adults

Among working-age White males and females (Table 4-1), an important takeaway is that mortality rates from several causes of death—including liver cancer; ENM diseases;[3] hypertensive heart disease; drug poisoning;

[3]ENM diseases include, but are not limited to, diabetes mellitus, disorders of the thyroid gland, hypoglycemia, disorders of other endocrine glands, malnutrition, obesity, disorders of lipoprotein metabolism and other lipidemias, cystic fibrosis, amyloidosis, and other metabolic disorders. In 2017, the most common cause of death within this category was diabetes mellitus, which was responsible for most of the deaths in this category, followed by obesity and hyperlipidaemia (CDC, 2020b). The committee initially examined trends in diabetes and obesity separately from those for other ENM diseases but decided that these trends did not differ substantively from those of the rest of the causes within this category.

BOX 4-2
Trends in Cause-Specific Mortality Among American Indians and Alaska Natives (AI/ANs)

There are considerable data quality concerns related to calculating cause-specific mortality rates for AI/ANs because of errors in race reports on vital statistics records (see the annex to Chapter 3 and the discussion of the quality of death certificate data in Chapter 5 for more information). Given these data concerns, the committee's original analyses on trends for this group are not presented in this report.

The table below shows the 10 leading causes of death for the AI/AN population compared with Whites, Blacks, and Hispanics in 2017. Because these results are based on vital statistics reports that do not correct for errors in race reporting, these tables do not include the estimated mortality rates for these populations; however, other research has demonstrated that the AI/AN population has the highest mortality rates for more causes of death relative to any racial/ethnic group (Espey et al., 2014; Sancar, Abbasi, and Bucher, 2017).

AI/AN individuals are more likely to die from diabetes, chronic liver disease, and suicide than are Whites, Blacks, and Hispanics. These cause-of-death rankings are very similar to those found in the 1999–2009 period for AI/AN populations living in 637 Contract Health Service Delivery Areas using data that were corrected for misclassification of AI/AN identity on death certificates (Annex Figure 4-1). Additional efforts are needed to better document the health and mortality of all AI/AN individuals and to address the pressing needs of this important population subgroup.

10 Leading Causes of Death Among the American Indian and Alaska Native (AI/AN) Population Compared with Whites, Blacks, and Hispanics, 2017

Cause of Death	AI/AN	White	Black	Hispanic
Heart diseases	1	1	1	2
Malignant neoplasms	2	2	2	1
Accidents (unintentional injuries)	3	4	3	3
Diabetes mellitus	4	7	5	5
Chronic liver disease and cirrhosis	5	11	14	7
Chronic lower respiratory disease	6	3	6	8
Cerebrovascular diseases	7	5	4	4
Suicide	8	9	16	9
Influenza and pneumonia	9	8	12	11
Alzheimer's disease	10	6	9	6

SOURCE: Heron (2019).

alcohol-induced causes; suicide;[4] mental and behavioral disorders; diseases of the nervous system; and diseases of the genitourinary system—increased in all three age groups over the study period. Drug poisoning was responsible for the largest mortality increases by far, particularly among younger White adults (ages 25–44). Even so, the consistency of increases in mortality across such a wide range of causes of death that span multiple disease categories and body systems is cause for concern and suggests that recent increases in working-age mortality rates go beyond increases in drug poisoning.

Among older working-age Whites (ages 45–64), these mortality increases were largely offset over the period by substantial decreases in the two leading causes of death in 1990–1993: ischemic heart disease and other circulatory diseases, and cancer. Because mortality from these two causes often results from long-term exposures and chronic disease, it plays a less prominent (though still important) role in mortality among many younger working-age adults (ages 25–44), who therefore experienced much smaller improvements in mortality from these causes over the period. In fact, among younger working-age White women, mortality due to ischemic heart disease and other circulatory diseases actually increased slightly over the period,[5] although their mortality from cancers other than liver cancer decreased. Moreover, while mortality from these causes declined between 1990–1993 and 2015–2017, their importance as two of the leading causes of death held over the period. In contrast, HIV/AIDS was the leading cause of death among working-age White men in 1990–1993, but by 2015–2017, mortality rates from this cause had declined by more than 95 percent so that it ranked 19th out of the 20 causes of death.

Previous studies have grouped mortality due to drug poisoning, alcohol, and suicide together and found that this set of causes was responsible for the largest increases in mortality over the period among working-age Whites (Case and Deaton, 2015, 2017). The committee made this same finding: Taken together, these causes of death were responsible for between one-third and more than 90 percent of the total increase in mortality that occurred between 1990–1993 and 2015–2017, depending on age group and sex. Nonetheless, drug poisoning was the single largest contributor to the increase in all-cause mortality rates among White males and females in all three age groups, with its contribution to the increase in all-cause mortality over the period ranging from a low of 17.5 percent among males ages

[4]In this analysis, the category of suicide excludes suicides due to drug poisoning, which are classified with other drug poisoning deaths.

[5]Among younger working-age White females, mortality due to ischemic heart disease was very low and did not change over the period. This increase was driven by increasing mortality due to other circulatory diseases.

55–64 to a high of 73.7 percent among males ages 25–44. Moreover, in 2015–2017, drug poisoning was the top cause of death among White males and females ages 25–44, and on its own, the increase in drug poisoning completely offset the declines in other causes of death that occurred among White females in this age group.

Suicide was the second largest contributor to the increase in all-cause mortality among White males ages 25–44 (12.8%), males ages 45–54 (11.1%), and females ages 25–44 (7.0%). Along with drug poisoning and suicide, Case and Deaton (2015, 2017) highlight the important contribution of alcohol-induced causes to increasing midlife mortality rates among Whites. The committee likewise found that deaths from alcohol-induced causes increased over this period among White males and females in all three age groups, and that these increases were larger among those ages 45–64 relative to the younger age group. This delayed onset is consistent with the clinical course of alcoholic liver disease (the largest contributor to mortality in this category), which develops over time and often results from years of chronic alcohol consumption. Among White females ages 45–54, deaths from alcohol-induced causes were the second largest contributor to the increase in all-cause mortality (representing 9.3% of the total increase).

The committee considered rates of mortality from mental and behavioral disorders in conjunction with those from drug poisoning and alcohol-induced causes because most deaths attributed to mental and behavioral disorders involve drugs or alcohol.[6] Mortality from mental and behavioral disorders also increased among males and females in all three age groups. These mortality increases were generally similar in magnitude to those due to alcohol-induced causes. Together, the causes of death in the substance use and mental health category made an overwhelming contribution to the increases in mortality among both males and females in all three age groups, contributing to 91 percent of the increase for males ages 25–44, 61 percent for males ages 45–54, 35 percent for males ages 55–65, 73 percent for females ages 25–44, 54 percent for females ages 45–54, and 35 percent for females ages 55–64.

Other significant contributors to the overall increase in mortality among working-age White adults were cardiometabolic diseases. Although combined mortality from all of these diseases decreased overall because of large decreases in mortality from ischemic heart disease and other circulatory system diseases, mortality from hypertensive heart disease and ENM diseases increased over the period among both males and females in all three age groups. These causes of death made important contributions to the increase in all-cause mortality in the 45–54 and 55–64 age groups, together

[6]More detail about the relationship between mental and behavioral disorders and drug poisoning and/or alcohol-induced causes is presented later in the chapter.

TABLE 4-1 Cause-Specific Mortality (deaths per 100,000 population), 1990–1993 and 2015–2017: Non-Hispanic White Adults Ages 25–64

	Non-Hispanic White Males											
Age Group	25–44				45–54				55–64			
All-Cause Mortality Rate 1990–1993	212.29				520.80				1345.21			
All-Cause Mortality Rate 2015–2017	222.62				493.39				1085.81			
Change 1990–1993 to 2015–2017	10.33				–27.41				–259.40			
(Change as % of 1990–1993 mortality)	4.87				–5.26				–19.28			
	Mortality Rate		Change		Mortality Rate		Change		Mortality Rate		Change	
Cause of Death	1990–1993	2015–2017	Abs Chg	% of Total +/– Chg	1990–1993	2015–2017	Abs Chg	% of Total +/– Chg	1990–1993	2015–2017	Abs Chg	% of Total +/– Chg
Infectious and Parasitic Diseases												
HIV/AIDS	35.83	1.05	–34.78	–56.7	25.76	3.81	–21.95	–15.4	10.16	3.49	–6.67	–1.6
Non-HIV/AIDS	3.62	2.57	–1.05	–1.7	6.54	11.03	4.49	3.9	13.68	29.26	15.59	9.5
Cancers												
Liver Cancer	0.49	0.52	0.03	0.0	2.54	5.03	2.50	2.2	8.75	25.79	17.04	10.3
Lung Cancer	4.34	1.61	–2.73	–4.5	52.60	21.22	–31.37	–22.0	198.91	90.90	–108.02	–25.5

continued

TABLE 4-1 Continued

Cause of Death	Mortality Rate 1990–1993	Mortality Rate 2015–2017	Change Abs Chg	Change % of Total +/– Chg	Mortality Rate 1990–1993	Mortality Rate 2015–2017	Change Abs Chg	Change % of Total +/– Chg	Mortality Rate 1990–1993	Mortality Rate 2015–2017	Change Abs Chg	Change % of Total +/– Chg
All Other Cancers	20.90	15.55	–5.35	–8.7	91.52	70.23	–21.29	–14.9	271.79	206.16	–65.63	–15.5
Cardio and Metabolic Diseases												
Endocrine, Nutritional, & Metabolic	5.12	7.00	1.88	2.6	14.95	24.93	9.97	8.6	36.03	56.26	20.23	12.3
Hypertensive Heart Disease	1.08	3.86	2.78	3.9	5.61	16.00	10.39	9.0	15.20	32.61	17.40	10.6
Ischemic & Other Circulatory System	30.76	24.52	–6.25	–10.2	177.57	110.92	–66.65	–46.7	517.49	279.65	–237.84	–56.1
Substance Use and Mental Health												
Drug Poisoning	9.55	62.43	52.88	73.7	4.39	47.73	43.34	37.6	2.58	31.42	28.84	17.5
Alcohol-Induced*	4.81	6.70	1.89	2.6	13.33	21.83	8.50	7.4	20.73	31.58	10.85	6.6
Suicide	23.09	32.30	9.21	12.8	22.36	35.21	12.85	11.1	24.75	32.79	8.03	4.9
Mental & Behavioral Disorders	3.84	5.10	1.26	1.8	7.35	12.69	5.34	4.6	11.37	21.01	9.64	5.9

Other Body System Diseases												
Nervous System	3.11	4.63	1.53	2.1	7.01	11.98	4.98	4.3	15.71	28.44	12.74	7.7
Genitourinary System	1.01	1.29	0.28	0.4	3.18	5.62	2.44	2.1	10.42	16.02	5.59	3.4
Respiratory System	4.49	4.23	–0.27	–0.4	18.32	20.26	1.94	1.7	85.70	81.10	–4.60	–1.1
Digestive System*	5.50	4.59	–0.91	–1.5	17.23	19.94	2.72	2.4	39.05	44.79	5.74	3.5
Other Causes of Death												
Homicide	8.12	5.66	–2.46	–4.0	6.26	4.78	–1.48	–1.0	4.76	3.42	–1.34	–0.3
Transport Accidents	27.21	22.22	–4.99	–8.1	20.37	22.13	1.77	1.5	20.00	23.30	3.30	2.0
Other External Causes of Death	12.69	10.43	–2.26	–3.7	14.19	15.99	1.80	1.6	19.63	25.58	5.95	3.6
All Other Causes of Death	6.72	6.38	–0.34	–0.6	9.74	12.04	2.31	2.0	18.49	22.24	3.76	2.3
Total Change: Increase (+)				71.74				115.33				164.70
Total Change: Decrease (–)				–61.40				–142.74				–424.09

continued

TABLE 4-1 Continued

	Non-Hispanic White Females											
Age Group	25–44				45–54				55–64			
All-Cause Mortality Rate 1990–1993	88.93				295.15				767.20			
All-Cause Mortality Rate 2015–2017	121.90				314.97				659.91			
Change 1990–1993 to 2015–2017	32.97				19.82				–107.29			
(Change as % of 1990–1993 mortality)	37.07				6.72				–13.98			
	Mortality Rate		Change		Mortality Rate		Change		Mortality Rate		Change	
Cause of Death	1990–1993	2015–2017	Abs Chg	% of Total +/– Chg	1990–1993	2015–2017	Abs Chg	% of Total +/– Chg	1990–1993	2015–2017	Abs Chg	% of Total +/– Chg
Infectious and Parasitic Diseases												
HIV/AIDS	2.09	0.37	–1.72	–13.3	1.07	0.71	–0.36	–0.6	0.67	0.58	–0.08	0.0
Non-HIV/AIDS	1.43	2.41	0.98	2.1	3.79	8.45	4.66	5.8	9.40	19.68	10.28	11.0
Cancers												
Liver Cancer	0.24	0.39	0.14	0.3	1.37	2.14	0.77	1.0	3.79	7.52	3.74	4.0
Lung Cancer	3.07	1.48	–1.59	–12.3	33.42	19.34	–14.08	–23.1	104.57	67.85	–36.72	–18.3
All Other Cancers	26.62	19.19	–7.43	–57.6	111.06	76.40	–34.67	–57.0	248.27	173.33	–74.95	–37.3

Cardio and Metabolic Diseases												
Endocrine, Nutritional, & Metabolic	3.06	4.92	1.86	4.0	10.04	14.76	4.72	5.9	28.45	32.24	3.78	4.0
Hypertensive Heart Disease	0.33	1.65	1.32	2.9	2.34	7.30	4.96	6.1	7.93	15.27	7.34	7.8
Ischemic & Other Circulatory System	11.88	13.09	1.21	2.6	60.58	49.74	–10.84	–17.8	207.31	120.51	–86.80	–43.2
Substance Use and Mental Health												
Drug Poisoning	4.10	30.36	26.26	57.2	4.14	33.26	29.12	36.1	3.45	22.56	19.12	20.4
Alcohol-Induced*	1.71	3.83	2.12	4.6	4.21	11.73	7.52	9.3	6.52	12.65	6.13	6.5
Suicide	4.64	7.85	3.21	7.0	5.48	9.10	3.62	4.5	5.16	7.59	2.44	2.6
Mental & Behavioral Disorders	1.24	3.08	1.84	4.0	2.25	5.92	3.67	4.5	3.81	8.86	5.06	5.4
Other Body System Diseases												
Nervous System	2.28	3.53	1.25	2.7	6.03	9.70	3.68	4.6	12.82	24.01	11.20	12.0
Genitourinary System	0.71	1.18	0.47	1.0	2.54	4.51	1.97	2.4	7.80	12.16	4.37	4.7
Respiratory System	3.03	4.18	1.15	2.5	14.45	20.87	6.41	7.9	60.25	68.84	8.58	9.2
Digestive System*	2.68	3.75	1.06	2.3	9.47	13.70	4.22	5.2	24.41	29.13	4.72	5.0
Other Causes of Death												
Homicide	3.36	2.59	–0.77	–5.9	2.48	2.10	–0.38	–0.6	1.72	1.57	–0.16	–0.1
Transport Accidents	9.60	8.22	–1.39	–10.7	8.54	8.04	–0.51	–0.8	9.29	7.12	–2.17	–1.1

continued

TABLE 4-1 Continued

	Mortality Rate		Change		Mortality Rate		Change		Mortality Rate		Change	
Other External Causes of Death	2.59	3.49	0.89	1.9	4.15	6.64	2.49	3.1	6.97	11.29	4.32	4.6
All Other Causes of Death	4.26	6.38	2.12	4.6	7.70	10.55	2.85	3.5	14.62	17.14	2.52	2.7
Total Change: Increase (+)				45.87				80.66				93.59
Total Change: Decrease (–)				–12.90				–60.83				–200.88

*Changes in the International Classification of Diseases (ICD)-10 code for diseases of the digestive system in 2006 affected the comparability of both digestive system deaths and alcohol-induced deaths before and after that year. More information is provided in Chapter 5.
NOTE: The top 3 causes of death for each age group in each period are highlighted in **bolded red** text.
Light orange highlights indicate an absolute increase in the overall mortality rate of ≥5 deaths/100,000 population.
Dark orange highlights indicate that a cause of death is responsible for ≥10 percent of the total increase in mortality.
Light green highlights indicate an absolute decrease in the overall mortality rate of ≥5 deaths/100,000 population.
Dark green highlights indicate that a cause of death is responsible for ≥10 percent of the total decrease in mortality.
The table shows mortality rates and change in all-cause and cause-specific mortality rates among non-Hispanic White working-age adults by age group (25–44, 45–54, and 55–64) for males (upper table) and females (lower table). Changes in mortality are presented as both the absolute change in mortality rates and the percentage of the total increase (or decrease) in mortality. Mortality rates within each age group are age-adjusted by single-year of age to match the age distribution of the U.S. population in 2000 in order to improve comparability over time. These causes of death are exhaustive of all underlying cause-of-death codes and are based on the ICD-9 (1990–1998) and ICD-10 (1999–2017) codes. More information about the classification of causes of death can be found in Chapter 5.
SOURCE: Data from https://www.cdc.gov/nchs/nvss/nvss-restricted-data.htm.

contributing 18 percent of the increase for males ages 45–54, 13 percent for males ages 55–64, and 12 percent each for females ages 45–54 and 55–64. Other large contributors to the increase in all-cause mortality included liver cancer for males ages 55–64 (representing 10% of the increase), non-HIV/AIDS infectious and parasitic diseases among females ages 55–64 (11% of the increase), and diseases of the nervous system among females ages 55–64 (12% of the increase).

These widespread mortality increases were offset by remarkable improvements in mortality rates from HIV/AIDS; lung and other cancers,[7] excluding liver cancer; and ischemic heart disease and other diseases of the circulatory system. Declines in mortality from HIV/AIDS contributed 57 percent of the decline in all-cause mortality among males ages 25–44, 15 percent among males ages 45–54, and 13 percent among females ages 25–44. Declines in mortality due to lung and other cancers contributed 13 percent of the overall decline in all-cause mortality among males ages 25–44, 37 percent among males ages 45–54, 41 percent among males ages 55–64, 70 percent among females ages 25–44, 80 percent among females ages 45–54, and 56 percent among females ages 55–64. Reductions in mortality from ischemic heart disease and other circulatory system diseases also contributed substantially to declines in all-cause mortality among all sex/age groups except females ages 25–44 (who already had comparatively low rates of mortality from this cause). Ischemic heart disease and other circulatory system diseases represented the single largest contributor to the decline in all-cause mortality among males ages 45–54 (47%), males ages 55–64 (56%), and females ages 55–64 (43%).

Working-Age Non-Hispanic Black Adults

At the beginning of the period, working-age cause-specific mortality was higher among Blacks than among Whites for nearly all causes of death (Table 4-2)—in many cases, significantly higher. Only suicide rates were consistently higher among working-age White adults, although older White females (ages 45–64) also had higher mortality from drug poisoning relative to similarly-ages Black females. In contrast to changes in mortality rates between 1990–1993 and 2015–2017 among working-age White adults, who saw widespread increases, the changes among working-age Blacks were characterized by dramatic decreases in mortality across a wide range of causes. Declines were larger (both absolutely and as a percentage) among Black males than among Black females, with the largest decreases occurring for mortality due to ischemic heart disease and other circulatory

[7] "Other cancers" include all cancers besides liver and lung. Large declines in breast cancer are responsible for most of the decline in mortality rates from "other cancers" among females.

system diseases and cancers other than liver cancer. Younger working-age Blacks also experienced large decreases in homicide rates and mortality from HIV/AIDS. Despite these massive declines in Black mortality rates, however, mortality among Black adults remained much higher than that among White adults for most causes of death in 2015–2017.

Despite the above improvements, Black working-age mortality rates did increase for a small number of causes of death, notably drug poisoning and diseases of the nervous system. Although drug poisoning mortality also increased among Black adults, these increases were generally smaller than those among White adults, except among older Black males (ages 55–64). Unlike working-age Whites, working-age Blacks did not experience increases in mortality due to alcohol-induced causes, suicide, or mental and behavioral disorders. For this reason, by 2015–2017, working-age Whites generally had higher rates of mortality from alcohol-induced causes, suicide, and mental and behavioral disorders relative to similarly ages Black adults. Among Black males and younger Black females, mortality due to ENM diseases and hypertensive heart disease increased. Overall, when cause-specific mortality increased, working-age Blacks experienced smaller mortality increases compared with working-age Whites, and these increases were more than offset by large reductions in mortality across multiple causes of death.

In all three age groups, mortality from drug poisoning increased among Black adults and was the single largest contributor to increases in all-cause mortality, just as was the case among working-age Whites. However, the increase in mortality due to drug poisoning was greater among working-age Whites than among working-age Blacks, with the exception of older males (ages 55–64). For this reason, the age patterns of these increases differed among working-age Blacks. Among White males, drug poisoning mortality rates increased most among younger adults (ages 25–44) and least among older adults (ages 55–64), but this pattern was reversed among Black males, so that older Black males (ages 55–64) experienced the largest increase, while younger Black males (ages 25–44) experienced the smallest increase. As with White females, the highest mortality rate and largest mortality increase for drug poisoning among Black females was in the middle age group (45–54). Mortality from alcohol-induced causes, suicide, and mental and behavioral disorders declined among Black adults overall between 1990–1993 and 2015–2017.

ENM diseases and hypertensive heart disease were also important contributors to increases in all-cause mortality among Black males and younger Black females, together contributing 13–35 percent of the increase. While increases in cause-specific mortality were more limited among working-age Blacks, three other causes of death—diseases of the nervous system, liver cancer, and non-HIV/AIDS infectious and parasitic diseases—increased

substantially for them over the period, although only the first of these causes increased among both sexes and all age groups. The increases in mortality from diseases of the nervous system among Black adults were similar to those among Whites: they were larger among females and increased with age. Among Black females ages 45–54, diseases of the nervous system represented the second largest increase in mortality, after drug poisoning. Non-HIV/AIDS infectious and parasitic diseases and liver cancer were important contributors to the total increase in mortality for Black adults ages 55–64, the former contributing 9.7 percent and 15.4 percent of the increase in all-cause mortality for males and females, respectively, and the latter 24.4 percent and 12 percent of the increase for males and females, respectively. After drug poisoning, liver cancer was the second largest contributor to the increase in mortality among males ages 55–64, and this increase in mortality from liver cancer was larger among Black males ages 55–64 than among similarly ages White males.

Relative to working-age Whites, working-age Blacks experienced declines in mortality from a wider range of causes, including HIV/AIDS (except Black females ages 55–64), lung and other nonliver cancers, ischemic heart disease and other diseases of the circulatory system, alcohol-induced causes, mental and behavioral disorders, diseases of the respiratory system, diseases of the digestive system, homicide, transport injuries, other external causes of death, and the category of all other causes. As was true among Whites, the largest improvements in mortality for working-age Blacks involved ischemic heart disease and other circulatory system diseases, lung and other nonliver cancers, and HIV/AIDS. The declines in mortality from these causes of death were remarkable and contributed to a major reduction in the Black–White gap in all-cause mortality. For each of these causes of death, Black adults experienced much larger declines relative to White adults, although their rates began at much higher starting points and remained higher in 2015–2017. The slight increase in HIV/AIDS mortality among Black females ages 55–64 is concerning; this is the only group that experienced an increase in mortality from this cause over the study period.

Hispanic Adults

Although cause-specific mortality was much lower among working-age Hispanics than among working-age Blacks over the period, their trends over time, while of smaller magnitude, were similar to those among Blacks (Table 4-3). Hispanic adults experienced large reductions in mortality across a wide range of causes of death, with the largest improvements seen in ischemic heart disease and other circulatory diseases, cancers other than liver cancer, HIV/AIDS, and homicide. These improvements more than

TABLE 4-2 Cause-Specific Mortality (deaths per 100,000 population), 1990–1993 and 2015–2017: Non-Hispanic Black Adults Ages 25–64

	Non-Hispanic Black Males									
Age Group	25–44				45–54				55–64	
All-Cause Mortality Rate 1990–1993	582.82				1236.32				2466.82	
All-Cause Mortality Rate 2015–2017	319.86				697.04				1669.83	
Change 1990–1993 to 2015–2017	–262.96				–539.28				–796.99	
(Change as % of 1990–1993 mortality)	–45.12				–43.62				–32.31	
	Mortality Rate		Change		Mortality Rate		Change		Mortality Rate		Change	
Cause of Death	1990–1993	2015–2017	Abs Chg	% of Total +/– Chg	1990–1993	2015–2017	Abs Chg	% of Total +/– Chg	1990–1993	2015–2017	Abs Chg	% of Total +/– Chg
Infectious and Parasitic Diseases												
HIV/AIDS	134.82	10.16	–124.66	–43.9	98.09	20.96	–77.13	–13.2	44.75	30.07	–14.68	–1.6
Non-HIV/AIDS	16.23	4.78	–11.46	–4.0	30.71	18.09	–12.62	–2.2	45.06	54.76	9.70	7.7
Cancers												
Liver Cancer	1.84	1.18	–0.66	–0.2	7.90	7.51	–0.39	–0.1	18.95	49.60	30.65	24.4
Lung Cancer	10.43	2.36	–8.07	–2.8	108.93	23.84	–85.10	–14.6	320.63	119.64	–200.99	–21.8
All Other Cancers	31.45	17.42	–14.03	–4.9	176.23	85.03	–91.21	–15.6	461.08	274.10	–186.98	–20.3

Cardio and Metabolic Diseases												
Endocrine, Nutritional, & Metabolic	13.04	15.39	2.35	11.2	43.04	46.95	3.91	8.7	92.87	103.79	10.92	8.7
Hypertensive Heart Disease	9.98	14.97	5.00	23.8	46.34	48.20	1.87	4.1	91.66	100.22	8.57	6.8
Ischemic & Other Circulatory System	76.27	49.51	–26.76	–9.4	357.62	188.32	–169.31	–29.0	869.66	474.12	–395.54	–42.9
Substance Use and Mental Health												
Drug Poisoning	19.45	32.54	13.09	62.4	12.92	48.91	36.00	79.8	4.79	55.90	51.11	40.7
Alcohol–Induced*	13.17	3.26	–9.92	–3.5	36.81	12.04	–24.77	–4.2	41.88	25.36	–16.53	–1.8
Suicide	17.11	15.67	–1.44	–0.5	12.65	10.64	–2.00	–0.3	10.50	8.17	–2.33	–0.3
Mental & Behavioral Disorders	14.37	3.53	–10.84	–3.8	32.90	10.69	–22.21	–3.8	38.14	26.84	–11.30	–1.2
Other Body System Diseases												
Nervous System	6.82	7.36	0.54	2.6	12.75	16.07	3.32	7.4	21.91	33.96	12.05	9.6
Genitourinary System	7.59	4.86	–2.73	–1.0	20.19	18.74	–1.45	–0.2	47.87	50.37	2.50	2.0
Respiratory System	19.29	8.52	–10.77	–3.8	52.38	27.31	–25.07	–4.3	136.43	92.21	–44.22	–4.8
Digestive System*	15.93	4.79	–11.13	–3.9	45.89	19.39	–26.50	–4.5	72.29	54.58	–17.71	–1.9
Other Causes of Death												
Homicide	92.84	69.24	–23.59	–8.3	45.41	27.69	–17.72	–3.0	29.74	17.05	–12.69	–1.4
Transport Accidents	34.30	30.37	–3.93	–1.4	31.78	27.25	–4.53	–0.8	31.16	29.09	–2.07	–0.2
Other External Causes of Death	23.88	11.50	–12.38	–4.4	31.46	17.58	–13.88	–2.4	43.80	30.69	–13.11	–1.4
All Other Causes of Death	24.03	12.46	–11.57	–4.1	32.33	21.84	–10.49	–1.8	43.67	39.35	–4.32	–0.5

continued

TABLE 4-2 Continued

Total Change: Increase (+)	20.98	45.10	125.49
Total Change: Decrease (–)	–283.93	–584.38	–922.47

	Non-Hispanic Black Females		
Age Group	25–44	45–54	55–64
All-Cause Mortality Rate 1990–1993	243.70	627.87	1367.98
All-Cause Mortality Rate 2015–2017	162.89	455.25	1002.30
Change 1990–1993 to 2015–2017	–80.81	–172.62	–365.68
(Change as % of 1990–1993 mortality)	–33.16	–27.49	–26.73

	Mortality Rate		Change		Mortality Rate		Change		Mortality Rate		Change	
Cause of Death	1990–1993	2015–2017	Abs Chg	% of Total +/– Chg	1990–1993	2015–2017	Abs Chg	% of Total +/– Chg	1990–1993	2015–2017	Abs Chg	% of Total +/– Chg
			–26.15	–27.5							8.91	15.4

Cancers												
Liver Cancer	0.59	0.53	–0.05	–0.1	2.30	3.19	0.90	3.7	6.52	13.48	6.97	12.0
Lung Cancer	4.59	1.19	–3.40	–3.6	39.66	16.35	–23.31	–11.8	108.58	68.08	–40.50	–9.6
All Other Cancers	41.80	27.61	–14.18	–14.9	163.83	107.15	–56.68	–28.8	342.25	242.89	–99.36	–23.5
Cardio and Metabolic Diseases												
Endocrine, Nutritional, & Metabolic	8.34	11.31	2.97	20.9	35.09	31.95	–3.13	–1.6	92.73	70.97	–21.76	–5.1
Hypertensive Heart Disease	5.65	7.51	1.87	13.1	28.67	27.15	–1.51	–0.8	61.95	53.22	–8.72	–2.1
Ischemic & Other Circulatory System	42.31	27.90	–14.40	–15.2	180.18	101.56	–78.62	–40.0	476.99	244.29	–232.70	–54.9
Substance Use and Mental Health												
Drug Poisoning	6.72	13.88	7.16	50.3	4.01	22.56	18.55	76.8	1.59	18.80	17.21	29.8
Alcohol-Induced*	5.38	2.15	–3.23	–3.4	11.24	6.58	–4.66	–2.4	12.18	9.26	–2.92	–0.7
Suicide	2.42	2.83	0.41	2.9	1.86	1.70	–0.16	–0.1	1.98	1.57	–0.41	–0.1
Mental & Behavioral Disorders	5.30	1.78	–3.52	–3.7	7.92	4.46	–3.46	–1.8	8.39	9.35	0.96	1.7
Other Body System Diseases												
Nervous System	4.21	5.93	1.72	12.1	9.31	13.72	4.41	18.3	15.27	27.98	12.71	22.0

continued

TABLE 4-2 Continued

	Mortality Rate		Change		Mortality Rate		Change		Mortality Rate		Change	
Genitourinary System	4.01	4.10	0.10	0.7	12.57	12.86	0.29	1.2	35.72	36.48	0.76	1.3
Respiratory System	11.40	6.47	–4.93	–5.2	29.91	24.42	–5.49	–2.8	67.09	72.12	5.03	8.7
Digestive System*	8.18	4.02	–4.16	–4.4	22.27	14.24	–8.03	–4.1	41.75	31.68	–10.08	–2.4
Other Causes of Death												
Homicide	19.64	8.11	–11.53	–12.1	7.82	4.67	–3.15	–1.6	5.66	3.50	–2.16	–0.5
Transport Accidents	9.83	8.20	–1.62	–1.7	8.73	7.90	–0.83	–0.4	10.44	7.06	–3.39	–0.8
Other External Causes of Death	5.15	3.36	–1.80	–1.9	8.04	6.53	–1.51	–0.8	12.72	11.22	–1.50	–0.4
All Other Causes of Death	17.41	**15.84**	–1.57	–1.7	22.73	22.66	–0.07	0.0	30.77	32.72	1.95	3.4
Total Change: Increase (+)				14.23				24.14				57.83
Total Change: Decrease (–)				–95.04				–196.76				–423.51

*Changes in the International Classification of Diseases (ICD)-10 code for diseases of the digestive system in 2006 affected the comparability of both digestive system deaths and alcohol-induced deaths before and after that year. More information is provided in Chapter 5.

NOTE: The top 3 causes of death for each age group in each period are highlighted in **bolded red** text.

Light orange highlights indicate an absolute increase in the overall mortality rate of ≥5 deaths/100,000 population.

Dark orange highlights indicate that a cause of death is responsible for ≥10 percent of the total increase in mortality.

Light green highlights indicate an absolute decrease in the overall mortality rate of ≥5 deaths/100,000 population.

Dark green highlights indicate that a cause of death is responsible for ≥10 percent of the total decrease in mortality.

The table shows mortality rates and change in all-cause and cause-specific mortality rates among non-Hispanic Black working-age adults by age group (25–44, 45–54, and 55–64) for males (upper table) and females (lower table). Changes in mortality are presented as both the absolute change in mortality rates and the percentage of the total increase (or decrease) in mortality. Mortality rates within each age group are age-adjusted by single-year of age to match the age distribution of the U.S. population in 2000 in order to improve comparability over time. These causes of death are exhaustive of all underlying cause-of-death codes and are based on the ICD-9 (1990–1998) and ICD-10 (1999–2017) codes. More information about the classification of causes of death can be found in Chapter 5.

SOURCE: Data from https://www.cdc.gov/nchs/nvss/nvss-restricted-data.htm.

offset the small increases in mortality experienced by Hispanics for several causes of death, including drug poisoning, hypertensive heart disease, ENM diseases, liver cancer, and nervous system diseases. Working-age Hispanic females also experienced increased mortality from several causes in the substance use and mental health category, including alcohol-induced causes, suicide, and mental and behavioral disorders, but these increases were mostly negligible. Like Black males, Hispanic males experienced decreases in mortality from these causes; however, rates of mortality from these causes were already much higher among both Black and Hispanic males than among Black and Hispanic females in 1990–1993, and they remained higher throughout the period.

Drug poisoning was the single largest contributor to increasing mortality among working-age Hispanics, except those ages 55–64 (for whom it was the second largest contributor among males and the third largest among females). However, absolute increases in mortality from drug poisoning among Hispanic adults were much smaller than those among White and Black adults. Although younger Hispanic adults (ages 25–44) had higher rates of mortality from drug poisoning in 1990–1993, older Hispanic adults, especially males, experienced larger increases over the period. By 2015–2017, rates of mortality from drug poisoning were similar across age groups among both Hispanic males and females. Mortality rates from alcohol-induced causes, suicide, and mental and behavioral disorders declined among Hispanic males in all three age groups, and while increasing negligibly among Hispanic females, remained relatively low throughout the period.

Beyond drug poisoning, mortality increased for very few causes of death among Hispanic adults, particularly in the two younger age groups. Negligible increases in mortality from ENM diseases and hypertensive heart disease occurred for males ages 25–54, and a small increase in diseases of the nervous system was seen among females ages 45–54. Table 4-3 shows large percentage contributions of these causes to changes in all-cause mortality, but this is only because the total increase across all causes of death was small. In the older age group (55–64), relatively large increases occurred in mortality from non-HIV/AIDS infectious and parasitic diseases, liver cancer, hypertensive heart disease, and diseases of the nervous system among males, along with increased mortality from non-HIV/AIDS infectious and parasitic diseases, liver cancer, and diseases of the nervous system among females.

As was true for Black males, Hispanic males experienced widespread improvements across multiple causes of death over the period, although the reductions among Hispanic males were much smaller because they began the period with much lower mortality rates. As with White and Black males, the most important contributors to mortality declines among Hispanic

males were HIV/AIDS, lung and other nonliver cancers (ages 45–64), and ischemic heart disease and other diseases of the circulatory system. However, Hispanic males also experienced notable declines in mortality from alcohol-induced causes, suicide, mental and behavioral disorders, homicide, and transport injuries. The trends in cause-specific mortality among Hispanic females resemble more closely those of White females, with increasing mortality across multiple causes of death. However, these increases were generally much smaller than those among White females; Hispanic females maintained a very favorable mortality profile, having experienced notable declines (on top of already low rates) in mortality from HIV/AIDS, nonliver cancers, and homicide (among those ages 25–44).

DISPARITIES IN CAUSE-SPECIFIC MORTALITY BY SOCIOECONOMIC STATUS

The literature on trends in educational attainment and working-age cause-specific mortality is fairly extensive; however, only a limited number of these studies focused on socioeconomic disparities in cause-specific mortality rates. Despite their limited number, these studies consistently found that increases in mortality due to drug poisoning, alcohol-induced causes, and suicide were the largest contributors to the growing gap in mortality by education among working-age Whites, and that increases in drug poisoning mortality were increasingly concentrated among working-age Whites with a high school degree or less. This group also experienced larger increases in mortality across a wide range of other causes of death, and this was especially true for White women with less education. In contrast, among working-age Black adults, education-based disparities in cause-specific mortality remained steady over time.

Sasson (2016) used vital statistics data to demonstrate steep increases in educational disparities in life expectancy for White men and women but little change for Black men and women between 1990 and 2010. He showed that the increases in the education-based mortality gap between working-age White men and women involved largely causes of death associated with smoking, external causes (including drug poisoning),[8] and cardiovascular diseases.

Case and Deaton (2015) delved into the specific causes of death associated with widening educational disparities in working-age mortality, focusing on adults ages 45–54 between 1999 and 2013. They showed that the death rate from poisoning among U.S. adults ages 45–54—which includes deaths from drug poisoning and alcohol-induced causes, both unintentional

[8]External causes of death are causes that are due to accidents and violence, including poisonings and environmental events.

TABLE 4-3 Cause-Specific Mortality (deaths per 100,000 population), 1990–1993 and 2015–2017: Hispanic Adults Ages 25–64

	Hispanic Males											
Age Group	25–44				45–54				55–64			
All-Cause Mortality Rate 2009–2011	287.25				520.73				1090.13			
All-Cause Mortality Rate 2015–2017	144.89				344.90				805.96			
Change 1990–1993 to 2015–2017	–142.36				–175.83				–284.17			
(Change as % of 1990–1993 mortality)	–49.56				–33.77				–26.07			
	Mortality Rate		Change		Mortality Rate		Change		Mortality Rate		Change	
Cause of Death	1990–1993	2015–2017	Abs Chg	% of Total +/– Chg	1990–1993	2015–2017	Abs Chg	% of Total +/– Chg	1990–1993	2015–2017	Abs Chg	% of Total +/– Chg
Infectious and Parasitic Diseases												
HIV/AIDS	70.91	2.38	–68.53	–45.5	57.24	6.36	–50.88	–25.0	30.35	7.86	–22.49	–6.3
Non-HIV/AIDS	6.84	2.32	–4.52	–3.0	11.55	12.28	0.73	2.6	19.35	31.13	11.78	16.5
Cancers												
Liver Cancer	0.83	0.54	–0.29	–0.2	5.32	8.93	3.61	13.1	16.59	35.64	19.05	26.6
Lung Cancer	2.08	0.73	–1.35	–0.9	18.58	5.51	–13.07	–6.4	77.14	30.51	–46.63	–13.1
All Other Cancers	15.65	11.95	–3.70	–2.5	64.00	48.70	–15.29	–7.5	194.86	143.86	–51.00	–14.3

continued

TABLE 4-3 Continued

	Mortality Rate		Change		Mortality Rate		Change		Mortality Rate		Change	
Cardio and Metabolic Diseases												
Endocrine, Nutritional, & Metabolic	4.65	5.09	0.44	5.4	18.19	20.72	2.53	9.2	54.32	58.01	3.70	5.2
Hypertensive Heart Disease	1.64	3.07	1.44	17.5	7.92	11.99	4.07	14.7	21.36	29.08	7.72	10.8
Ischemic & Other Circulatory System	23.58	16.19	–7.39	–4.9	128.06	70.47	–57.59	–28.3	385.36	200.92	–184.44	–51.8
Substance Use and Mental Health												
Drug Poisoning	16.26	22.39	6.13	74.8	10.16	24.45	14.29	51.7	4.25	19.36	15.11	21.1
Alcohol-Induced*	12.60	7.78	–4.82	–3.2	37.36	27.02	–10.34	–5.1	50.36	43.74	–6.62	–1.9
Suicide	14.42	12.94	–1.48	–1.0	14.04	11.59	–2.45	–1.2	16.28	10.78	–5.50	–1.5
Mental & Behavioral Disorders	7.33	2.92	–4.41	–2.9	14.66	8.26	–6.40	–3.1	15.28	13.83	–1.46	–0.4
Other Body System Diseases												
Nervous System	2.53	2.72	0.19	2.3	5.60	7.13	1.53	5.5	10.49	17.62	7.13	10.0
Genitourinary System	1.46	1.14	–0.33	–0.2	5.28	6.14	0.86	3.1	13.55	19.06	5.51	7.7
Respiratory System	6.30	2.97	–3.33	–2.2	17.25	10.39	–6.86	–3.4	51.06	37.73	–13.33	–3.7
Digestive System*	8.58	3.95	–4.62	–3.1	29.07	18.61	–10.47	–5.1	52.63	44.78	–7.84	–2.2

Other Causes of Death												
Homicide	38.69	12.75	–25.94	–17.2	22.98	6.89	–16.10	–7.9	14.04	5.47	–8.56	–2.4
Transport Accidents	30.75	19.41	–11.34	–7.5	27.37	17.55	–9.82	–4.8	27.08	20.08	–6.99	–2.0
Other External Causes of Death	13.90	8.68	–5.22	–3.5	15.85	13.24	–2.62	–1.3	20.54	19.69	–0.86	–0.2
All Other Causes of Death	8.26	4.96	–3.30	–2.2	10.26	8.69	–1.57	–0.8	15.26	16.80	1.54	2.2
Total Change: Increase (+)				8.20				27.62				71.54
Total Change: Decrease (–)				–150.56				–203.45				–355.72

continued

TABLE 4-3 Continued

	Hispanic Females									
Age Group	25–44				45–54				55–64	
All-Cause Mortality Rate 1990–1993	89.67				243.72				596.84	
All-Cause Mortality Rate 2015–2017	66.32				184.45				440.99	
Change 1990–1993 to 2015–2017	–23.35				–59.27				–155.85	
(Change as % of 1990–1993 mortality)	–26.04				–24.32				–26.11	
	Mortality Rate		Change		Mortality Rate		Change		Mortality Rate		Change	
Cause of Death	1990–1993	2015–2017	Abs Chg	% of Total +/– Chg	1990–1993	2015–2017	Abs Chg	% of Total +/– Chg	1990–1993	2015–2017	Abs Chg	% of Total +/– Chg
Infectious and Parasitic Diseases												
HIV/AIDS	11.74	0.54	–11.20	–37.8	7.38	1.33	–6.05	–8.5	4.38	1.88	–2.50	–1.4
Non-HIV/AIDS	2.57	1.41	–1.16	–3.9	5.38	6.13	0.75	6.2	13.41	16.70	3.29	14.7
Cancers												
Liver Cancer	0.29	0.39	0.10	1.7	1.90	2.24	0.35	2.9	6.17	9.96	3.79	17.0
Lung Cancer	1.03	0.71	–0.32	–1.1	8.09	4.92	–3.18	–4.4	26.69	18.45	–8.25	–4.6
All Other Cancers	21.79	17.24	–4.56	–15.4	85.51	60.52	–24.99	–35.0	178.44	135.29	–43.15	–24.2

Cardio and Metabolic Diseases												
Endocrine, Nutritional, & Metabolic	2.50	2.88	0.39	6.2	12.99	11.82	–1.17	–1.6	50.36	35.63	–14.73	–8.3
Hypertensive Heart Disease	0.80	1.00	0.20	3.3	4.06	4.73	0.67	5.5	12.34	13.04	0.70	3.1
Ischemic & Other Circulatory System	10.17	7.07	–3.10	–10.5	53.33	28.78	–24.55	–34.4	175.91	81.51	–94.41	–53.0
Substance Use and Mental Health												
Drug Poisoning	2.91	7.25	4.34	69.7	2.24	9.05	6.81	55.8	2.15	7.54	5.39	24.2
Alcohol-Induced*	2.00	2.50	0.50	8.1	6.07	6.95	0.88	7.2	8.38	9.27	0.89	4.0
Suicide	2.24	2.66	0.42	6.7	1.78	2.40	0.62	5.1	1.92	1.97	0.05	0.2
Mental & Behavioral Disorders	1.29	0.83	–0.46	–1.5	1.63	2.03	0.40	3.3	3.03	4.02	0.99	4.4
Other Body System Diseases												
Nervous System	1.54	1.80	0.26	4.2	3.30	5.00	1.71	14.0	7.38	13.44	6.07	27.2
Genitourinary System	0.98	0.99	0.01	0.2	4.48	4.33	–0.15	–0.2	12.33	12.98	0.66	2.9
Respiratory System	3.47	1.93	–1.54	–5.2	10.53	8.00	–2.53	–3.5	29.20	27.01	–2.19	–1.2
Digestive System*	2.50	2.14	–0.36	–1.2	10.94	8.82	–2.12	–3.0	33.06	26.52	–6.54	–3.7

continued

TABLE 4-3 Continued

Cause of Death	Mortality Rate 1990–1993	Mortality Rate 2015–2017	Change Abs Chg	Change % of Total +/– Chg	Mortality Rate 1990–1993	Mortality Rate 2015–2017	Change Abs Chg	Change % of Total +/– Chg	Mortality Rate 1990–1993	Mortality Rate 2015–2017	Change Abs Chg	Change % of Total +/– Chg
Other Causes of Death												
Homicide	6.26	2.75	–3.51	–11.9	3.84	2.02	–1.82	–2.5	2.54	1.24	–1.30	–0.7
Transport Accidents	8.49	5.70	–2.79	–9.4	8.92	5.59	–3.33	–4.7	10.70	6.45	–4.25	–2.4
Other External Causes of Death	1.79	1.41	–0.38	–1.3	2.59	2.55	–0.04	–0.1	5.09	5.56	0.47	2.1
All Other Causes of Death	5.33	5.13	–0.20	–0.7	8.77	7.23	–1.53	–2.1	13.37	12.53	–0.85	–0.5
Total Change: Increase (+)				6.23				12.19				22.29
Total Change: Decrease (–)				–29.58				–71.46				–178.14

*Changes in the International Classification of Diseases (ICD)-10 code for diseases of the digestive system in 2006 affected the comparability of both digestive system deaths and alcohol-induced deaths before and after that year. More information is provided in Chapter 5.
NOTE: The top 3 causes of death for each age group in each period are highlighted in **bolded red** text.
Light orange highlights indicate an absolute increase in the overall mortality rate of ≥5 deaths/100,000 population.
Dark orange highlights indicate that a cause of death is responsible for ≥10 percent of the total increase in mortality.
Light green highlights indicate an absolute decrease in the overall mortality rate of ≥5 deaths/100,000 population.
Dark green highlights indicate that a cause of death is responsible for ≥10 percent of the total decrease in mortality.

The table shows mortality rates and change in all-cause and cause-specific mortality rates among Hispanic working-age adults by age group (25–44, 45–54, and 55–64) for males (upper table) and females (lower table). Changes in mortality are presented as both the absolute change in mortality rates and the percentage of the total increase (or decrease) in mortality. Mortality rates within each age group are age-adjusted by single-year of age to match the age distribution of the U.S. population in 2000 in order to improve comparability over time. These causes of death are exhaustive of all underlying cause-of-death codes and are based on the ICD-9 (1990–1998) and ICD-10 (1999–2017) codes. More information about the classification of causes of death can be found in Chapter 5.

SOURCE: Data from https://www.cdc.gov/nchs/nvss/nvss-restricted-data.htm.

and of undetermined intentionality—increased during that period for White adults of all educational levels, as well as for Black and Hispanic adults. However, the increase was especially pronounced for White adults with a high school degree or less; in this group the death rate from poisoning increased more than four-fold over the period, from 14 per 100,000 population in 1999 to 58 per 100,000 population in 2013. Death rates due to poisonings among White adults with some college education and those with a college degree or more in this age group also increased rapidly but started at a lower level in 1999, and the increases across the time period were less pronounced. For example, the poisoning death rate for White adults ages 45–54 with some college increased from 6 per 100,000 to 21 per 100,000, while the increase for White adults with a college degree or more was from 3 per 100,000 to 8 per 100,000.

These findings provided the first clear evidence that mortality from drug poisoning among working-age Whites was increasing more rapidly among those with less versus those with more education. Unfortunately, Case and Deaton (2015) did not break down results for working-age Black or Hispanic adults by educational attainment. Notably, though, mortality from poisoning among Black and Hispanic adults ages 45–54 increased between 1999 and 2013, from 18 to 22 and from 10 to 14 per 100,000 population, respectively. Case and Deaton (2017) later updated these descriptive trends, examining them separately by sex, but the main finding of growing education-based disparities in mortality among working-age White adults did not change.

Most recently, Geronimus and colleagues (2019) documented changes in educational disparities in mortality between 1990 and 2015 for working-age (and older) Black and White women and men. They measured educational attainment in quartiles to help account for compositional changes within education categories due to increasing educational attainment across time. Thus, they compared changes in mortality disparities between 1990 and 2015 for those in the bottom 25 percent versus those in the top 25 percent of the educational attainment distribution.

Geronimus and colleagues (2019) found that among White adults, increasing drug-related mortality was especially concentrated among those with less education, accounting for 73 percent and 44 percent of the increased educational disparity in working-age mortality for White men and White women, respectively. White men and women also exhibited modest increases in educational disparities in working-age mortality due to suicide and liver disease. Educational disparities in working-age mortality for White women also widened over the period for a range of causes of death, including cardiovascular disease, nonlung cancers, non-HIV infectious diseases, lower respiratory diseases, and other internal causes, and only (very modestly) narrowed for homicide. Among White men, educational

disparities in working-age mortality widened for some of the same causes as those seen among White women (e.g., other cancers, other infectious diseases, other internal causes) but narrowed for others (e.g., lung cancer, accidents).

Thus in all, one-half (White women) to 80 percent (White men) of the increasing educational disparity in working-age mortality over the 1990–2015 period was due to what some researchers have referred to as "despair-related" causes, with educational differences in mortality from drug poisoning being particularly important for understanding the widening of educational disparities in working-age mortality that occurred among White men and women over the period. Furthermore, and particularly for White women, increasing educational disparities in working-age mortality were also seen for a range of other causes. By contrast, there was virtually no change in educational disparities in working-age mortality for Black men and women between 1990 and 2015. Increasing drug-related mortality among Black women and men differed only modestly by educational attainment and thus had very little influence on changing educational disparities in working-age mortality. Geronimus and colleagues (2019) also found only minor changes in educational disparities in mortality from lung cancer, cardiovascular diseases, diabetes, and a range of other causes for Black men and women across the 25-year time period. In other words, declines in working-age mortality over the period for Black men and Black women unfolded in parallel fashion across educational attainment groupings.

CAUSE-SPECIFIC MORTALITY TRENDS BY METROPOLITAN STATUS

All-cause mortality trends were most favorable in large central metropolitan areas (hereafter referred to as "large central metros") and less favorable in less-populated areas over the period, often leading to a widening mortality gap across these areas (see Chapter 3). The findings reported in this section show that most cause-specific mortality rates followed a similar trend, suggesting that the growing geographic mortality gap was the cumulative result of underlying processes that produced metro status differences for multiple causes of death. In general, when cause-specific mortality rates decreased over the period, they declined the most in large central metros; when they increased, either mortality continued to decline, or the increases were smaller in large central metros. Detailed tables showing the change in cause-specific mortality between 1990–1993 and 2015–2017 by sex, age group, and metropolitan status can be found in the annex at the end of this chapter (Annex Tables 4-1 to 4-3). Cause-specific mortality rates by sex, age group, and metropolitan status are in Appendix A.

This pattern was most consistent for working-age Whites, among whom most causes of death contributed to the growing disparities between large central metros and less-populated areas. In contrast, for working-age Black and Hispanic adults, cause-specific mortality rates did not consistently decline the most in large central metros. Although Black and Hispanic adults in large central metros did experience greater improvements in mortality from many causes of death, those in nonmetropolitan areas (hereafter referred to as "nonmetros") often experienced larger decreases in mortality from the causes of death that were key drivers of reductions in mortality over the period (e.g., cancers other than liver cancer or ischemic heart disease and other circulatory diseases). Nonetheless, Black and Hispanic adults in large central metros, particularly males, saw much larger declines in mortality from HIV/AIDS and homicides that offset these differences and drove the greater overall improvements in mortality in large central metros.

Areas outside of large central metros were more likely to experience larger increases in mortality across more causes of death. Often, the largest increases in cause-specific mortality occurred within nonmetros. Mortality due to drug poisoning was a notable exception to this pattern. Among White males and older (ages 45–64) Black males and females, large metros saw the biggest increases in mortality from drug poisoning, while nonmetros experienced smaller increases. Among White males, the largest increases occurred in large fringe metropolitan areas (hereafter referred to as "large fringe metros"), while among older Black adults, the largest increases occurred in large central metros. This meant that drug poisoning did not contribute to, and in fact offset, the growing mortality disparity between large central metros and less-populated areas among White males and older (ages 45–64) Black males and females. In contrast, White females, Hispanics, and younger working-age (ages 25–44) Black adults experienced smaller increases in mortality due to drug poisoning overall, with the smallest increases occurring in large central metros. Younger (ages 25–44) Black adults living in nonmetros also experienced a smaller overall increase in drug poisoning mortality.

TEMPORAL PATTERNS IN CAUSE-SPECIFIC MORTALITY TRENDS

The changes described above reflect changes in cause-specific mortality rates over the full period (1990–1993 to 2015–2017). In this section, the total change in each cause-specific mortality rate over the period is decomposed into changes within three periods that correspond roughly to decades. This decomposition makes it possible to determine when the changes occurred for different causes of death and identify the causes of

death responsible for those changes. These periods of change are referred to as the 1990s (1990–1993 to 2000–2002), the 2000s (2000–2002 to 2009–2011), and the 2010s (2009–2011 to 2015–2017).

Figures 4-1 and 4-2 (showing changes in cause-specific mortality rates by time period for males and females, respectively) reveal two main findings beyond those already discussed earlier in this chapter. First, among the causes of death for which mortality rates declined in meaningful ways (i.e., HIV/AIDS, nonliver cancers, ischemic heart disease and other diseases of the circulatory system, and homicides [among young Black and Hispanic males]), most improvements occurred in the 1990s or 2000s and stagnated or even reversed in the 2010s. This pattern appeared most consistently for ischemic heart disease and other circulatory diseases, which saw a slowing rate of improvement in mortality over the period among most working-age adults. Among White females, younger Black males, and younger Hispanic males and females, progress in lowering mortality from ischemic heart disease and other circulatory diseases ceased in the 2010s as all-cause mortality rates began to increase.[9]

Most of the progress in reducing mortality from HIV/AIDS occurred in the 1990s among White and Hispanic males and younger Black males. However, mortality from HIV/AIDS continued to increase among Black females and older Black males in the 1990s and did not begin to decrease until the 2000s; among Black females ages 55–64, mortality from HIV/AIDS did not begin to decline until the 2010s. By 2015–2017, mortality from HIV/AIDS among White and Hispanic adults was sufficiently low that continued progress in reducing it would not have substantially affected future mortality trends. In contrast, mortality from HIV/AIDS remained high among working-age Black adults in 2015–2017; therefore, efforts to address this cause of death in this population could affect future mortality trends, as well as mortality disparities between Blacks and Whites. Although homicide rates decreased substantially in the 1990s, especially among Black and Hispanic males, progress slowed in the 2000s. By the 2010s, most groups had experienced at least a small increase in the homicide rate; the increases were particularly large among younger working-age Black males. In contrast to other leading causes of death, progress on lung and other nonliver cancers continued into the 2010s, although possibly at a slower rate.

The second main finding is that mortality rates increased among multiple demographic groups across a wide range of causes. These causes

[9]Although reductions in mortality ceased for the category of ischemic heart disease and other circulatory diseases as a whole, some causes of death continued to decrease during this period, though at an attenuated rate.

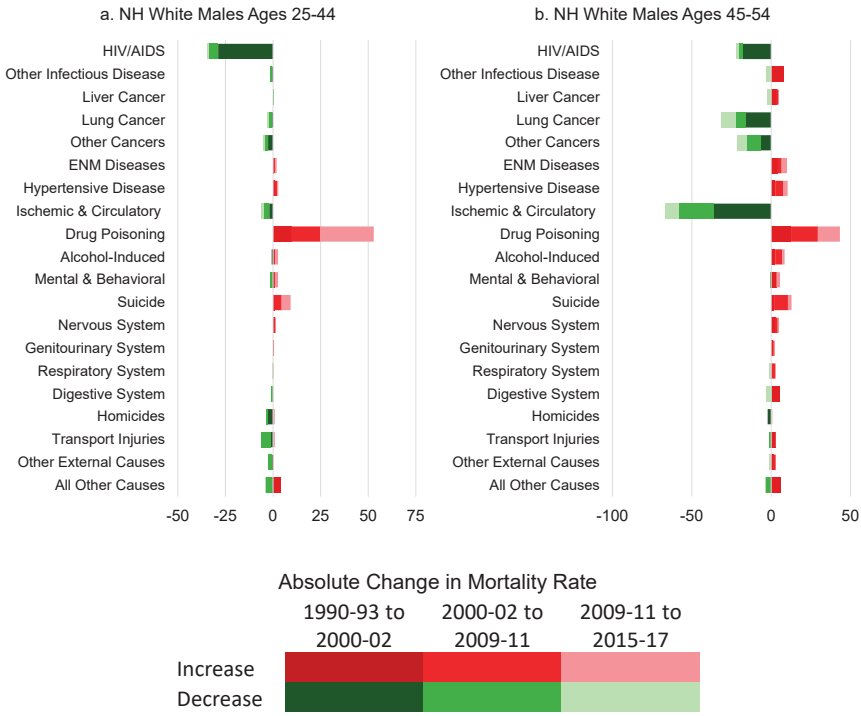
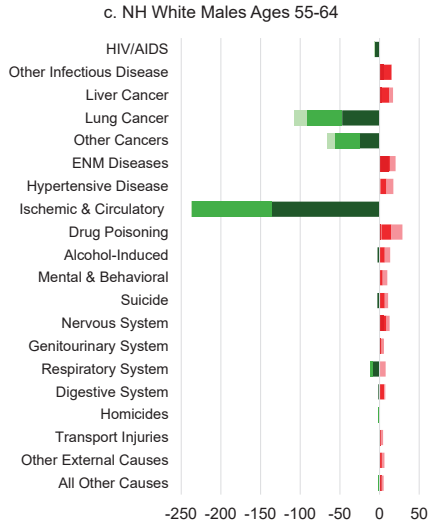
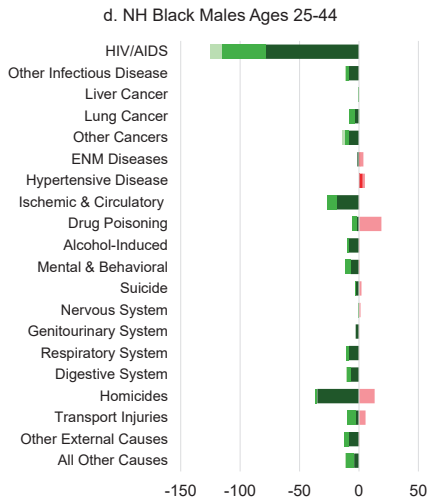
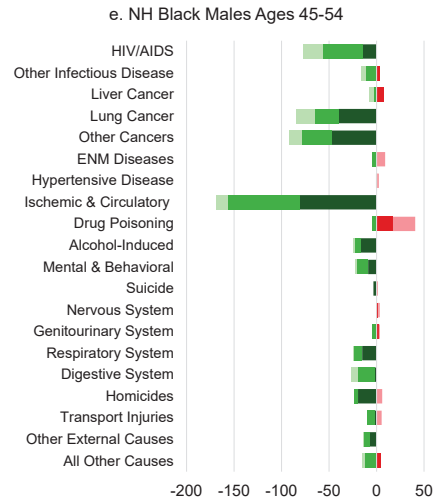
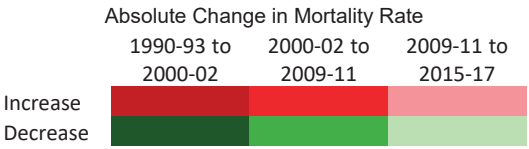


FIGURE 4-1 Decomposition of changes in cause-specific mortality rates (deaths per 100,000 population) by time period: Males.


c. NH White Males Ages 55-64
HIV/AIDS
Other Infectious Disease
Liver Cancer
Lung Cancer
Other Cancers
ENM Diseases
Hypertensive Disease
Ischemic & Circulatory
Drug Poisoning
Alcohol-Induced
Mental & Behavioral
Suicide
Nervous System
Genitourinary System
Respiratory System
Digestive System
Homicides
Transport Injuries
Other External Causes
All Other Causes
-250 -200 -150 -100 -50 0 50


d. NH Black Males Ages 25-44
HIV/AIDS
Other Infectious Disease
Liver Cancer
Lung Cancer
Other Cancers
ENM Diseases
Hypertensive Disease
Ischemic & Circulatory
Drug Poisoning
Alcohol-Induced
Mental & Behavioral
Suicide
Nervous System
Genitourinary System
Respiratory System
Digestive System
Homicides
Transport Injuries
Other External Causes
All Other Causes
-150 -100 -50 0 50


e. NH Black Males Ages 45-54
HIV/AIDS
Other Infectious Disease
Liver Cancer
Lung Cancer
Other Cancers
ENM Diseases
Hypertensive Disease
Ischemic & Circulatory
Drug Poisoning
Alcohol-Induced
Mental & Behavioral
Suicide
Nervous System
Genitourinary System
Respiratory System
Digestive System
Homicides
Transport Injuries
Other External Causes
All Other Causes
-200 -150 -100 -50 0 50


Absolute Change in Mortality Rate
1990-93 to 2000-02
2000-02 to 2009-11
2009-11 to 2015-17
Increase
Decrease

continued

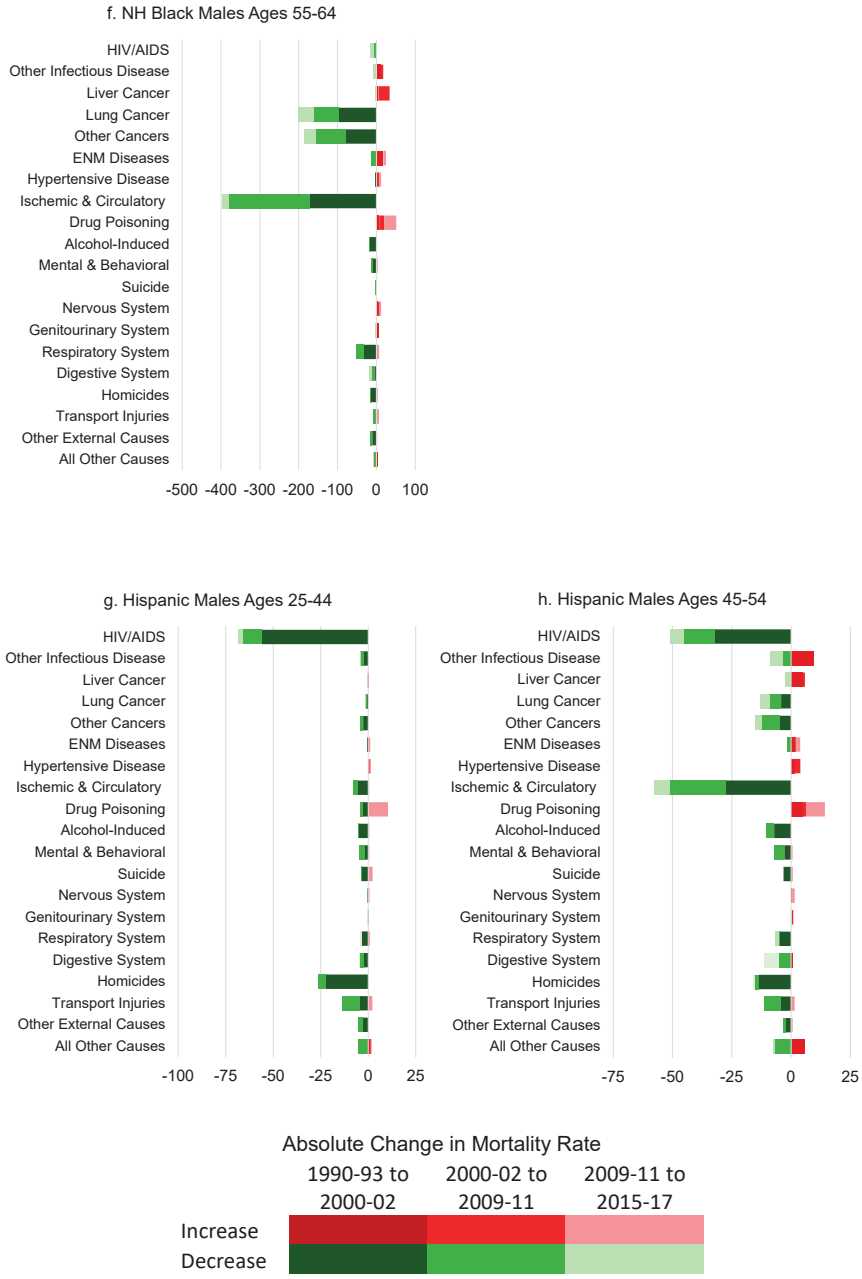
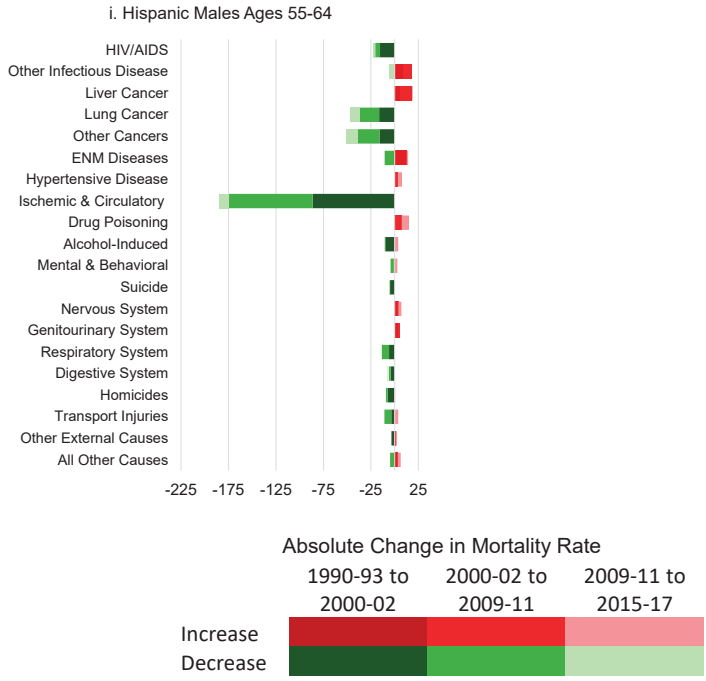


FIGURE 4-1 Continued



NOTE: The decomposition of the total change in cause-specific mortality rates between 1990 and 2017 is shown for three periods corresponding roughly to the changes in mortality within the 1990s (1990–1993 to 2000–2002), 2000s (2000–2002 to 2009–2011), and 2010s (2009–2011 to 2015–2017) for males ages 25–64. Each decomposition is shown separately for White (panels a, b, c), Black (panels d, e, f), and Hispanic (panels g, h, i) males in each of three age groups (25–44 [panels a, d, g], 45–54 [panels b, e, h], and 55–64 [panels c, f, i]). The green bars represent declines in mortality (dark green = decline in the 1990s, medium green = decline in the 2000s, and light green = decline in the 2010s), while the red bars represent increases in mortality (dark red = increase in the 1990s, medium red = increase in the 2000s, pink = increase in the 2010s). Readers should be mindful that the x-axis differs across panels. The causes of death shown are exhaustive of all underlying cause-of-death codes and are based on the International Classification of Diseases (ICD)-9 (1990–1998) and ICD-10 (1999–2017) codes. Mortality rates within each age group are age-adjusted by single-year of age to match the age distribution of the U.S. population in 2000 in order to improve comparability over time. More information about the classification of causes of death can be found in Chapter 5. ENM = endocrine, nutritional, and metabolic diseases.
SOURCE: Data from https://www.cdc.gov/nchs/nvss/nvss-restricted-data.htm.

included ENM diseases, hypertensive heart disease, drug poisoning, alcohol-induced causes, mental and behavioral disorders, suicide, and diseases of the nervous system. Some of these causes increased throughout the entire period (1990–1993 to 2015–2017), whereas other causes increased only recently (in the 2010s). For example, mortality from diseases of the nervous system increased in all three periods among most groups, although these increases were generally small among younger working-age adults (ages 25–44). The exception was younger Black males, among whom mortality from diseases of the nervous system decreased slightly in the 1990s and 2000s before increasing in the 2010s.

The timing of the increases in mortality from the two cardiometabolic diseases—hypertensive heart disease and ENM diseases—differed between older and younger working-age adults. Mortality for both increased in all three periods among younger working-age adults. Although older working-age adults saw increases in mortality from ENM diseases in the 1990s and 2010s, they also experienced much larger reductions in between (in the 2000s) that were often large enough to offset the much smaller mortality increases in both the earlier and later periods. Mortality from hypertensive heart disease increased in all three periods among older working-age (ages 45–64) White males and females and Hispanic males, but did not start to rise until the 2000s among older Black males, Black females ages 45–54, and Hispanic females ages 45–54. Black and Hispanic females ages 55–64 continued to experience reductions in mortality from hypertensive heart disease until the 2010s, during which it increased.

As noted earlier, previous studies have considered mortality from drug poisoning, alcohol-induced causes, and suicide together as a group. However, the timing of the increases in these causes of death differed. Increases occurred in drug poisoning mortality in all three periods among White males and females and Hispanic females, but only White males ages 45–54 also saw concurrent increases in mortality from both alcohol-induced causes and suicide. White females ages 25–44 experienced increasing mortality from alcohol-induced causes in all three periods, but their suicide rates did not increase until the 2000s. Neither mortality due to alcohol-induced causes nor suicide rates increased until the 2000s among other working-age White adults, and suicide rates remained flat among Hispanic females in all three periods.

Mortality from drug poisoning also increased in each period among older working-age (ages 45–64) Black males and females and older working-age Hispanic males, although none of these groups experienced an increase in mortality from alcohol-induced causes or suicide. The exception was Hispanic males ages 55–64, who saw a small increase in alcohol-induced mortality in the 2010s. In fact, alcohol-induced mortality decreased among

Hispanic males in the 1990s and 2000s. Among Black males ages 45–54, mortality from drug poisoning increased in the 1990s but fell in the 2000s before sharply increasing again in the 2010s, while mortality from alcohol-induced causes decreased in each period, and suicide rates decreased in the 1990s and 2000s. Mortality due to drug poisoning and suicide did not increase among younger Black adults and younger Hispanic males (ages 25–44) until the 2010s, except among younger Black females, who saw no change in suicide rates. Mortality from alcohol-induced causes remained flat throughout the period among these younger working-age adults.

Taken together, the differing trends in mortality for each of these three causes of death (drug poisoning, alcohol-induced causes, and suicide) suggest that there are limitations to considering these causes of death together, particularly when one is examining younger, female, and non-White working-age adults. Drug poisoning was by far the largest contributor to the overall increase in mortality for most working-age adults during the period, but the comparisons in Figures 4-1 and 4-2 demonstrate, importantly, that the timing of the increase in mortality from drug poisoning (the largest contributor to the overall increase in all-cause mortality for several groups) varied by sex, race and ethnicity, and age group.

SUMMARY

The recent trends in all-cause mortality among working-age adults are the result of the confluence of two important trends: (1) rising mortality from drug poisoning and other causes of death, such as nervous system diseases, hypertensive heart disease, and ENM diseases; and (2) slower progress in lowering mortality from heart diseases and other leading causes of death that drove improvements in all-cause mortality rates in prior decades. Table 4-4 summarizes the findings for each of the 20 causes of death considered in this chapter, showing how each contributed to changes in mortality over the period by age group, sex, and race and ethnicity. Subsequent chapters examine in greater detail the trends in the key causes of death that have driven the recent increases in mortality among working-age adults, either through increasing mortality or through a reduction or reversal of progress in reducing mortality in the most recent period. These trends are used to assess how consistent the prevailing explanations for recent increases in mortality in the research literature are with these cause-specific mortality trends.

Based on the findings presented in this chapter, the key drivers of the increases in working-age mortality since 2010 are grouped into three categories, each of which is addressed in detail in Part II of this report. The first category is drug poisoning and alcohol-induced causes (Chapter 7). In addition to mortality from drug poisoning and alcohol-induced causes, this

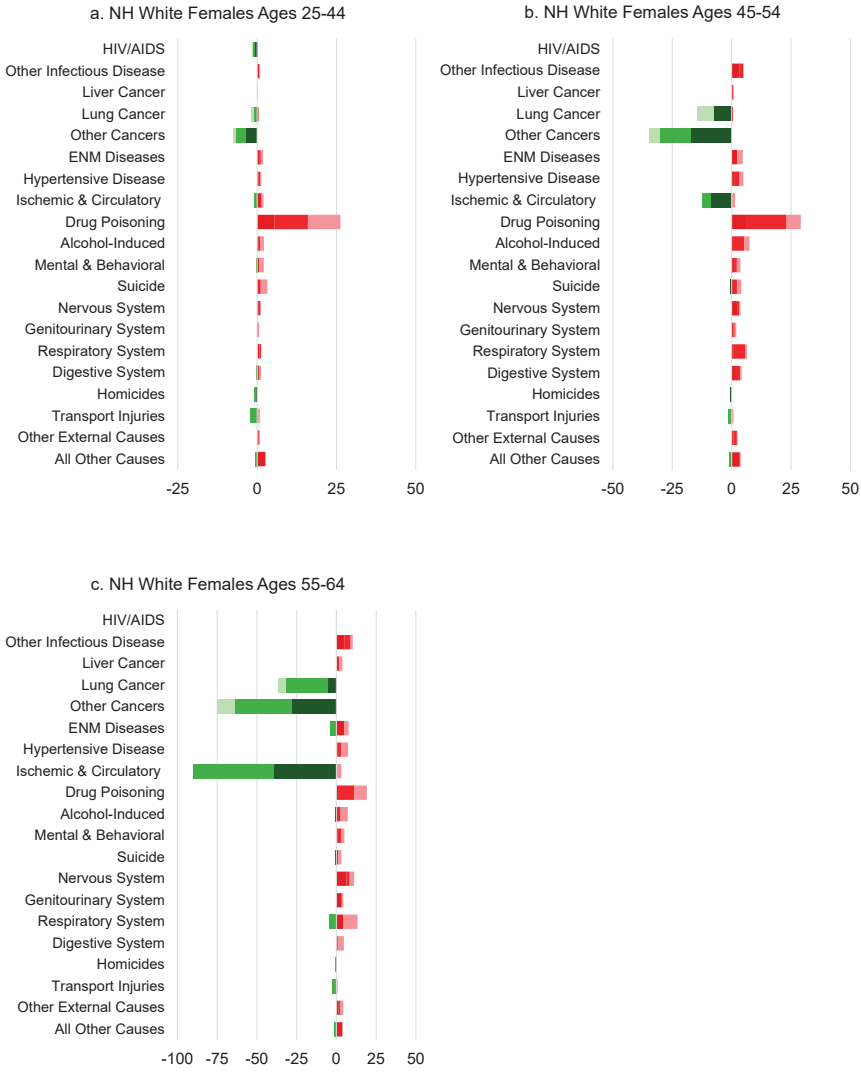
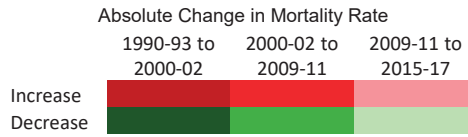
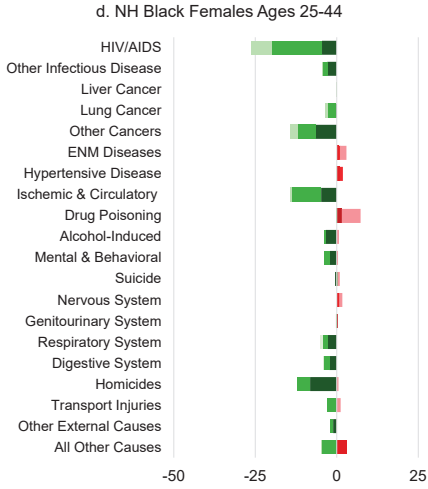
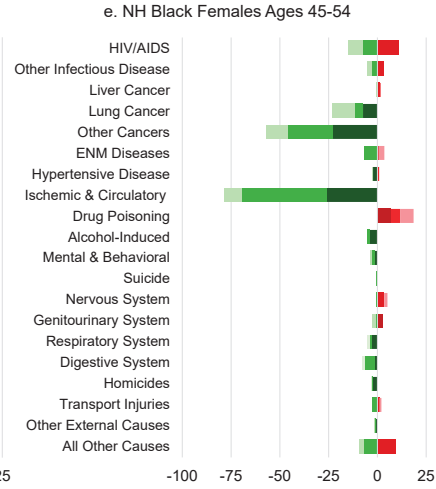
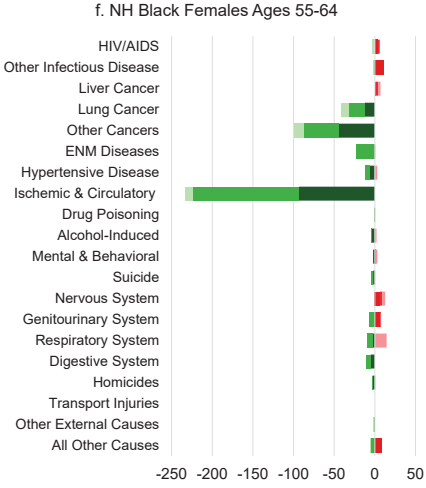


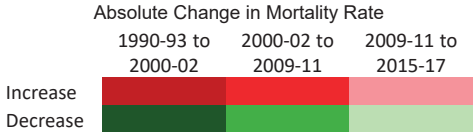
FIGURE 4-2 Decomposition of change in cause-specific mortality rates (deaths per 100,000 population) by time period: Females.




d. NH Black Females Ages 25-44
HIV/AIDS
Other Infectious Disease
Liver Cancer
Lung Cancer
Other Cancers
ENM Diseases
Hypertensive Disease
Ischemic & Circulatory
Drug Poisoning
Alcohol-Induced
Mental & Behavioral
Suicide
Nervous System
Genitourinary System
Respiratory System
Digestive System
Homicides
Transport Injuries
Other External Causes
All Other Causes
-50
-25
0
25


e. NH Black Females Ages 45-54
HIV/AIDS
Other Infectious Disease
Liver Cancer
Lung Cancer
Other Cancers
ENM Diseases
Hypertensive Disease
Ischemic & Circulatory
Drug Poisoning
Alcohol-Induced
Mental & Behavioral
Suicide
Nervous System
Genitourinary System
Respiratory System
Digestive System
Homicides
Transport Injuries
Other External Causes
All Other Causes
-100
-75
-50
-25
0
25


f. NH Black Females Ages 55-64
HIV/AIDS
Other Infectious Disease
Liver Cancer
Lung Cancer
Other Cancers
ENM Diseases
Hypertensive Disease
Ischemic & Circulatory
Drug Poisoning
Alcohol-Induced
Mental & Behavioral
Suicide
Nervous System
Genitourinary System
Respiratory System
Digestive System
Homicides
Transport Injuries
Other External Causes
All Other Causes
-250
-200
-150
-100
-50
0
50


Absolute Change in Mortality Rate
1990-93 to 2000-02
2000-02 to 2009-11
2009-11 to 2015-17
Increase
Decrease

continued

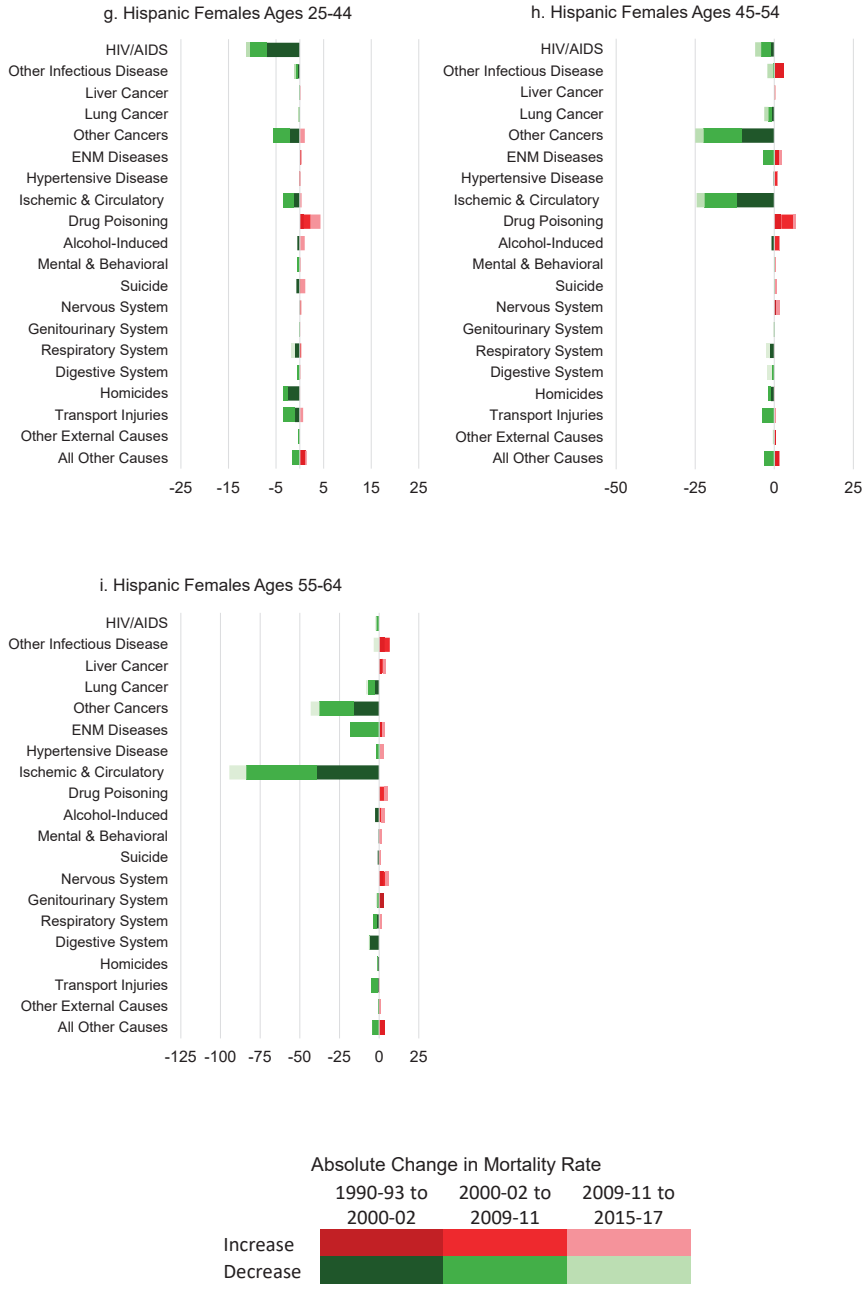


FIGURE 4-2 Continued

NOTE: The decomposition of the total change in cause-specific mortality rates between 1990 and 2017 is shown for three periods corresponding roughly to the changes in mortality within the 1990s (1990–1993 to 2000–2002), 2000s (2000–2002 to 2009–2011), and 2010s (2009–2011 to 2015–2017) for females ages 25–64. Each decomposition is shown separately for White (panels a, b, c), Black (panels d, e, f), and Hispanic (panels g, h, i) females in each of three age groups (25–44 [panels a, d, g], 45–54 [panels b, e, h], and 55–64 [panels c, f, i]). The green bars represent declines in mortality (dark green = decline in the 1990s, medium green = decline in the 2000s, and light green = decline in the 2010s), while the red bars represent increases in mortality (dark red = increase in the 1990s, medium red = increase in the 2000s, pink = increase in the 2010s). Readers should be mindful that the x-axis differs across panels. The causes of death shown are exhaustive of all underlying cause-of-death codes and are based on the International Classification of Diseases (ICD)-9 (1990–1998) and ICD-10 (1999–2017) codes. Mortality rates within each age group are age-adjusted by single-year of age to match the age distribution of the U.S. population in 2000 in order to improve comparability over time. More information about the classification of causes of death can be found in Chapter 5. ENM = endocrine, nutritional, and metabolic diseases.
SOURCE: Data from https://www.cdc.gov/nchs/nvss/nvss-restricted-data.htm.

one substance. Thus, drug use is often involved in deaths for which the underlying cause is coded as alcohol-induced, and vice versa.

As noted earlier and discussed in Chapter 7, most deaths for which the underlying cause of death is classified as a mental or behavioral disorder involve either drug poisoning or alcohol (Figure 4-3). In 1990, more than 70 percent of deaths due to a mental or behavioral disorder were due to alcohol, while nearly 15 percent were due to drug use. Over the 1990s and early 2000s, the percentage of these deaths due to alcohol decreased steadily, reaching a low of 55 percent in 2007. At the same time, the percentage due to drug poisoning increased steadily, reaching 23 percent in 2006. Throughout the period, more than 70 percent of all deaths due to a mental or behavioral disorder were due to either alcohol or drug use. For this reason, the explanations for mortality due to drug poisoning, alcohol-induced causes, and mental and behavioral disorders are discussed together in Chapter 7.

The second key driver of mortality evaluated by the committee in detail is suicide. Arguments can be made for examining suicide alongside mortality due to drugs and alcohol. For one, it can be difficult for medical examiners to distinguish between accidental and intentional drug poisoning, leading some suicides to be misclassified as accidental poisoning and vice versa. However, the committee sidestepped this classification problem by categorizing all mortality due to drug poisoning, both intentional and

category also includes mortality due to mental and behavioral disorders, which often involve drugs or alcohol (see Figure 4-3). The second category includes suicides that do not involve drug poisoning (Chapter 8). The third is mortality due to cardiometabolic diseases, which include ENM diseases, hypertensive heart disease, and ischemic heart disease and other circulatory diseases. The results presented in Table 4-4 indicate that in addition to these key causes of death, several other causes—including infectious and parasitic diseases other than HIV/AIDS, liver cancer, diseases of the nervous system, transport accidents, and homicide—also contributed to rising mortality over the period.

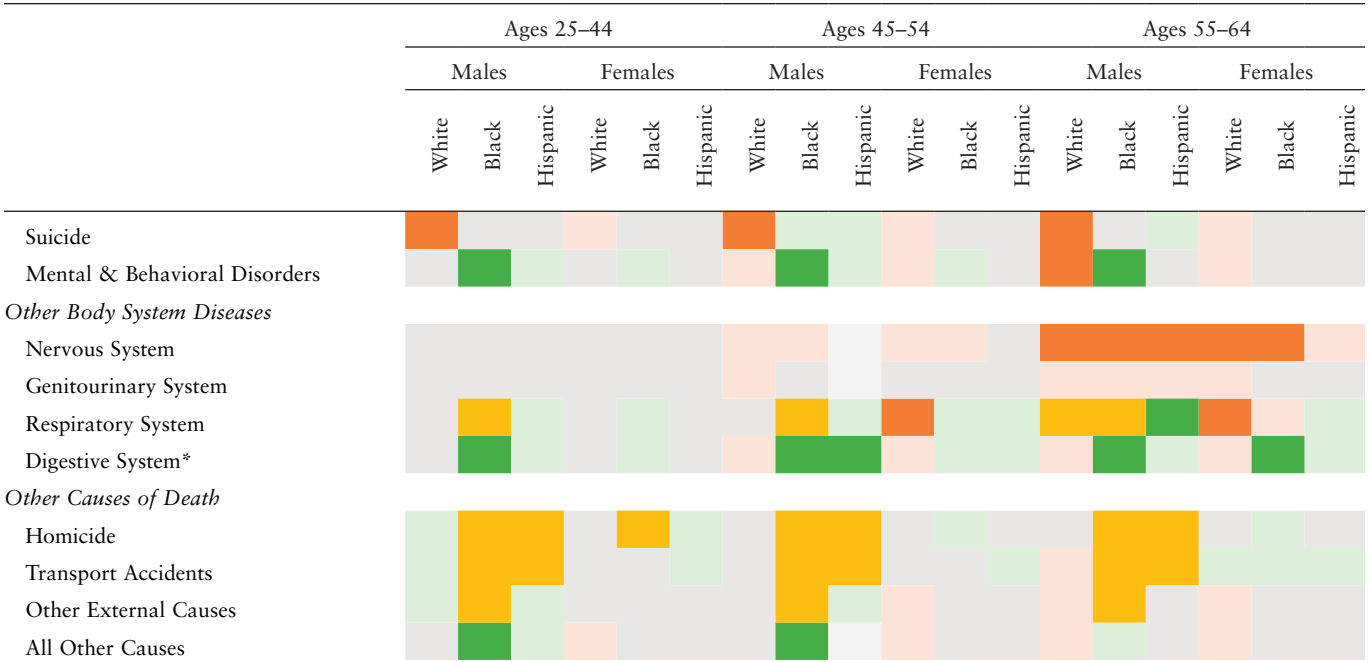
The results presented in this chapter demonstrate that drug poisoning mortality rose throughout the study period and was the single largest contributor to the overall increases in mortality among working-age adults, except older (ages 45–64) Hispanics. The largest increases during the period occurred among White adults, particularly White males and older Black males. Among working-age Whites, increases in mortality due to drug poisoning were largest among younger males (ages 25–44), those with a high school degree or less, and those living in large metropolitan areas. In contrast, among Black adults, the largest increases in mortality occurred among older males (ages 55–64) in large central metros, but there was no difference in drug poisoning mortality by educational attainment. Alcohol-induced mortality also increased among working-age Whites throughout the period, while increases among Black and Hispanic adults did not begin until the 2010s. Mortality from alcohol-induced causes declined among working-age Black and Hispanic males throughout the 1990s and early 2000s, but these declines leveled off during the 2000s and began to increase in the 2010s. Moreover, the increases in alcohol-induced mortality among working-age Whites followed different patterns than the increases in mortality from drug poisoning, which could reflect temporal differences in the etiology of these causes of death. Mortality due to alcohol-induced causes increased more among older working-age Whites than among other groups and outside of large central metros.

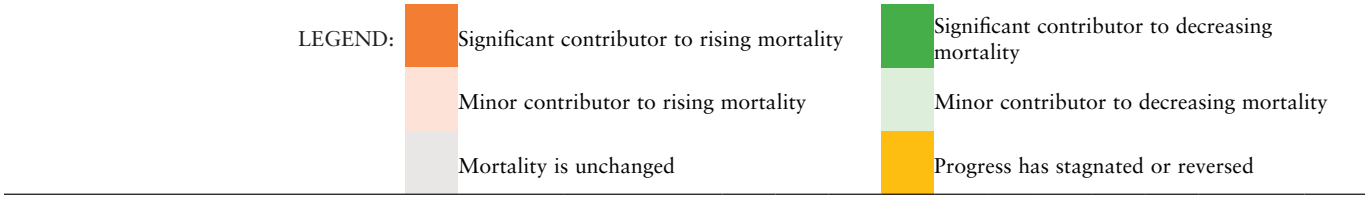
Despite these different trends in drug- and alcohol-induced mortality, there are important reasons to consider the explanations for these trends in concert with each other. For example, Case and Deaton (2015, 2017, 2020) posit that these deaths are the result of an underlying root cause: the erosion of economic and social stability within the White working class has increased physical, emotional, and psychological pain, leading to increases in substance use and mortality, particularly among less-educated White men. Considering these causes of death together allowed the committee to better evaluate the evidence underlying this "deaths of despair" hypothesis. However, a second, more practical, reason to consider these causes of death in parallel is that most substance-induced deaths involve more than

TABLE 4-4 Summary of Findings: Cause-Specific Mortality Among Working-Age Adults, 1990–2017

continued

TABLE 4-4 Continued





*Changes in the International Classification of Diseases (ICD)-10 code for diseases of the digestive system in 2006 affected the comparability of both digestive system deaths and alcohol-induced deaths before and after that year. More information is provided in Chapter 5.
SOURCE: Data from https://www.cdc.gov/nchs/nvss/nvss-restricted-data.htm.

accidental, as drug poisoning. Moreover, the underlying trends for suicide differ in important ways from those for drug poisoning and alcohol-induced mortality. Unlike increased mortality due to drug poisoning, increases in suicide deaths occurred primarily among working-age Whites, particularly White men. Among White adults, suicide rates were highest in nonmetros and lowest in large central metros—whereas the largest increases in mortality from drug poisoning were within large central metros—and were higher among older (ages 45–64) White adults compared with their younger counterparts. These differences between the trends in suicide and in drug poisoning deaths suggest that the explanations for the two causes may differ. Potential explanations for suicide trends are therefore evaluated separately in Chapter 8.

The final category of causes of death evaluated by the committee in detail is mortality due to cardiometabolic diseases. This category encompasses two causes of death that increased among most working-age adults over the study period (hypertensive heart disease and ENM diseases), as well as a cause of death that had previously seen dramatic improvements but on which progress stalled or reversed (the combined category of ischemic heart disease and other circulatory diseases). Although the overall

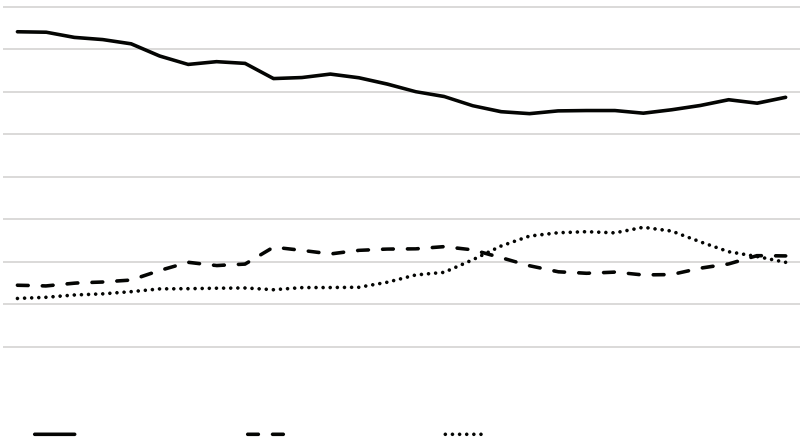


FIGURE 4-3 Percentage of mental and behavioral disorder–related deaths due to alcohol, drugs, and all other causes, ages 25–64, 1990–2017.
NOTE: The figure shows the percentage of all deaths for which mental and behavioral disorders are identified as the underlying cause of death and that can be attributed to alcohol use (solid black line), drug use (dashed black line), and any other cause (dotted black line). See Chapter 5 (Table 5–1) for specific International Classification of Diseases (ICD) codes included in each category.
SOURCE: Data from CDC (2020b).

trends in mortality for the three cardiometabolic causes of death differ in direction, all experienced a common slowdown or reversal of progress in reducing mortality, and there is reason to suspect that they share an underlying proximate cause, such as obesity, that justifies examining them together.

Mortality due to hypertensive heart disease and ENM diseases increased among most working-age males and White females. These increases were larger outside of large central metros, particularly among White males and females, contributing to the growing mortality gap between large central metros and less-populated areas. As discussed at length in Chapter 9, although mortality due to the combined category of ischemic heart disease and other circulatory diseases declined dramatically overall during the study period among working-age adults, these gains slowed and, in some cases, reversed in the 2010s. Even among those working-age adults for whom mortality from this cause continued to decrease, such as Black females, it decreased at a slower rate in that period, and it began to increase among many younger (ages 25–44) adults and older (ages 45–64) White females. Trends in metropolitan areas differed by race and ethnicity but generally contributed to mortality gaps by metropolitan status. Working-age Whites in large central metros continued to see reductions in mortality due to ischemic heart disease and other circulatory diseases, while those in nonmetros were most likely to experience increasing mortality from this set of causes. The combination of these trends contributed to an expanding mortality gap between large central metros and nonmetros among White adults. In contrast, older Black and Hispanic adults in nonmetros experienced larger reductions in mortality relative to those in more populous areas.

As noted above, other causes of death—including infectious and parasitic diseases (excluding HIV/AIDS), liver cancer, diseases of the nervous system, transport accidents, and homicide—contributed to increasing working-age mortality during the 1990–2017 period. Although these other causes of death are not addressed in detail in this report, they do merit attention. The committee therefore included detailed tables with cause-specific trends for these causes of death in Appendix A.

Although some of these other causes of death did not, on their own, contribute meaningfully to the recent alarming increases in mortality, the results presented in Tables 4-1 to 4-3 demonstrate that their combined influence on working-age mortality trends was not trivial. Moreover, important details about deaths from certain causes shed light on their potential role, or lack thereof, in explaining increases in working-age mortality over the 1990–2017 period or ongoing racial/ethnic disparities in mortality. For example, mortality rates for HIV/AIDS, as well as other infectious and parasitic diseases, increased predominantly in the 1990s and subsequently fell; therefore, they do not help explain the current rise in working-age

mortality, although the delayed progress in reducing mortality from HIV/AIDS among older working-age Black males and females has contributed to continuing racial/ethnic disparities in mortality.

In a similar vein, mortality due to transport accidents and homicides decreased overall between 1990 and 2017, primarily as a result of large reductions during the 1990s. In the 2010s, however, these gains began to reverse, particularly among younger Black and Hispanic working-age males, who otherwise experienced decreases in overall mortality during this period. Overall, neither transport accidents nor homicides were a significant contributor to the recent increase in mortality among working-age White males and females, but like the slower progress in reducing mortality from HIV/AIDS among older Black males and females compared with Whites, these causes of death contributed to mortality disparities between younger Black and White males. The reasons for these recent changes in transport accidents and homicides are not well understood. Research suggests that a recent increase in fatal police shootings is a leading cause of homicide among young Black males (Edwards, Lee, and Esposito, 2019), but inconsistency across states in data collection on these shootings makes it difficult to assess whether this increase can explain the rise in homicides among younger Black and Hispanic males.

Mortality due to nervous system diseases increased among working-age adults regardless of age, sex, and race and ethnicity, although these increases were often very small. Recent studies have noted that similar increases in mortality due to nervous system diseases occurred internationally (Pritchard et al., 2017), but the committee is not aware of research offering an explanation for this trend. The increase in deaths from neurologic diseases among the elderly is an expected outcome of an aging population, but reasons for the increase among working-age adults, before age 65, are less clear.

Many of the cause-of-death categories included in this report are broadly defined, often by the body system affected, and encompass a wide range of diseases and disorders. For example, mortality from ischemic heart disease and other circulatory system diseases was examined as a combined group, but "other circulatory system diseases" encompassed all circulatory system diseases besides ischemic heart disease and hypertensive heart disease, including arrhythmias, cardiomyopathy, heart failure, cardiac arrest, stroke, intracerebral hemorrhage, and pulmonary embolism. Because the committee's purpose was to identify the key drivers of recent changes in working-age mortality rather than to fully explore recent changes in all causes of death, this report focuses on mortality trends for entire body systems, such as the nervous, genitourinary, respiratory, and digestive systems, without detailing more pronounced increases in mortality from specific diseases within these body systems. However, readers should note that within each of the broad cause-of-death categories, there may be heterogeneity in the

magnitude—and sometimes the direction—of the changes in cause-specific mortality that occurred between 1990 and 2017, particularly for rare causes of death that result in only a small number of deaths per year among working-age adults.

It will be important for future research to seek explanations for the increase in working-age mortality across these conditions. To some extent, increased death rates in working age may be coincidental and reflect independent causal pathways. For example, increases in working-age mortality from cerebral palsy may reflect medical advances that have enabled children with these conditions to survive into adulthood (Woolf et al., 2018). Increased cellphone use could contribute to the increase in transport injuries but would not explain deaths from chronic diseases.

Another possibility is that some increases in working-age mortality may be secondary to the primary causes this report examines—drugs, alcohol, suicide, and cardiometabolic diseases. Deaths from other causes that stem from these primary causes might be considered "collateral" deaths. Mortality due to liver cancer, for example, increased among older working-age adults (ages 45–64) regardless of race and ethnicity. This increase could potentially have resulted from several underlying causes, including increasing alcohol and drug use, as well as viral hepatitis, diabetes, and non-alcohol-induced fatty liver disease. This complexity in potential etiology complicates any attempt to use the trends in liver cancer to evaluate potential explanations for the increase in alcohol- and drug-related deaths. However, to the extent that the recent increases in mortality due to liver cancer are linked to drug and alcohol use or diabetes, the explanations for these trends in Chapters 7 and 9, respectively, may be relevant.

This "collateral" mortality effect could also have contributed to changes in more detailed causes of death contained within the 20 broad cause-of-death categories examined in this chapter. For example, increased use of injection drugs could explain not only overdose deaths but also increases in deaths from viral hepatitis, infectious valvular heart diseases, and other drug-related complications. Likewise, alcohol use can increase an individual's risk of death from atrial fibrillation and other arrhythmias, transport accidents, and other causes for which working-age mortality has increased. People who initially survive a suicide attempt may die in the hospital from secondary complications. And obesity and other contributors to deaths from cardiometabolic diseases could help explain increased mortality from renal failure.

It is difficult, however, to identify all of the detailed causes of death that could potentially be considered collateral consequences of the larger trends in substance use, suicide, and cardiometabolic diseases. In addition, because death is the result of complex processes that unfold over the life course, trends in mortality are rarely so simply explained. Despite these

complications, the evidence gaps noted above and the loss of life involved provide a strong argument for a research agenda to seek the underlying explanations for the large number of causes of death for which mortality has been increasing. Of necessity, the next chapters focus on the main drivers of increasing working-age mortality—drugs, alcohol, suicide, and cardiometabolic diseases—but the committee encourages the research community to continue the work of exploring the explanations underlying increases in working-age mortality due to the range of other causes of death identified in this report.

ANNEX 4-1

Trends in Cause-Specific Mortality Among American Indians and Alaska Natives

The American Indian/Alaska Native (AI/AN) population has the highest mortality rates for more causes of death of all racial/ethnic groups. Epsey and colleagues (2014) (reproduced in Sancar, Abbasi, and Bucher, 2017) present leading causes of death for 1999–2009 among AI/ANs living in 637 Contract Health Service Delivery Areas (CHSDAs). These data were taken from death certificates, corrected for misclassification of AI/AN identity and from National Vital Statistics Reports for 2017 for the entire United States, without such correction. Annex Figure 4-1 presents the leading causes of death for the CHSDA counties in 1999–2009. AI/AN individuals are more likely to die from diabetes, chronic liver disease, and suicide than are Whites, and also more likely to die from these causes than are non-Hispanic Blacks and Hispanics.

	FEMALE		MALE	
	AI/AN	W	AI/AN	W
Cancer	1	2	2	2
Heart Disease	2	1	1	1
Accidents	3	6	3	4
Diabetes mellitus	4	8	4	6
Stroke	5	3	8	5
Chronic liver disease	6	12	5	10
Chronic lower respiratory disease	7	4	7	3
Influenza and pneumonia	8	7	10	8
Kidney disease	9	9	11	11
Septicemia	10	10	12	13
Alzheimer disease	11	5		
Suicide	12	16	6	7
Assault (homicide)			9	19

ANNEX FIGURE 4-1 Leading causes of death in Contract Health Service Delivery Area (CHSDA) counties in 1999–2009.
SOURCE: Espey et al. (2014).

CAUSE-SPECIFIC MORTALITY TRENDS BY METROPOLITAN AREA STATUS

ANNEX TABLE 4-1 Absolute Change in Cause-Specific Mortality and Percentage of Total Increase or Decrease in Mortality by Size of Metropolitan Area, 1990–1993 to 2015–2017: Non-Hispanic White Adults

MALES	Ages 25–44				Ages 45–64			
	Large Central Metro	Large Fringe Metro	Small or Medium Metro	Nonmetro	Large Central Metro	Large Fringe Metro	Small or Medium Metro	Nonmetro
All-Cause Mortality Rate 1990–1993	224.09	196.33	195.06	206.06	838.81	830.48	865.54	878.35
All-Cause Mortality Rate 2015–2017	198.33	238.70	236.00	267.70	643.75	767.65	812.83	872.60
Change 1990–1993 to 2015–2017	–25.76	42.37	40.94	61.63	–195.06	–62.83	–52.71	–5.75
(Change as % of 1990–1993 mortality)	–11.50	21.58	20.99	29.91	–23.25	–7.57	–6.09	–0.65

Cause of Death	Abs Chg	% of Total +/– Chg	Abs Chg	% of Total +/– Chg	Abs Chg	% of Total +/– Chg	Abs Chg	% of Total +/– Chg	Abs Chg	% of Total +/– Chg	Abs Chg	% of Total +/– Chg	Abs Chg	% of Total +/– Chg	Abs Chg	% of Total +/– Chg
Infectious and Parasitic Diseases																
HIV/AIDS	–51.60	–57.9	–23.30	–55.5	–16.18	–44.9	–10.39	–41.1	–27.71	–9.2	–8.42	–3.8	–5.14	–2.4	–3.09	–1.7
Non-HIV/AIDS	–2.36	–2.6	0.13	0.1	0.63	0.8	1.58	1.8	6.20	5.8	12.20	7.6	12.17	7.5	14.61	8.4
Cancers																
Liver Cancer	0.00	0.0	–0.02	0.0	0.18	0.2	0.11	0.1	6.72	6.3	10.02	6.2	9.62	5.9	9.75	5.6
Lung Cancer	–2.88	–3.2	–2.55	–6.1	–2.62	–7.3	–2.44	–9.7	–66.59	–22.1	–58.74	–26.3	–61.14	–28.5	–51.07	–28.4
All Other Cancers	–7.48	–8.4	–5.33	–12.7	–4.36	–12.1	–3.07	–12.1	–46.97	–15.6	–27.35	–12.2	–22.27	–10.4	–12.66	–7.0

Cardio and Metabolic Diseases																
Endocrine, Nutritional, & Metabolic	0.24	0.4	3.02	3.6	3.33	4.3	4.53	5.2	9.51	8.9	16.72	10.4	17.55	10.8	21.87	12.6
Hypertensive Heart Disease	2.35	3.7	3.32	3.9	2.73	3.6	3.43	3.9	11.08	10.4	15.28	9.5	14.79	9.1	15.51	8.9
Ischemic & Other Circulatory System	–9.09	–10.2	–5.28	–12.6	–4.12	–11.4	0.42	0.5	–146.78	–48.7	–126.26	–56.5	–124.69	–58.0	–112.26	–62.4
Substance Use and Mental Health																
Drug Poisoning	54.32	85.6	58.84	69.7	46.76	60.7	46.21	53.1	40.15	37.8	42.73	26.6	32.55	20.1	30.68	17.6
Alcohol-Induced*	–0.74	–0.8	1.40	1.7	1.91	2.5	1.81	2.1	4.05	3.8	9.82	6.1	11.05	6.8	9.09	5.2
Suicide	4.99	7.9	12.29	14.6	12.73	16.5	16.16	18.6	8.74	8.2	12.04	7.5	14.11	8.7	14.56	8.4
Mental & Behavioral Disorders	0.91	1.4	1.66	2.0	2.43	3.2	1.80	2.1	5.93	5.6	9.53	5.9	10.64	6.6	6.23	3.6
Other Body System Diseases																
Nervous System	0.63	1.0	2.14	2.5	2.20	2.9	2.41	2.8	6.17	5.8	8.97	5.6	10.37	6.4	10.13	5.8
Genitourinary System	–0.13	–0.1	0.48	0.6	0.74	1.0	0.96	1.1	2.30	2.2	4.11	2.6	4.90	3.0	6.27	3.6
Respiratory System	–1.49	–1.7	0.34	0.4	0.98	1.3	1.93	2.2	–6.21	–2.1	2.36	1.5	2.41	1.5	9.87	5.7
Digestive System*	–2.15	–2.4	–0.06	–0.2	0.10	0.1	1.55	1.8	–1.14	–0.4	6.56	4.1	9.83	6.1	12.49	7.2
Other Causes of Death																
Homicide	–4.01	–4.5	–1.64	–3.9	–0.53	–1.5	–1.40	–5.5	–3.00	–1.0	–0.92	–0.4	–1.64	–0.8	–0.86	–0.5
Transport Injuries	–5.05	–5.7	–3.84	–9.1	–6.70	–18.6	–5.52	–21.8	0.85	0.8	3.40	2.1	4.29	2.6	5.03	2.9
Other External Causes of Death	–1.30	–1.5	0.25	0.3	–1.54	–4.3	–2.47	–9.8	4.59	4.3	6.72	4.2	5.56	3.4	4.94	2.8

continued

ANNEX TABLE 4-1 Continued

All Other Causes of Death	–0.90	–1.0	0.52	0.6	2.28	3.0	4.05	4.7	–2.94	–1.0	–1.60	–0.7	2.33	1.4	3.17	1.8
Total Change: Increase (+)	63.43		84.39		76.99		86.94		106.29		160.47		162.17		174.20	
Total Change: Decrease (–)	–89.19		–42.01		–36.05		–25.31		–301.35		–223.29		–214.88		–179.94	

FEMALES	Ages 25–44				Ages 45–64			
	Large Central Metro	Large Fringe Metro	Small or Medium Metro	Nonmetro	Large Central Metro	Large Fringe Metro	Small or Medium Metro	Nonmetro
All-Cause Mortality Rate 1990–1993	87.28	87.77	90.17	94.04	483.81	474.45	490.11	485.17
All-Cause Mortality Rate 2015–2017	103.91	132.37	132.61	156.66	396.31	471.08	503.41	557.48
Change 1990–1993 to 2015–2017	16.63	44.60	42.43	62.63	–87.50	–3.37	13.29	72.31
(Change as % of 1990–1993 mortality)	19.05	50.81	47.06	66.60	–18.08	–0.71	2.71	14.90

Cause of Death	Abs Chg	% of Total +/– Chg	Abs Chg	% of Total +/– Chg	Abs Chg	% of Total +/– Chg	Abs Chg	% of Total +/– Chg	Abs Chg	% of Total +/– Chg	Abs Chg	% of Total +/– Chg	Abs Chg	% of Total +/– Chg	Abs Chg	% of Total +/– Chg
Infectious and Parasitic Diseases																
HIV/AIDS	–2.70	–14.7	–1.18	–10.6	–0.90	–7.6	–0.43	–6.1	–0.69	–0.5	–0.24	–0.2	–0.08	–0.1	0.13	0.1
Non-HIV/AIDS	0.41	1.2	1.20	2.2	1.42	2.6	2.58	3.7	5.08	8.4	7.43	7.4	8.66	7.8	10.59	7.8
Cancers																
Liver Cancer	0.13	0.4	0.25	0.4	0.08	0.2	0.10	0.1	1.77	2.9	1.96	2.0	2.23	2.0	2.32	1.7
Lung Cancer	–1.98	–10.8	–1.52	–13.7	–1.02	–8.5	–0.72	–10.1	–31.63	–21.4	–20.18	–19.5	–17.77	–18.3	–5.75	–9.1
All Other Cancers	–8.72	–47.5	–6.37	–57.3	–7.59	–63.5	–5.66	–79.9	–62.00	–41.9	–45.07	–43.5	–46.05	–47.3	–33.41	–53.0
Cardio and Metabolic Diseases																
Endocrine, Nutritional, & Metabolic	0.84	2.4	2.06	3.7	2.15	4.0	4.51	6.5	1.61	2.6	5.19	5.2	6.75	6.1	9.92	7.3
Hypertensive Heart Disease	0.95	2.7	1.54	2.8	1.57	2.9	1.98	2.8	4.66	7.7	6.68	6.7	6.55	5.9	8.07	6.0
Ischemic & Other Circulatory System	–1.30	–7.1	1.95	3.5	3.20	5.9	6.77	9.7	–50.49	–34.1	–36.65	–35.4	–32.76	–33.7	–23.66	–37.6
Substance Use and Mental Health																
Drug Poisoning	24.73	70.7	30.63	55.0	25.58	47.0	27.23	39.1	23.64	39.0	27.91	27.8	25.61	23.2	26.92	19.9
Alcohol-Induced*	0.94	2.7	2.12	3.8	2.35	4.3	2.09	3.0	4.68	7.7	7.20	7.2	7.73	7.0	6.58	4.9

continued

ANNEX TABLE 4-1 Continued

Cause of Death	Abs Chg	% of Total +/– Chg	Abs Chg	% of Total +/– Chg	Abs Chg	% of Total +/– Chg	Abs Chg	% of Total +/– Chg	Abs Chg	% of Total +/– Chg	Abs Chg	% of Total +/– Chg	Abs Chg	% of Total +/– Chg	Abs Chg	% of Total +/– Chg
Suicide	2.24	6.4	4.48	8.0	4.18	7.7	4.06	5.8	2.42	4.0	3.86	3.9	3.73	3.4	4.26	3.1
Mental & Behavioral Disorders	1.53	4.4	2.25	4.0	2.32	4.3	2.39	3.4	3.90	6.4	4.58	4.6	5.09	4.6	4.77	3.5
Other Body System Diseases																
Nervous System	0.53	1.5	1.43	2.6	2.06	3.8	2.29	3.3	4.84	8.0	7.53	7.5	8.37	7.6	9.19	6.8
Genitourinary System	0.20	0.6	0.64	1.1	0.34	0.6	1.18	1.7	1.48	2.4	3.55	3.5	4.69	4.2	5.12	3.8
Respiratory System	0.09	0.3	1.48	2.7	2.07	3.8	3.41	4.9	–0.71	–0.5	9.24	9.2	13.49	12.2	23.42	17.3
Digestive System*	0.34	1.0	1.59	2.9	1.66	3.1	2.60	3.7	0.95	1.6	6.10	6.1	7.45	6.7	11.09	8.2
Other Causes of Death																
Homicide	–1.42	–7.7	–0.76	–6.9	–1.07	–8.9	–0.28	–3.9	–0.63	–0.4	–0.52	–0.5	–0.28	–0.3	–0.16	–0.3
Transport Injuries	–2.25	–12.2	–1.28	–11.5	–1.37	–11.4	1.17	1.7	–1.92	–1.3	–0.90	–0.9	–0.35	–0.4	0.35	0.3
Other External Causes of Death	0.97	2.8	1.50	2.7	1.61	3.0	1.81	2.6	3.32	5.5	4.40	4.4	4.52	4.1	5.05	3.7
All Other Causes of Death	1.10	3.2	2.61	4.7	3.78	6.9	5.54	7.9	2.23	3.7	4.58	4.6	5.71	5.2	7.49	5.5
Total Change: Increase (+)	35.01		55.71		54.38		69.70		60.58		100.20		110.58		135.28	
Total Change: Decrease (–)	–18.38		–11.12		–11.95		–7.08		–148.08		–103.57		–97.28		–62.98	

*Changes in the International Classification of Diseases (ICD)-10 code for diseases of the digestive system in 2006 affected the comparability of both digestive system deaths and alcohol-induced deaths before and after that year. More information is provided in Chapter 5.
NOTE: The top 3 causes of death for each age group in each period are highlighted in bolded red text.
Light orange highlights indicate an absolute increase in the overall mortality rate of ≥5 deaths/100,000 population.
Dark orange highlights indicate that a cause of death is responsible for ≥10 percent of the total increase in mortality.
Light green highlights indicate an absolute decrease in the overall mortality rate of ≥5 deaths/100,000 population.
Dark green highlights indicate that a cause of death is responsible for ≥10 percent of the total decrease in mortality.
The table shows the change in all-cause and cause-specific mortality rates among non-Hispanic White working-age males (upper table) and females (lower table) by age group (25–44 and 45–64) and size of metropolitan area (large central metropolitan area, large fringe metropolitan area, small/medium metropolitan area, and nonmetropolitan area). These causes of death are exhaustive of all underlying cause-of-death codes and are based on the International Classification of Diseases (ICD)-9 (1990–1998) and ICD-10 (1999–2017) codes. Mortality rates within each age group are age-adjusted by single-year of age to match the age distribution of the U.S. population in 2000 in order to improve comparability over time. More information about the classification of causes of death can be found in Chapter 5.
SOURCE: Data from https://www.cdc.gov/nchs/nvss/nvss-restricted-data.htm.

ANNEX TABLE 4-2 Absolute Change in Cause-Specific Mortality and Percentage of Total Increase or Decrease in Mortality by Size of Metropolitan Area, 1990–1993 to 2015–2017: Non-Hispanic Black Adults

MALES	Ages 25–44				Ages 45–64			
	Large Central Metro	Large Fringe Metro	Small or Medium Metro	Nonmetro	Large Central Metro	Large Fringe Metro	Small or Medium Metro	Nonmetro
All-Cause Mortality Rate 1990–1993	642.70	477.67	430.49	466.44	1728.58	1622.77	1717.77	1809.05
All-Cause Mortality Rate 2015–2017	314.95	336.96	323.99	316.37	1033.27	1128.13	1176.07	1255.89
Change 1990–1993 to 2015–2017	–327.75	–140.71	–106.50	–150.07	–695.31	–494.64	–541.70	–553.16
(Change as % of 1990–1993 mortality)	–51.00	–29.46	–24.74	–32.17	–40.22	–30.48	–31.53	–30.58

Cause of Death	Abs Chg	% of Total +/– Chg	Abs Chg	% of Total +/– Chg	Abs Chg	% of Total +/– Chg	Abs Chg	% of Total +/– Chg	Abs Chg	% of Total +/– Chg	Abs Chg	% of Total +/– Chg	Abs Chg	% of Total +/– Chg	Abs Chg	% of Total +/– Chg
Infectious and Parasitic Diseases																
HIV/AIDS	–162.04	–47.2	–81.50	–45.7	–45.50	–30.4	–39.25	–22.1	–77.72	–10.1	–26.20	–4.3	–10.06	–1.6	–7.98	–1.2
Non-HIV/AIDS	–12.25	–3.6	–4.90	–2.7	–2.80	–1.9	–0.96	–0.5	–4.33	–0.6	12.25	11.2	8.95	10.4	14.35	14.9

Cancers																
Liver Cancer	–0.55	–0.2	–1.18	–0.7	–0.23	–0.2	–0.53	–0.3	9.88	13.9	18.67	17.0	12.89	14.9	12.74	13.2
Lung Cancer	–8.09	–2.4	–7.39	–4.1	–6.04	–4.0	–8.52	–4.8	–134.27	–17.5	–124.09	–20.5	–131.94	–21.0	–117.10	–18.0
All Other Cancers	–14.93	–4.3	–13.49	–7.6	–11.47	–7.7	–13.93	–7.9	–111.57	–14.6	–94.25	–15.6	–108.05	–17.2	–83.64	–12.9
Cardio and Metabolic Diseases																
Endocrine, Nutritional, & Metabolic	0.38	2.4	4.81	12.8	10.49	24.3	6.26	22.9	3.58	5.1	13.13	12.0	5.77	6.7	17.77	18.4
Hypertensive Heart Disease	4.18	26.6	7.13	19.0	5.95	13.8	6.00	22.0	1.28	1.8	10.33	9.4	10.95	12.7	14.99	15.5
Ischemic & Other Circulatory System	–28.35	–8.3	–17.41	–9.8	–20.47	–13.7	–30.82	–17.4	–256.29	–33.5	–235.33	–38.9	–254.34	–40.5	–299.87	–46.2
Substance Use and Mental Health																
Drug Poisoning	11.05	70.3	22.90	61.1	24.57	57.0	13.34	48.8	50.97	71.9	39.01	35.6	29.02	33.6	19.22	19.9
Alcohol-Induced*	–12.07	–3.5	–9.45	–5.3	–8.81	–5.9	–8.91	–5.0	–26.93	–3.5	–18.42	–3.0	–18.61	–3.0	–13.97	–2.1
Suicide	–2.03	–0.6	1.09	2.9	–2.70	–1.8	–1.70	–1.0	–2.65	–0.3	–0.44	–0.1	0.44	0.5	–2.83	–0.4
Mental & Behavioral Disorders	–12.03	–3.5	–7.93	–4.4	–6.72	–4.5	–8.77	–4.9	–18.97	–2.5	–13.54	–2.2	–15.63	–2.5	–18.73	–2.9
Other Body System Diseases																
Nervous System	0.10	0.7	1.06	2.8	1.65	3.8	0.65	2.4	5.14	7.2	8.96	8.2	11.32	13.1	8.96	9.3

continued

ANNEX TABLE 4-2 Continued

Genitourinary System	–4.49	–1.3	0.50	1.3	0.44	1.0	0.90	3.3	–3.88	–0.5	7.36	6.7	7.14	8.3	8.48	8.8
Respiratory System	–13.85	–4.0	–4.84	–2.7	–4.10	–2.7	–4.51	–2.5	–37.46	–4.9	–24.95	–4.1	–15.87	–2.5	–22.45	–3.5
Digestive System*	–12.84	–3.7	–7.46	–4.2	–7.59	–5.1	–6.22	–3.5	–27.37	–3.6	–15.86	–2.6	–11.26	–1.8	–9.43	–1.5
Other Causes of Death																
Homicide	–34.09	–9.9	–0.65	–0.4	–10.80	–7.2	–13.36	–7.5	–19.44	–2.5	–9.25	–1.5	–17.15	–2.7	–17.44	–2.7
Transport Injuries	–1.40	–0.4	–3.04	–1.7	–4.82	–3.2	–20.26	–11.4	–2.80	–0.4	–3.50	–0.6	–6.62	–1.1	–6.78	–1.0
Other External Causes of Death	–10.21	–3.0	–12.44	–7.0	–16.07	–10.7	–19.66	–11.1	–10.85	–1.4	–11.45	–1.9	–15.20	–2.4	–22.70	–3.5
All Other Causes of Death	–14.23	–4.1	–6.54	–3.7	–1.49	–1.0	0.16	0.6	–31.65	–4.1	–27.07	–4.5	–23.44	–3.7	–26.77	–4.1
Total Change: Increase (+)	15.71		37.51		43.10		27.32		70.86		109.71		86.48		96.51	
Total Change: Decrease (–)	–343.46		–178.21		–149.60		–177.39		–766.17		–604.35		–628.18		–649.68	

FEMALES	Ages 25–44				Ages 45–64			
	Large Central Metro	Large Fringe Metro	Small or Medium Metro	Nonmetro	Large Central Metro	Large Fringe Metro	Small or Medium Metro	Nonmetro
All-Cause Mortality Rate 1990–1993	252.59	218.15	217.28	231.14	919.69	889.80	948.43	951.95
All-Cause Mortality Rate 2015–2017	150.31	180.52	184.40	217.15	640.65	701.81	762.33	805.23
Change 1990–1993 to 2015–2017	–102.28	–37.62	–32.88	–13.99	–279.04	–187.99	–186.10	–146.71
(Change as % of 1990–1993 mortality)	–40.49	–17.25	–15.13	–6.05	–30.34	–21.13	–19.62	–15.41

Cause of Death	Abs Chg	% of Total +/– Chg	Abs Chg	% of Total +/– Chg	Abs Chg	% of Total +/– Chg	Abs Chg	% of Total +/– Chg	Abs Chg	% of Total +/– Chg	Abs Chg	% of Total +/– Chg	Abs Chg	% of Total +/– Chg	Abs Chg	% of Total +/– Chg
Infectious and Parasitic Diseases																
HIV/AIDS	–35.37	–31.4	–16.95	–26.6	–7.03	–10.7	–5.16	–10.4	–6.61	–2.1	2.83	5.1	1.67	2.5	4.64	6.5
Non-HIV/AIDS	–4.08	–3.6	–2.00	–3.1	–2.31	–3.5	2.95	8.3	1.79	5.5	8.67	15.5	11.32	16.7	11.65	16.4
Cancers																
Liver Cancer	0.00	0.0	–0.16	–0.3	–0.01	0.0	–0.22	–0.5	3.11	9.6	4.96	8.9	2.19	3.2	2.84	4.0
Lung Cancer	–3.55	–3.2	–3.15	–4.9	–3.52	–5.4	–1.97	–4.0	–34.61	–11.1	–23.38	–9.6	–26.96	–10.6	–12.41	–5.7
All Other Cancers	–14.26	–12.7	–11.25	–17.7	–11.10	–16.9	–16.42	–33.2	–77.78	–25.0	–62.91	–25.8	–71.53	–28.2	–41.80	–19.2

continued

ANNEX TABLE 4-2 Continued

Cardio and Metabolic Diseases																
Endocrine, Nutritional, & Metabolic	0.99	9.7	6.51	25.0	8.44	25.9	9.23	26.0	–11.21	–3.6	–12.12	–5.0	–3.65	–1.4	–3.58	–1.6
Hypertensive Heart Disease	1.13	11.1	3.42	13.1	4.21	12.9	3.45	9.7	–6.99	–2.2	2.71	4.9	–4.48	–1.8	2.93	4.1
Ischemic & Other Circulatory System	–15.89	–14.1	–7.93	–12.4	–16.67	–25.5	–7.11	–14.4	–139.59	–44.8	–130.14	–53.4	–127.93	–50.4	–145.76	–67.0
Substance Use and Mental Health																
Drug Poisoning	6.68	65.5	10.37	39.8	10.66	32.7	6.76	19.0	21.16	65.4	15.30	27.3	14.01	20.7	10.15	14.3
Alcohol-Induced*	–4.01	–3.6	–3.28	–5.2	–2.61	–4.0	–3.59	–7.3	–4.97	–1.6	–4.50	–1.8	–4.06	–1.6	–3.26	–1.5
Suicide	0.26	2.5	0.68	2.6	0.84	2.6	0.77	2.2	–0.25	–0.1	0.05	0.1	–0.99	–0.4	–0.31	–0.1
Mental & Behavioral Disorders	–3.78	–3.4	–2.51	–3.9	–1.93	–2.9	–3.13	–6.3	–1.45	–0.5	–1.99	–0.8	–3.50	–1.4	–0.79	–0.4
Other Body System Diseases																
Nervous System	1.15	11.2	2.79	10.7	2.46	7.5	2.82	7.9	6.28	19.4	10.19	18.2	10.24	15.1	11.90	16.8
Genitourinary System	–0.70	–0.6	1.43	5.5	1.45	4.4	3.66	10.3	–2.33	–0.7	6.43	11.5	8.74	12.9	6.08	8.6
Respiratory System	–6.59	–5.9	–1.25	–2.0	–1.97	–3.0	–0.36	–0.7	–5.20	–1.7	1.77	3.2	13.01	19.2	13.47	19.0
Digestive System*	–4.74	–4.2	–2.61	–4.1	–2.08	–3.2	–1.42	–2.9	–10.82	–3.5	–5.08	–2.1	–3.72	–1.5	–1.13	–0.5

Other Causes of Death															
Homicide	–12.88	–11.5	–10.87	–17.1	–10.14	–15.5	–8.63	–17.4	–3.59	–1.2	–3.22	–1.3	–1.59	–0.6	–3.14	–1.4
Transport Injuries	–1.61	–1.4	–1.04	–1.6	–2.98	–4.5	0.84	2.4	–1.90	–0.6	–0.58	–0.2	–0.76	–0.3	–3.28	–1.5
Other External Causes of Death	–1.56	–1.4	–0.69	–1.1	–3.11	–4.8	–1.50	–3.0	–1.23	–0.4	0.60	1.1	–4.67	–1.8	–2.13	–1.0
All Other Causes of Death	–3.45	–3.1	0.89	3.4	4.53	13.9	5.03	14.2	–2.84	–0.9	2.43	4.3	6.54	9.7	7.21	10.2
Total Change: Increase (+)	10.20		26.06		32.58		35.51		32.34		55.93		67.72		70.86	
Total Change: Decrease (–)	–112.48		–63.69		–65.46		–49.51		–311.38		–243.92		–253.82		–217.58	

*Changes in the International Classification of Diseases (ICD)-10 code for diseases of the digestive system in 2006 affected the comparability of both digestive system deaths and alcohol-induced deaths before and after that year. More information is provided in Chapter 5.
NOTE: The top 3 causes of death for each age group in each period are highlighted in **bolded red** text.
Light orange highlights indicate an absolute increase in the overall mortality rate of ≥5 deaths/100,000 population.
Dark orange highlights indicate that a cause of death is responsible for ≥10 percent of the total increase in mortality.
Light green highlights indicate an absolute decrease in the overall mortality rate of ≥5 deaths/100,000 population.
Dark green highlights indicate that a cause of death is responsible for ≥10 percent of the total decrease in mortality.
The table shows the change in all-cause and cause-specific mortality rates among non-Hispanic Black working-age males (upper table) and females (lower table) by age group (25–44 and 45–64) and size of metropolitan area (large central metropolitan area, large fringe metropolitan area, small/medium metropolitan area, and nonmetropolitan area). These causes of death are exhaustive of all underlying cause-of-death codes and are based on the International Classification of Diseases (ICD)-9 (1990–1998) and ICD-10 (1999–2017) codes. Mortality rates within each age group are age-adjusted by single-year of age to match the age distribution of the U.S. population in 2000 in order to improve comparability over time. More information about the classification of causes of death can be found in Chapter 5.
SOURCE: Data from https://www.cdc.gov/nchs/nvss/nvss-restricted-data.htm.

ANNEX TABLE 4-3 Absolute Change in Cause-Specific Mortality and Percentage of Total Increase or Decrease in Mortality by Size of Metropolitan Area, 1990–1993 to 2015–2017: Hispanic Adults

MALES	Ages 25–44				Ages 45–64			
	Large Central Metro	Large Fringe Metro	Small or Medium Metro	Nonmetro	Large Central Metro	Large Fringe Metro	Small or Medium Metro	Nonmetro
All-Cause Mortality Rate 1990–1993	305.04	245.27	220.74	241.11	743.83	731.25	699.50	783.61
All-Cause Mortality Rate 2015–2017	136.69	165.00	156.08	165.45	493.84	604.94	571.72	614.95
Change 1990–1993 to 2015–2017 (Change as % of 1990–1993 mortality)	–168.35	–80.28	–64.67	–75.66	–249.99	–126.30	–127.78	–168.67
	–55.19	–32.73	–29.30	–31.38	–33.61	–17.27	–18.27	–21.52

Cause of Death	Abs Chg	% of Total +/– Chg	Abs Chg	% of Total +/– Chg	Abs Chg	% of Total +/– Chg	Abs Chg	% of Total +/– Chg	Abs Chg	% of Total +/– Chg	Abs Chg	% of Total +/– Chg	Abs Chg	% of Total +/– Chg	Abs Chg	% of Total +/– Chg
Infectious and Parasitic Diseases																
HIV/AIDS	–86.08	–48.9	–31.87	–34.9	–20.92	–25.6	–14.90	–16.2	–54.30	–18.6	–10.18	–5.4	–12.18	–6.4	–5.14	–2.2
Non-HIV/AIDS	–5.29	–3.0	–1.42	–1.6	–1.35	–1.6	–0.53	–0.6	3.74	8.9	11.34	18.5	11.98	19.2	13.81	21.4
Cancers																
Liver Cancer	–0.39	–0.2	–0.04	0.0	–0.45	–0.5	0.48	3.0	8.21	19.4	12.74	20.8	13.01	20.9	14.16	22.0
Lung Cancer	–1.59	–0.9	–0.48	–0.5	0.16	1.0	–1.64	–1.8	–26.36	–9.0	–24.47	–13.0	–24.03	–12.6	–29.55	–12.7
All Other Cancers	–4.04	–2.3	–3.28	–3.6	–3.93	–4.8	–3.98	–4.3	–25.68	–8.8	–27.68	–14.7	–22.46	–11.8	–20.00	–8.6

Cardio and Metabolic Diseases																
Endocrine, Nutritional, & Metabolic	0.32	4.2	0.37	3.3	2.35	13.8	1.09	6.7	4.87	11.5	0.96	1.6	–9.40	–4.9	–0.75	–0.3
Hypertensive Heart Disease	1.36	17.6	1.68	15.1	1.09	6.4	2.30	14.2	4.52	10.7	9.08	14.8	6.66	10.7	6.79	10.5
Ischemic & Other Circulatory System	–8.06	–4.6	–5.16	–5.6	–5.65	–6.9	–4.11	–4.5	–108.74	–37.2	–94.01	–50.1	–83.15	–43.7	–131.73	–56.5
Substance Use and Mental Health																
Drug Poisoning	6.01	78.2	8.44	75.6	8.30	48.6	11.02	68.1	15.14	35.9	18.98	30.9	18.61	29.9	15.29	23.7
Alcohol-Induced*	–7.03	–4.0	–5.04	–5.5	–3.80	–4.6	–6.84	–7.4	–14.79	–5.1	–3.21	–1.7	0.41	0.7	–7.06	–3.0
Suicide	–1.79	–1.0	–1.47	–1.6	2.68	15.7	–3.01	–3.3	–4.40	–1.5	–0.14	–0.1	–5.33	–2.8	–3.36	–1.4
Mental & Behavioral Disorders	–4.49	–2.5	–3.87	–4.2	–4.88	–6.0	–3.32	–3.6	–4.67	–1.6	–4.85	–2.6	1.08	1.7	–3.13	–1.3
Other Body System Diseases																
Nervous System	–0.16	–0.1	0.56	5.0	2.28	13.4	1.07	6.6	3.00	7.1	5.08	8.3	5.99	9.6	4.28	6.6
Genitourinary System	–0.51	–0.3	0.12	1.1	0.18	1.1	0.23	1.4	2.39	5.7	1.28	2.1	4.30	6.9	7.70	12.0
Respiratory System	–4.02	–2.3	–1.67	–1.8	0.04	0.2	–1.98	–2.2	–10.84	–3.7	–7.04	–3.8	–5.11	–2.7	–3.46	–1.5
Digestive System*	–4.97	–2.8	–4.29	–4.7	–1.89	–2.3	–1.82	–2.0	–13.10	–4.5	0.36	0.6	–6.68	–3.5	2.34	3.6

continued

ANNEX TABLE 4-3 Continued

Cause of Death	Abs Chg	% of Total +/– Chg	Abs Chg	% of Total +/– Chg	Abs Chg	% of Total +/– Chg	Abs Chg	% of Total +/– Chg	Abs Chg	% of Total +/– Chg	Abs Chg	% of Total +/– Chg	Abs Chg	% of Total +/– Chg	Abs Chg	% of Total +/– Chg
Other Causes of Death																
Homicide	–31.86	–18.1	–13.15	–14.4	–11.69	–14.3	–13.57	–14.8	–17.63	–6.0	–6.02	–3.2	–4.89	–2.6	–8.28	–3.6
Transport Injuries	–8.66	–4.9	–15.28	–16.7	–22.63	–27.7	–25.82	–28.1	–6.93	–2.4	–10.01	–5.3	–15.16	–8.0	–18.44	–7.9
Other External Causes of Death	–3.68	–2.1	–2.99	–3.3	–3.75	–4.6	–10.32	–11.2	0.35	0.8	–0.06	0.0	–1.65	–0.9	0.03	0.0
All Other Causes of Death	–3.42	–1.9	–1.41	–1.5	–0.83	–1.0	–0.03	0.0	–4.78	–1.6	1.55	2.5	0.25	0.4	–2.14	–0.9
Total Change: Increase (+)	7.69		11.16		17.09		16.20		42.23		61.36		62.28		64.39	
Total Change: Decrease (–)	–176.04		–91.44		–81.76		–91.85		–292.22		–187.67		–190.06		–233.06	

FEMALES	Ages 25–44				Ages 45–64			
	Large Central Metro	Large Fringe Metro	Small or Medium Metro	Nonmetro	Large Central Metro	Large Fringe Metro	Small or Medium Metro	Nonmetro
All-Cause Mortality Rate 1990–1993	90.29	84.27	86.95	92.35	368.11	402.60	416.57	450.12
All-Cause Mortality Rate 2015–2017	60.57	77.35	77.73	89.30	266.90	327.26	309.09	364.25
Change 1990–1993 to 2015–2017	–29.71	–6.92	–9.22	–3.05	–101.21	–75.33	–107.49	–85.87
(Change as % of 1990–1993 mortality)	–32.91	–8.21	–10.60	–3.30	–27.49	–18.71	–25.80	–19.08

Cause of Death	Abs Chg	% of Total +/– Chg	Abs Chg	% of Total +/– Chg	Abs Chg	% of Total +/– Chg	Abs Chg	% of Total +/– Chg	Abs Chg	% of Total +/– Chg	Abs Chg	% of Total +/– Chg	Abs Chg	% of Total +/– Chg	Abs Chg	% of Total +/– Chg
Infectious and Parasitic Diseases																
HIV/AIDS	–14.03	–41.0	–5.60	–29.0	–2.97	–12.7	–0.95	–4.9	–6.70	–5.9	–0.61	–0.6	–0.59	–0.4	0.21	0.6
Non-HIV/AIDS	–1.68	–4.9	0.42	3.4	0.32	2.3	0.63	3.8	0.69	5.8	4.78	16.6	7.52	28.1	4.00	10.9
Cancers																
Liver Cancer	0.12	2.7	0.11	0.9	–0.08	–0.3	0.14	0.8	1.89	15.8	1.12	3.9	1.49	5.6	2.15	5.9
Lung Cancer	–0.49	–1.4	–0.05	–0.3	0.52	3.7	0.29	1.8	–4.42	–3.9	–6.68	–6.4	–6.39	–4.8	–7.15	–5.8

continued

ANNEX TABLE 4-3 Continued

Cause of Death	Abs Chg	% of Total +/– Chg	Abs Chg	% of Total +/– Chg	Abs Chg	% of Total +/– Chg	Abs Chg	% of Total +/– Chg	Abs Chg	% of Total +/– Chg	Abs Chg	% of Total +/– Chg	Abs Chg	% of Total +/– Chg	Abs Chg	% of Total +/– Chg
All Other Cancers	–3.71	–10.8	–5.46	–28.3	–5.64	–24.0	–7.28	–37.6	–29.93	–26.5	–28.06	–27.0	–40.93	–30.5	–47.70	–39.0
Cardio and Metabolic Diseases																
Endocrine, Nutritional, & Metabolic	0.18	3.9	0.91	7.4	0.78	5.5	0.72	4.4	–3.86	–3.4	–10.14	–9.7	–15.25	–11.4	–16.33	–13.3
Hypertensive Heart Disease	0.10	2.1	0.51	4.1	0.57	4.0	0.59	3.6	0.27	2.3	2.14	7.5	0.74	2.7	2.19	6.0
Ischemic & Other Circulatory System	–3.24	–9.4	–2.74	–14.2	–1.59	–6.8	–2.08	–10.7	–51.57	–45.6	–52.02	–50.0	–58.10	–43.3	–47.75	–39.0
Substance Use and Mental Health																
Drug Poisoning	3.75	82.8	6.37	51.5	7.23	50.7	7.91	48.5	5.41	45.2	10.36	36.1	8.15	30.4	7.51	20.5
Alcohol-Induced*	–0.11	–0.3	0.36	2.9	0.12	0.8	2.28	14.0	–0.28	–0.2	0.64	2.2	2.31	8.6	5.48	15.0
Suicide	0.23	5.0	0.59	4.8	1.31	9.2	1.62	9.9	–0.06	–0.1	1.50	5.2	1.83	6.8	1.18	3.2
Mental & Behavioral Disorders	–0.53	–1.5	0.03	0.3	0.41	2.9	–0.51	–2.6	0.52	4.3	0.82	2.9	0.09	0.3	2.55	7.0
Other Body System Diseases																
Nervous System	0.15	3.4	0.54	4.4	–0.37	–1.6	–0.37	–1.9	2.83	23.6	4.58	16.0	3.56	13.3	5.54	15.1
Genitourinary System	–0.05	–0.1	0.12	1.0	0.53	3.7	0.02	0.1	–0.05	0.0	0.71	2.5	–1.03	–0.8	2.57	7.0

Respiratory System	–1.83	–5.3	–0.63	–3.3	–1.76	–7.5	–0.18	–0.9	–3.69	–3.3	0.72	2.5	1.10	4.1	0.09	0.2
Digestive System*	–0.56	–1.6	0.43	3.5	–0.76	–3.2	0.62	3.8	–4.21	–3.7	–2.45	–2.4	–6.15	–4.6	1.04	2.8
Other Causes of Death																
Homicide	–4.40	–12.9	–1.78	–9.2	–2.33	–9.9	–4.22	–21.8	–2.38	–2.1	–0.59	–0.6	–0.82	–0.6	–0.65	–0.5
Transport Injuries	–2.43	–7.1	–3.04	–15.8	–7.90	–33.7	–3.77	–19.5	–3.76	–3.3	–3.49	–3.4	–3.35	–2.5	–2.85	–2.3
Other External Causes of Death	–0.20	–0.6	0.18	1.4	–0.07	–0.3	0.06	0.4	0.35	2.9	1.04	3.6	–0.24	–0.2	0.84	2.3
All Other Causes of Death	–0.98	–2.9	1.81	14.6	2.47	17.3	1.43	8.8	–2.24	–2.0	0.30	1.0	–1.43	–1.1	1.22	3.3
Total Change: Increase (+)	4.53		12.38		14.26		16.32		11.95		28.70		26.79		36.57	
Total Change: Decrease (–)	–34.24		–19.30		–23.48		–19.37		–113.16		–104.03		–134.28		–122.43	

*Changes in the International Classification of Diseases (ICD)-10 code for diseases of the digestive system in 2006 affected the comparability of both digestive system deaths and alcohol-induced deaths before and after that year. More information is provided in Chapter 5.
NOTE: The top 3 causes of death for each age group in each period are highlighted in **bolded red** text.
Light orange highlights indicate an absolute increase in the overall mortality rate of ≥5 deaths/100,000 population.
Dark orange highlights indicate that a cause of death is responsible for ≥10 percent of the total increase in mortality.
Light green highlights indicate an absolute decrease in the overall mortality rate of ≥5 deaths/100,000 population.
Dark green highlights indicate that a cause of death is responsible for ≥10 percent of the total decrease in mortality.
The table shows the change in all-cause and cause-specific mortality rates among Hispanic working-age males (upper table) and females (lower table) by age group (25–44 and 45–64) and size of metropolitan area (large central metropolitan area, large fringe metropolitan area, small/medium metropolitan area, and nonmetropolitan area). These causes of death are exhaustive of all underlying cause-of-death codes and are based on the International Classification of Diseases (ICD)-9 (1990–1998) and ICD-10 (1999–2017) codes. Mortality rates within each age group are age-adjusted by single-year of age to match the age distribution of the U.S. population in 2000 in order to improve comparability over time. More information about the classification of causes of death can be found in Chapter 5.
SOURCE: Data from https://www.cdc.gov/nchs/nvss/nvss-restricted-data.htm.

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U.S. Mortality Data: Data Quality, Methodology, and Recommendations

Monitoring of trends and disparities in U.S. working-age mortality requires accurate vital statistics data on deaths, including causes of death, as well as accurate estimates of the size of the population at risk of death, to calculate consistent and accurate mortality rates. This chapter reviews the sources used to generate these estimates, including the quality and limitations of this information and how these data were used to produce the analyses presented in this report. The chapter covers issues related to the methodologies used to collect death certificate data and link them to survey data, the advantages and limitations of these types of mortality data, and the analytical methodology used by the committee in conducting its analyses. The chapter also includes the committee's recommendations for improving data quality to expand the capacity for future research on trends and disparities in U.S. working-age mortality.

THE U.S. NATIONAL VITAL STATISTICS SYSTEM (NVSS) AND THE CONSTRUCTION OF MORTALITY RATES

Death certificate records are an important component of the U.S. system of vital records. Within the United States, the responsibility for collecting death records is delegated to individual U.S. states and territories. They report this information to the federal government, which serves as the national repository of these records. This national repository of vital records, the NVSS, is maintained by the National Center for Health Statistics (NCHS) of the Centers for Disease Control and Prevention (CDC). NCHS death record files are considered 100 percent complete, although

there may be a small number of deaths for which registration is delayed, such as when an American dies outside of the United States or in the case of "missing persons" for whom the courts have not (yet) assigned certification of death. Vital statistics death certificate data include a limited amount of information about each decedent, including age at death, sex, race and ethnicity, educational attainment, place of residence, location of death, and cause(s) of death, as well as a few other items.

In addition to serving as the final repository for vital statistics records, NCHS assists the states in maintaining the best-quality vital records possible and provides resources and guidance for the structure and collection of vital records data, including recommendations and guidelines regarding the coding of such demographic information as race and ethnicity and educational attainment to improve the uniformity of coding across U.S. states. NCHS also cooperates with the World Health Organization (WHO), helping to improve comparability with international vital statistics data, particularly with respect to cause-of-death coding.

NCHS vital statistics data are released annually in data files that are structured to provide information about all deaths that occurred during a given year. Most studies of cause-specific mortality rely on the "underlying cause of death" coded in the files, which is defined as "the disease or injury which initiated the train of morbid events leading directly to death, or the circumstances of the accident or violence which produced the fatal injury" (World Health Organization [WHO], 2011, p. 31). In practice, the underlying cause is selected from the conditions entered by the medical certifier on the death certificate, thus making the training and qualifications of medical certifiers critical for accurate documentation of causes of death (Kochanek et al., 2019). When more than one cause or condition is entered, "the underlying cause is determined by the sequence of conditions on the certificate, provisions of ICD [International Classification of Diseases], and associated selection rules and modifications" (Kochanek et al., 2019, p. 62). Because most death certificates list more than one cause of death, more medical information is reported on death certificates than is directly reflected in the underlying cause of death. This additional information is available in the NCHS multiple cause-of-death data files.

Vital statistics data files do not include information about the size of the population that was at risk of death, which, as noted above, is necessary to calculate mortality rates. Therefore, this information must be drawn from a separate source—the U.S. Census. The U.S. Census Bureau conducts a census of the U.S. population every 10 years. During the intercensal period, the Census Bureau generates annual midyear population estimates for the country as a whole, as well as for demographic subgroups (e.g., age, sex, race and ethnicity), states, and local areas. Together, the demographic information included in the NCHS death certificate data files and the U.S.

Census Bureau's population estimates are used to calculate mortality rates by age, sex, and race and Hispanic ethnicity, both for the nation as a whole and for smaller geographic areas. Thus, the calculation of subgroup-specific mortality rates requires subgroup-specific population estimates that are based on comparable definitions of these subgroups.

Because denominator data for the calculation of U.S. mortality rates come from decennial U.S. Census data or the U.S. Census Bureau's population estimates for intercensal years, it is critically important that the decennial U.S. Census count be as accurate as possible. Similarly, the accuracy of the annual estimates of the size of the population by age, sex, and race and ethnicity, as well as geographic area, are vital for generating estimates of U.S. mortality patterns and trends.

As of this writing, the U.S. Census Bureau is weighing strategies for implementing a statistical practice known as "differential privacy" to protect the privacy of respondents to the decennial Census and other U.S. Census Bureau survey products by reducing the risk of disclosing information that, when combined with other publicly or privately available data sources, such as social media accounts, could allow respondents to be personally identified. In essence, the application of differential privacy would infuse statistical "noise" into the data, potentially affecting the accuracy of population counts for important subgroups of the population that are used to calculate mortality rates. The effects of introducing noise into population estimates are certain to be variable across subgroups and potentially could be large for small geographic areas and racial/ethnic groups with small populations. The magnitude of the impact of this change on subgroup population counts, and therefore its effect on the accuracy of mortality rates, will depend on how differential privacy is implemented by the U.S. Census Bureau. Evaluating the impact of these changes on mortality estimates will be of crucial importance for future mortality researchers.

Limitations and Quality of Mortality Data

Although they serve as a complete record of all U.S. deaths, death certificate data have important limitations that restrict the types of analysis and questions that researchers can address. The process by which death certificate data are collected can also result in issues of data quality and accuracy that affect the quality of mortality estimates and the comparability of these estimates over time. This section outlines the limitations of death certificate data and the steps that data providers have taken to address these issues so as to improve the utility of these data and expand the types of research questions they can be used to address. It then reviews known issues with the quality and accuracy of death record data that affect the quality of mortality estimates.

Limitations of Death Certificate Data and Use of Linked Mortality Data

The relatively limited information about the decedent on U.S. death certificates noted earlier (age, sex, race and ethnicity, educational attainment, place of residence, location of death, and cause(s) of death, as well as a few other items) is useful for examining disparities in mortality by these factors, but it limits the characteristics that can be examined. For example, educational attainment is only one dimension of socioeconomic status; other socioeconomic factors, such as income and wealth, may be important for understanding trends and disparities in working-age mortality, but this information is not available on death certificates. In addition to restricting the types of mortality disparities that can be examined, the relatively modest set of characteristics available on death certificates restricts researchers' ability to examine the factors that might explain mortality trends and disparities.

To examine a wider range of characteristics that might be related to mortality, researchers tend to rely on death certificates that are statistically linked to other data sources, most notably large social and health surveys. In some countries, death records are routinely linked to population registries (e.g., census records) to provide additional demographic and contextual information for death data. Such linked datasets have become especially important because they provide the research community with rich survey data on individuals, who are then followed statistically across multiple years to document who lives and who dies. But linking U.S. survey data to death certificates poses its own difficulties and limitations. To link death records to individual-level surveys, one must be able to identify individuals within both the death record data and the survey data, putting such linkages beyond the reach of most researchers.

To address this difficulty, several nationally representative governmental surveys, such as the National Health Interview Survey and the National Health and Nutrition Examination Survey, merge death record information from the annual National Death Index (NDI) with the survey data so that users can track mortality among those who appear in the surveys.[1] Because these surveys collect detailed demographic, income, behavioral, and health data and are representative of the U.S. population, this information can be used to calculate mortality risks by individual-level characteristics. However, one limitation of these linked datasets is that deaths occur quite infrequently during the follow-up period unless a survey includes a very large sample size, has a high proportion of elderly respondents, and/or has a long follow-up period. This limitation can lead to imprecise estimates of

[1]See https://www.cdc.gov/nchs/data-linkage/mortality-public.htm.

mortality and hamper the ability to compare mortality risks across groups; states and local areas; or social, behavioral, and health characteristics.

Some surveys with large sample sizes (e.g., the National Health Interview Survey) or long follow-up periods (e.g., the Panel Study of Income Dynamics and the Health and Retirement Survey that covers individuals ages 50 and over) may include enough deaths to enable the calculation of stable mortality rates. While these surveys greatly improve upon the quality and quantity of socioeconomic, behavioral, and health information available for studying relationships between individual-level factors and mortality risk, they also suffer from their own limitations, many of which may lead to underestimation of mortality rates. First, because these surveys are generally limited to the noninstitutionalized U.S. population, the individuals they include are healthier than the U.S. population as a whole, and underestimation of mortality rates may result (Keyes et al., 2018). Second, deaths are assessed by linking death records to the survey data, often using algorithms based on a set of respondent characteristics. When an individual is not linked, that individual is usually assumed to be alive; because of imperfect linkages between the death record data and the survey data, however, not all deaths may be recorded for the survey respondents. This is particularly the case, for example, when an individual in one of these surveys moves out of the United States and dies in a different country. Third, such datasets tend to be exceptionally useful for nationally based mortality estimates but typically do not include enough deaths to enable estimation of state- or local-level mortality patterns and trends. Finally, the small sample size and number of linked deaths among those at the oldest ages often lead to improbably low mortality rates for the oldest old within these surveys, among whom the proportion institutionalized grows with age.

Each decennial Census contains demographic information for every U.S. resident, but linkages between decennial Censuses and death certificates are not routinely conducted by the U.S. Census Bureau. However, a subset of records from the 1980 U.S. Census are linked to death certificate data as part of the National Longitudinal Mortality Study (NLMS). The NLMS is an important example of a linked dataset that was created to enable the study of mortality. It uses data from the Current Population Survey Annual Social and Economic Supplements and the 1980 U.S. Census linked to death certificate information for many years after individuals were included in the survey or U.S. Census. The dataset contains information from 3.8 million individuals and more than 550,000 death certificates.[2] The large sample size and number of deaths, combined with detailed information on socioeconomic status (SES), make the NLMS an important resource for studying the relationship between SES and mortality. In addition to the

[2]See https://www.census.gov/did/www/nlms.

above linked data sources, the linkage of the 2008 American Community Survey (ACS) to the NDI provides another large data source for studying mortality by individual-level characteristics.[3] However, another limitation of these linked datasets, other than the Panel Study of Income Dynamics and the Health and Retirement Study, is that survey information is collected at a single point in time; this point in time may precede death by a decade or more and therefore may not reflect the individual's SES at the time of death.

Despite these limitations, the above linked mortality datasets provide a wealth of information that would otherwise not be available using vital statistics data alone. The ability to link the NDI data to existing survey datasets provides an invaluable resource for researchers, public health officials, and policy makers. Thus, the committee concluded that no reduction or changes in the content of the information collected for these datasets is warranted, and that the research community would be well served if the datasets were made as widely and easily available to researchers as possible. Currently, many survey datasets that are linked to death records do not make these linked data publicly accessible or provide only limited mortality information in public-use data files; more detailed information on cause of death and geography are available only through restricted-use data files. Making available the linking algorithm and weighting scheme used to produce linkages between the NDI and individual-level survey data would also help researchers determine any potential biases in the linkages and enable them to better assess how such biases might influence their estimates.

Quality and Accuracy of Death Certificate Data

Several well-known data collection and coding issues affect the quality and accuracy of the data on U.S. death certificates, related to the coding of cause-of-death information by medical examiners and the assignment of other demographic information. While the accuracy and quality of these records is constantly evolving, there have also been ongoing efforts to progressively improve their accuracy and utility for public health purposes.

A number of factors can influence the quality and accuracy of the cause-of-death information on death certificates, such as the era (period) in which the death occurred, changes in the ICD, place of death, available local resources, training of the medical certifiers, and the complexity of the chain of medical diagnoses that led to death. The era in which the death record was generated matters for several reasons, but in particular because advances in diagnostic and forensic technology can change how causes of death are identified and coded.

The United States transitioned from the ICD-9 to the ICD-10 coding system for cause-of-death data in 1999. This was the first major change in

[3]See https://www.census.gov/mdac.

the cause-of-death coding system in the United States since the implementation of the ICD-9 system in 1979. One of the features of the ICD-10 system is a standardized schedule for introducing updates to the codes to ensure that the system is flexible and remains consistent with current medical practice and knowledge. The advent of new versions or modifications of the current ICD and related taxonomic rubrics, such as environmental causes of death, can spur training in new coding systems and improve the accuracy of data coding as knowledge evolves, but can also lead to inconsistencies with coding from previous eras that impede comparisons over time. WHO will introduce ICD-11 in 2022, and the National Committee on Vital and Health Statistics (2019) advised the U.S. Department of Health and Human Services to take a proactive approach to preparing for its release.[4]

The quality of death record reporting is also affected by the training and resources available to local medical certifiers. The availability of modern local resources and quality control programs in a region or jurisdiction affects the provision of the services of medical examiners and coroners with appropriate forensic, clinical pathology, and toxicology expertise. Consistent and accurate coding of causes of death can be difficult given the frequent complexity and uncertainty of clinical diagnoses, and a lack of training can lead to inconsistent coding of more complex causes of death. In general, diseases that are clinically easier to diagnose and/or have longer clinical courses are the most likely causes of death to be identified accurately. In contrast, diseases with short and more diverse clinical presentations and courses are more likely to be misclassified (Mieno et al., 2016). NCHS has developed an automated system to standardize and improve the coding of underlying cause of death. This system, the Automated Classification of Medical Entities (ACME), reads in the multiple cause-of-death data reported on the death certificate and applies decision rules developed by WHO to assign the underlying cause of death.[5] Misclassification may also depend on the specific level of the condition at hand. For example, there is evidence that liver disease in general is underrepresented in death records (Durante et al., 2008), whereas primary liver cancer may be overrepresented (Polednak, 2013) because of misclassification of metastatic disease. In addition, chronic conditions are often missing from death certificates or assigned the status of a contributing cause rather than the underlying cause of death, even though they are relatively easy to diagnose (Gao et al., 2018). The training of certifying professionals is an important factor in the accuracy and completeness of these cause-of-death reports. Because the quality and training of medical certifiers are of paramount importance for

[4]See https://ncvhs.hhs.gov/wp-content/uploads/2019/12/Recommendation-Letter-Preparing-for-Adoption-of-ICD-11-as-a-Mandated-US-Health-Data-Standard-final.pdf.

[5]See https://www.cdc.gov/nchs/nvss/mmds/about_mmds.htm.

high-quality cause-of-death data, states and local health agencies need to ensure that certifiers are well trained in cause-of-death recording, including the coding of both underlying and multiple causes of death.

Differences in resources and training across jurisdictions can lead to local, state, and regional variations in standard practices for cause-of-death coding (Cheng et al., 2012; Cheng, Lu, and Kawachi, 2012). Although NCHS produces decision tables in the ACME to improve the consistency of cause-of-death reporting and identification of the underlying cause of death in death certificate data, these decision tables rely on not only the conditions reported as cause of death but also the causal sequencing of these reports. Reporting errors that can lead to discrepancies in the identification of the underlying cause of death remain common (Lu, Anderson, and Kawachi, 2010) and show considerable variation by cause of death (Falci et al., 2018), particularly when multiple comorbidities are present and may contribute to death (Lu, Anderson, and Kawachi, 2010). The level of reporting errors varies substantially across states, which affects the comparability of cause-specific mortality rates (Cheng, Lu, and Kawachi, 2012).

Data quality issues are a particular concern in examining deaths from acute poisoning and drug overdose. The term "drug overdose" is often used synonymously with acute poisoning, but it has at least three different coding definitions (Slavova, Bunn, and Talbert, 2014) and is often difficult to define toxicologically and pharmacologically. Moreover, many decedents are found to have multiple substances in their blood or other issues that create uncertainty in identifying the specific cause of death (Ruhm, 2018a, p. 1339). In some cases, for example, drugs associated with the treatment of drug addiction, such as methadone or buprenorphine, are present alongside other substances, further complicating the assignment of a specific cause of death and raising the question of whether these treatment medications should be assigned a dedicated cause-of-death code (Darke et al., 2019).

U.S. states vary substantially with respect to laws regarding the types of deaths that require a formal autopsy, as well as the resources provided to coroners and medical examiners for conducting the autopsies and biochemical determinations necessary to record cause of death accurately. Taken together, these variations lead in turn to local, regional, and national variation in the quality and accuracy of cause-of-death recording, making trend analyses and geographic comparisons of causes of death challenging. In light of the state-by-state variation and increasing geographic inequalities in U.S. mortality, studies to evaluate state-level variation in coding practices are warranted (Dwyer-Lindgren et al., 2016).

Given uncertainties in the coding of underlying causes of death, useful and detailed information may be contained in the contributing causes of death, depending on the goal of the analysis (Remund, Camarda, and Riffe, 2018). Yet most analyses, including those presented in this report and the

standard reports on mortality published by NCHS, document mortality patterns and trends according to underlying cause of death while ignoring associated causes. This is the case largely because of the complexity involved in coding and use of the full range of causes on death certificates. However, methods that account for multiple causes of death can reduce the impact of coding errors in the underlying cause of death that arise from misreporting of the causal ordering of conditions on the death certificate. In light of developments in data analysis (e.g., machine learning, weighting of multiple causes of death) and computing power, analysts could consider ways in which the full range of causes available on the death certificate can be used to document the complexity of causes from which U.S. adults are dying (Dwyer-Lindgren et al., 2016; Eberstein, Nam, and Heyman, 2008; Piffaretti et al., 2016).

Accurate coding of demographic information included on death certificates depends on a different set of factors. On most surveys, including the U.S. Census, demographic information is either self-reported or reported by a knowledgeable proxy. However, decedents obviously are not able to self-report demographic information. Therefore, this information is often provided by surviving relatives or friends or can sometimes be drawn from other sources, such as medical or official records. In some cases, however, the information may be left to the medical examiner or coroner to report based on physical examination or ancillary sources, and these reports may not be consistent with how this information would have been reported by the decedent, particularly when the reports refer to such characteristics as educational attainment, race, and ethnicity. This issue is compounded when death counts based on death certificate data are divided by population estimates from the U.S. Census Bureau to calculate mortality rates. The most common U.S. Census-based sources for population estimates provide data on such characteristics as race, ethnicity, and educational attainment that are collected, reported, and coded differently from similar data appearing on death records. These inconsistencies can compound the effects of recording errors in the death certificate data, leading to biased estimates of mortality and mortality disparities.

The documentation of race and ethnicity on U.S. death certificates, while improving, continues to be far less than 100 percent accurate. This is especially the case for American Indian and Alaska Native (AI/AN) populations, among whom official mortality rates have been deemed far too low (Arias, Heron, and Hakes, 2016). The committee found that death certificates continue to misclassify AI/AN individuals to such an extent that official mortality estimates for these populations are not valid. This in turn limits the ability of public health officials to track and highlight mortality within these populations, despite indications from other data sources that they experience much higher mortality relative to most other racial/ethnic

groups, with the possible exception of Blacks (Espey et al., 2014; Hummer and Gutin, 2018). Together, researchers from NCHS, a state vital statistics agency, and the Tribal Epidemiology Center have developed a set of promising ideas for improving the estimation of AI/AN mortality (Anderson, Copeland, and Hayes, 2014); below, the committee recommends further work in this area to make such improvements.

At the same time, according to the 2016 update to an earlier NCHS evaluation of the quality of race and ethnicity data on death certificates, the classification of race and ethnicity for Asians and Pacific Islanders and Hispanics had improved relative to earlier reports, and the accuracy of reporting for these populations was almost as good as that for Whites and Blacks, which previously was of higher quality. Although improvements over time in the quality of death certificate reports for Asians limited the committee's ability to interpret their mortality trends over the period covered by this report, reporting of Asian race on death certificates has improved substantially as well and is now of sufficient quality that researchers, policy makers, and public health officials can have confidence in mortality estimates for this population. Given that underascertainment of mortality among Asians is now estimated to be about 3 percent, similar to that for Hispanics, it is time for Asians to be included in regular NCHS reports on life expectancy. Doing so is particularly important given the rapidly increasing size of the U.S. Asian population.

An additional complication in the coding of race and ethnicity on death certificates is the change in racial/ethnic reporting categories over time. Prior to changes recommended by the Office of Management and Budget in 2003, death certificates recorded only a single race and included less-detailed race and ethnicity categories. Subsequent changes in reporting were adopted inconsistently across states, potentially leading to difficulties in creating comparable race and ethnicity groups across time. Moreover, these measurement differences can reduce the comparability of race and ethnicity reports on death records and in population data, in turn reducing the accuracy of mortality rates. NCHS has developed bridged-race estimates that use an empirically derived algorithm to reassign multiracial individuals to one of the single-race categories in use prior to 2003; however, NCHS also used these bridged-race estimates to evaluate the quality of race reporting on death certificates. To the committee's knowledge, no research to date has evaluated the concordance between multiracial race reports on death certificates and self-reports of multiracial identity in survey data. Racial/ethnic groups that make up a smaller percentage of the population and have a higher percentage of members who identify as multiracial, such as AI/ANs and Native Hawaiians and Pacific Islanders, are more likely to be misidentified on death certificates. Finally, despite the expanded race and ethnicity options introduced in 2003, patterns and trends in working-age

mortality of "other" racial/ethnic groups, such as persons who (wish to) identify as Middle Eastern, are unknown because neither death certificate numerator data nor U.S. Census-based denominator data include options for reporting such identities.

Similar reporting issues affect the quality of educational attainment reports on death certificates. Vital statistics and census data are the only large-enough data sources that allow for detailed examination of educational disparities in mortality by specific place of residence. However, the documentation of educational attainment on U.S. death certificates is often inaccurate (Rostron, Boies, and Arias, 2010) because this information is reported by proxy sources. NCHS reported that when educational attainment on death records was compared with corresponding information from the Current Population Survey, substantial differences were found between the two sources (Rostron, Boies, and Arias, 2010). For example, when educational attainment data on death certificates are inaccurate, high school completion tends to be overreported, leading to overstatement of deaths among those with a high school degree and understatement of deaths among those with less than a high school degree (Sorlie and Johnson, 1996).

Often the denominator information for the size of educational attainment groups is drawn from population data, such as the decennial U.S. Census, or federal data, such as population projections or the ACS. These sources are more likely than death records to contain self-reported data or data reported by a knowledgeable respondent. In calculating mortality rates, data on educational attainment from death records are combined with population estimates from the decennial U.S. Census to calculate mortality rates. When census population data are combined with death counts that underreport the number of deaths among those with less education because of misclassification, lower mortality rates among this group result. Similarly, when deaths for those with higher levels of education are overcounted and the resulting numbers are combined with accurate population size estimates, the resulting mortality rates overstate mortality in this population. This inaccuracy affects not only the estimates of mortality within each educational attainment group but also estimates of the disparities among those groups.

These accuracy issues raise important concerns about the validity of using death records to study educational disparities in mortality. States and local health agencies may need to initiate additional training and guidance for those who provide this information on death certificates. Given not only the documented inaccuracy of educational attainment data on death certificates but also possible state-by-state and local-area variation in the recording of this information, researchers would be well advised to use studies based on self-reported survey data linked to death certificate

mortality data to assess the accuracy of data on educational attainment and their geographic variation on death certificates.

Place of birth is also recorded on U.S. death certificates, but data on place of birth are not made available to researchers in public-use vital statistics mortality files. This is an important concern given the growth of the foreign-born population in recent decades, along with its heterogeneity. Moreover, variation in mortality by nativity has become critical for understanding U.S. mortality trends and differentials (Dupre, Gu, and Vaupel, 2012; Lariscy, Hummer, and Hayward, 2015; Lauderdale and Kestenbaum, 2002; Turra and Elo, 2008). In addition, the size of the foreign-born population in the U.S. Census denominator data is subject to underestimation, especially if large segments of the foreign-born population are missed because of their documentation status. Given the current lack of information on nativity (i.e., foreign-born vs. U.S.-born) in publicly available vital statistics data, researchers need to tackle both racial/ethnic and nativity disparities in U.S. working-age mortality using survey-based data linked to the NDI or Social Security Administration data on older Americans, or make a special request for restricted death record files.

ESTIMATION OF MORTALITY RATES IN THIS REPORT

The data used to produce the mortality rates presented in this report were drawn from death certificate data provided by the CDC in the NVSS restricted death certificate files for 1990–2017 (National Center for Health Statistics [NCHS], 2018). These files include information about the decedent's date and underlying cause of death; place of residence; and a small number of demographic characteristics, including sex, age, and race and ethnicity. In 2003, during the period covered by the committee's analyses, NVSS adopted new recommendations for the coding of racial/ethnic data on death certificates that allowed the reporting of more than one race; however, the timing of the adoption of this change varied across states. For this reason, the population counts by age, sex, and race and ethnicity that the committee combined with these mortality data to calculate mortality rates were based on the U.S. Census bridged-race estimates of the U.S. resident population on July 1 of each year, produced by the U.S. Census Bureau (U.S. Department of Health and Human Services [HHS], 2018). These bridged-race estimates reclassify multiracial individuals into one of the single-race categories that were in use prior to 2003. State-level mortality rates were drawn from the CDC WONDER database (Centers for Disease Control and Prevention [CDC], 2020b).

The analyses presented herein are based on trends for multiyear age groups. Previous researchers have noted that because mortality rates increase with age in adulthood, differences in the age composition of two

populations can affect the comparability of their mortality rates when broad age categories are used (Gelman and Auerbach, 2016). To ensure comparability over time and across subpopulations, rates were age-adjusted by single year of age and standardized to reflect the age distribution of the U.S. population in 2000, unless otherwise noted in the text. Throughout, mortality rates are presented as the number of deaths per 100,000 population. Deaths were pooled across 3-year periods from 1990–2017, with the exception of the first period (1990–1993), which includes 4 years. The data were pooled across years to smooth fluctuations in mortality trends that sometimes occur for smaller populations with relatively low death counts. Many of the analyses presented in this report rely heavily on mortality rates from four periods corresponding roughly to the beginning and end of the 1990s, the 2000s, and the 2010s: 1990–1993, 2000–2002, 2009–2011, and 2015–2017.

This report's summary of research on mortality trends and differentials by educational attainment draws on previously published studies that use both vital statistics data and survey-based data linked with the NDI. Given the issues of the quality of educational attainment data raised above, especially in vital statistics mortality data, the committee used only the highest-quality studies in this area in which such data quality issues are best taken into account. Provided below are the committee's recommendations for studying and improving data quality in this important area of mortality study.

In preliminary analysis, the committee also examined mortality patterns and trends for Asians and Pacific Islanders and for AI/ANs. Because of concerns about data quality—specifically, that the errors in racial classification on death certificates for these populations changed over time as discussed above (Arias, Heron, and Hakes, 2016)—those trends are not presented in this report. To the extent that such information is available, summaries of the existing literature on mortality trends for these groups are provided to ensure that their experiences are represented. Within this literature, however, researchers may have used different classifications for cause of death from those used for the original analyses presented in this chapter, and therefore these analyses may not be directly comparable.

Metropolitan status was based on a modified version of the geographic identifiers developed by the Economic Research Service (ERS) of the U.S. Department of Agriculture and NCHS.[6] The committee classified U.S. counties into four types of metropolitan areas: large central metropolitan areas (counties in metropolitan statistical areas [MSAs] of more than 1 million population, including counties that contain all or part of the area's inner cities), large fringe metropolitan areas (surrounding counties of the

[6]See https://www.cdc.gov/nchs/data_access/urban_rural.htm.

large central metros, including suburbs), medium/small metropolitan areas (counties in MSAs of 50,000–999,999 population), and nonmetropolitan areas (counties outside of metropolitan areas). To maintain consistency over time, the counties' metropolitan categories were assigned based on the 2013 ERS classification scheme (sensitivity analyses showed only minor differences using earlier classification schemes).

For the analyses in this report, cause of death was assigned to one of 20 broad, nonoverlapping categories based on the underlying cause of death identified on the decedent's death certificate (Table 5-1). The committee identified these categories after reviewing trends across more detailed categorizations. After that detailed review, causes with low death counts and similar temporal trends were collapsed into larger categories. Causes of death were determined based on ICD-9 for 1990–1998 and ICD-10 for 1999–2017. This change in coding systems could have led to discontinuities in cause-specific mortality trends at the points at which the new ICD-10 coding system was adopted even within categories whose definitions had not changed. The committee was cognizant of this possibility and examined cause-specific mortality trends that covered the full 1990–2017 period in order to assess evidence of a discontinuity in the cause-specific trends. These results are presented in Appendix A.

Because of a small change in ICD codes used over the study period, readers should exercise caution when interpreting changes in two of the committee's broad cause-of-death categories: alcohol-induced diseases and diseases of the digestive system. ICD-10 code K85 (acute pancreatitis) was discontinued in 2006 and replaced with several K subcodes. One of those subcodes (K85.2, alcohol-induced acute pancreatitis) is included in the committee's "alcohol-induced" category for 2006–2017 but could not be broken out prior to 2006. From 2006 to 2017 (the years during which K85.2 was used), there were only 3,279 deaths in that category among all working-age (ages 25–64) adults (both sexes, all racial/ethnic groups). Any bias this coding change may have introduced into the committee's temporal comparison would be observed in a jump in alcohol-related deaths and a decline in diseases of the digestive system between 2005 and 2006.

Chapter 4 presents trends over time in the percentages of deaths due to mental and behavioral disorders that were drug- or alcohol-related. The assignment of these deaths to these two categories was based on the ICD-9 and ICD-10 codes listed in Table 5-2.

RECOMMENDATIONS

Based on the issues discussed above, the committee offers the following recommendations for improving data quality and availability to support analyses of patterns and trends in U.S. working-age mortality.

TABLE 5-1 Assignment to 20 Cause-of-Death Categories

Cause-of-Death Category	ICD-9 Codes	ICD-10 Codes
HIV/AIDS	042–044	B20–B24
Non-HIV/AIDS Infectious and Parasitic Diseases	001–041, 045–139	A00–A99, B00–B19, B25–B99
Liver Cancer	155	C22
Lung Cancer	162	C33, C34
All Other Cancers	140–239, exc. 155, 162	C00–D49, exc. C22, C33, C34
Endocrine, Nutritional, and Metabolic Diseases	240–279	E00–E88, exc. E24.4
Hypertensive Disease	401–405	I10–I15
Ischemic Heart Disease and Other Diseases of the Circulatory System	390–459, exc. 401–405, 425.5	I00–I99, exc. I10–I15, I142.6
Mental and Behavioral Disorders	290–319	F01–F99
Diseases of the Nervous System	320–359, exc. 357.5	G00–G98, exc. G31.2, G62.1, G72.1
Diseases of the Respiratory System	460–519	J00–J98
Diseases of the Digestive System	520–579, exc. 535.3, 571.0–571.3	K00–K92, exc. K29.2, K85.2, K86.0
Diseases of the Genitourinary System	580–629	N00–N98
Homicide	E960, E961, E962.1, E962.2, E962.9, E963–E969	X86–X99, Y00–Y09, Y87.1
Alcohol-Induced	357.5, 425.5, 535.3, 571.0–571.3 790.3, E860	E24.4, G31.2, G62.1, G72.1, I42.6, K70, R78.0, X45, X65, Y15
Drug Poisoning	E850–E858, E950.0–E950.5, E962.0, E980.0–E980.5	X40–X44, X60–X64, X85, Y10–Y14
Suicide	E950.6, E950.7, E950.8, E950.9, E951–E959	X66–X84, Y87.0
Transport Accidents	E800–E848, E929.0, E929.1	V01–V99, Y85
Other External Causes of Death	E861–E899, E900–E928, E929.2–E929.9, E930–E949, E970–E979, E980.6–E980.9, E981–E999	W00–W99, X00–X39, X46–X59, Y16–Y36, Y40–Y84, Y86, Y87.2, Y88, Y89
All Other Causes	280–289, 360–379, 380–389, 630–676, 680–709, 710–739,740–759, 760–779, 780–799 (exc. 790.3)	D50–D89, H00–H57, H60–H93, L00–L98, M00–M99, O00–O99, P00–P96, Q00–Q99, R00–R99 (exc R78.0), U00–U99

SOURCES: Data from https://wonder.cdc.gov/controller/datarequest/D16 (ICD-9) and https://wonder.cdc.gov/controller/datarequest/D76 (ICD-10).

TABLE 5-2 ICD-9 and ICD-10 Codes for Drug- and Alcohol-Related Deaths due to Mental and Behavioral Disorders

Cause-of-Death Category	ICD-9 Codes	ICD-10 Codes
Mental and Behavioral Disorders	290–319	F01–F99
• Due to alcohol	305.0, 291, 303	F10
• Due to drugs	292, 304, 305.2–305.9	F11–F16, F19

SOURCES: Data from https://wonder.cdc.gov/controller/datarequest/D16 (ICD-9) and https://wonder.cdc.gov/controller/datarequest/D76 (ICD-10).

RECOMMENDATION 5-1: The National Center for Health Statistics (NCHS), state vital statistics offices, and local-area health agencies should work together to develop a plan and set of activities for improving the accuracy of reporting on U.S. death certificates of educational attainment, American Indian and Alaska Native identity, and multiple causes of death. NCHS should also continue to conduct or facilitate studies on the accuracy of reporting on U.S. death certificates of educational attainment (particularly as such reports may vary across states and local areas) and American Indian and Alaska Native identity (particularly as such reports may vary across states, tribal affiliations, and local areas).

RECOMMENDATION 5-2: The National Center for Health Statistics and the National Institutes of Health should undertake and/or fund studies to evaluate state- and local-level variation in cause-of-death coding practices, explore how such variation may contribute to observed mortality trends, and make recommendations for reducing such variation.

RECOMMENDATION 5-3: The National Center for Health Statistics should include Asians in its regular reports on life expectancy estimates and trends in the United States and make an item on place of birth available to researchers in the public-use files, even if such information is at first categorical (e.g., foreign-born vs. U.S.-born) rather than granular.

RECOMMENDATION 5-4: To enable robust research on rural–urban trends in health and mortality, the National Institutes of Health and other research agencies and funders should support the oversampling of rural populations on national health and social surveys, including both existing (e.g., Health and Retirement Study, Behavioral Risk Factor Surveillance System, National Longitudinal Study of Adolescent to Adult Health [Add Health], National Survey on Drug Use and Health, National Health Interview Survey, National Health and Nutrition Examination Survey) and new surveys.

PART II

6

A Framework for Developing Explanations of Working-Age Mortality Trends

Part I of this report describes the trends in mortality in the United States over the past few decades. This chapter provides a framework developed by the committee to guide its development of potential explanations regarding the drivers of these trends. The term "drivers" refers to causes in the broadest sense, encompassing both upstream or distal and more proximal causes—factors at different stages in the chain of causation. Also considered are how factors at various levels may interact. For example, low socioeconomic status may place individuals at higher risk of drug use, but use may occur only in the context of greater access to prescription opioids. In this case, the more proximate low socioeconomic status can be thought of as comprising "vulnerability" factors and the more distal contextual drug availability as comprising "precipitating" factors.

This chapter begins by describing the conceptual framework used by the committee to frame its broad discussion of the drivers of mortality trends. It then reviews the considerations used by the committee in developing potential explanations for the observed trends.

A CONCEPTUAL FRAMEWORK FOR U.S. WORKING-AGE MORTALITY

Figure 6-1 depicts the committee's framework for the general set of factors that may have contributed to high and rising mortality among U.S working-age adults. The factors involved are shown to operate at three different levels: macro or societal, meso or community (including family), and individual (Braveman, Egerter, and Williams, 2011; Hertzman and Boyce,

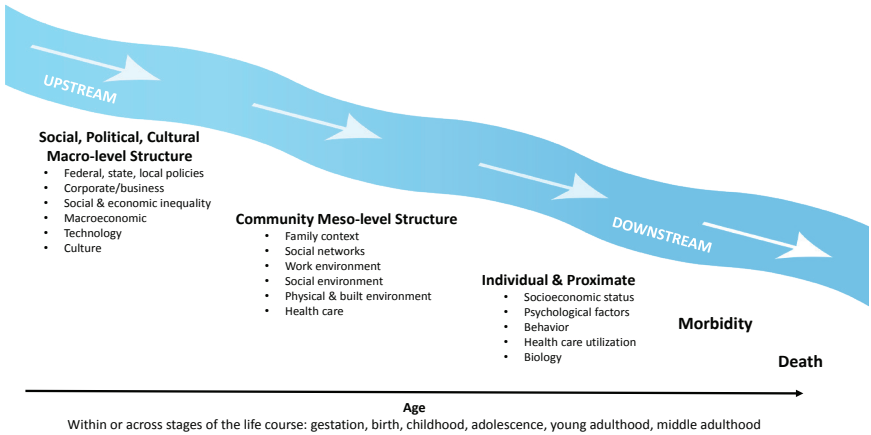


FIGURE 6-1 Conceptual framework: A life-course multilevel model of factors involved in high and rising mortality among working-age adults.

2010). As noted above, these factors can also be categorized as distal (factors that affect health through long causal chains and many intermediary factors) or more proximal (factors that impact health more directly). Age represents the time dimension across which the multilevel factors operate, which can span the entire life course, from conception to death, or the set of ages within specific life stages—gestation, birth, childhood, adolescence, young and middle adulthood—that shape working-age mortality. In addition, although not explicitly shown in the figure, factors can interact or act synergistically such that the presence of two or more factors is necessary for morbidity and mortality outcomes to occur. In such cases, one factor, such as low socioeconomic status, may make individuals vulnerable to the presence of another, such as increased drug availability.

The most distal factors are referred to as "upstream" drivers, which generally fall within the category of "social, political, and cultural macro-level structure." Intermediate factors, those that are influenced by the macrostructural factors and represent the community-level contexts in which individuals live their daily lives, fall within the category of "meso-level structure" in the figure. Upstream and intermediate factors are considered structural by virtue of their higher order or aggregate level of measurement and analysis (e.g., social and economic inequality, institutional or governmental policies, neighborhood, social networks). The more proximal factors (through which the macro- and meso-level structural factors exert health effects on individuals) include individual-level psychosocial and behavioral characteristics, as well as the biological consequences of

behaviors or environmental exposures. These "downstream" consequences of the more distal macro- or meso-level factors are important to fully understand the causality involved in working-age morbidity and mortality risk, but are not necessarily the main drivers that have changed in society to bring about the increase in working-age mortality during the 1990–2017 study period documented in Part I of this report.

While the committee uses the theoretical language of causality in describing this multilevel conceptual model, the framework is not necessarily deterministic. Rather, it organizes the "causes" of working-age mortality in a multilevel order of their proximal distance from the event of death. Death occurs at an individual level, and the proximate cause (e.g., drug overdose, cardiac failure) is identified at the individual level, but this proximate cause is often the result of higher-order upstream macrostructural and environmental influences (e.g., decisions by the pharmaceutical industry, the obesogenic environment, health care access). Focusing greater attention on the upstream drivers of mortality enables policy makers to intervene on these factors, alter their influences on health outcomes, and reduce mortality risks and disparities.

In addition, not all causal processes flow downstream from the macrostructural to the individual level in one direction or in this order. Causal factors can be introduced at any point in the process and create feedback loops, and the causal direction may flow upstream temporarily. For example, the changing composition of families in a particular community or state (e.g., a higher percentage of low-income or single-parent families) may shift social policies in that location to be more supportive of those families (e.g., Medicaid expansion, higher minimum wage). In addition, exogenous factors can intrude at any point. For example, the effect of natural disasters (e.g., hurricanes, floods, pandemics) can adversely impact meso-level environmental conditions and, in turn, individual risks of mortality. A disaster may then have lagged effects that alter upstream social inequality and policies (e.g., insurance disparities, government assistance) in the disaster locations, and changes in these factors can flow downstream to influence individual-level risks of morbidity and mortality.

While structural features of the environment are correlated with individual mortality risks, these structural features are often the result of individual characteristics and actions.

For example, high-income individuals can afford to live in neighborhoods with green space for physical activity, healthy food choices, health care resources, and high-quality schools for children. Similarly, individuals may choose to live in certain states based on family networks, occupational opportunities, or quality-of-life issues. Adults of lower socioeconomic status tend to have less choice in where they live and work, and these characteristics, which are correlated with state-level policies (e.g., Medicaid

expansion, taxes) and state-level characteristics (e.g., proportion of the population that is poor, pollution), may also determine health outcomes.

Macro-Level Upstream Factors in Working-Age Mortality

Increasing awareness of the importance of upstream factors in health and mortality grew out of a research emphasis on the social determinants of health, conceptualized as the conditions under which people are born, grow, live, work, and age (World Health Organization [WHO], 2008). Social determinants of health represent nonmedical factors influencing health and the risk of death, including upstream social structural influences on health and health systems; government policies; and the social, economic, political, cultural, and environmental factors that determine health, often through downstream health-related knowledge, attitudes, beliefs, and behaviors (Braveman, Egerter, and Williams, 2011). Upstream structural factors affect lifestyle choices and behaviors and are shaped by public policies, which in turn are modifiable and subject to change. Thus, research evidence on this set of factors provides important opportunities for reducing mortality risks and disparities by intervening to change these factors that harm health through downstream mechanisms.

Federal, State, and Local Policies

The most upstream drivers include features of the social, political, and economic structure. These factors (and their health consequences) can, to a certain extent, be affected by public policies, including those at the federal, state, and local levels (Institute of Medicine and National Research Council [IOM and NRC], 2013). For example, federal policies determine spending on safety net programs, environmental regulations for clean air and water, housing, gun safety, taxes, and health care. Similar policies are implemented by state governments (e.g., welfare eligibility and benefits, Earned Income Tax Credit, cigarette taxes, Medicaid expansion for health care via the Affordable Care Act, transportation services, family planning). Local political and administrative units (e.g., school districts, counties, cities) are governed by federal and state policies but also have autonomy to establish their own policies that influence community resources and mortality risks through spending on education; health care services; mental health and substance use programs; local health-related infrastructure, such as parks and sidewalks; and more. Together, these federal, state, and local policies are thought to drive subgroup and geographic differences in social and economic inequality, environmental conditions affecting health, and health care access (Hummer and Hamilton, 2019; Montez, Hayward, and Wolf, 2017).

Corporate/Business Organizations

A second set of macro-level factors involves corporate/business organizations, such as pharmaceutical and drug manufacturing companies, the insurance business, the tobacco industry, and the food and beverage industries. Business interests may align with public health priorities (Adams, 2019) but may also run counter to family and individual health interests. Examples include beverage companies' investments in and marketing of sugary and alcoholic drinks, food companies' investments in and marketing of unhealthy foods, and pharmaceutical companies' production and marketing of pain medications. Corporate goals tend to be focused primarily on increasing profit margins and satisfying shareholders, motivating heavy promotion of their products. Some companies—such as those that cater to a growing market for health-oriented products and services or invest in strategies to promote the health and safety of their employees and the communities in which they live—find a business case that aligns with public health. Other companies that profit from unhealthy or unsafe products (e.g., tobacco companies, opioid manufacturers) have strong incentives to minimize or even intentionally hide adverse health effects associated with their products.

Social and Economic Inequality

Social and economic inequality limit the degree of economic opportunity and social mobility that exists in national, state, and local contexts. Much attention has been given to the high levels and continued growth of income and wealth inequality in the United States compared with other industrialized nations (Piketty and Saez, 2014). As a smaller and smaller proportion of the population (e.g., "the 1 percent") owns the vast majority of America's wealth, economic opportunities become cemented into the social hierarchy, constraining intergenerational mobility for most. Social inequality further defines differential access to opportunities and resources that promote longevity and prevent premature mortality according to such attributes as gender, race and ethnicity, nativity, LGBTQ status, and educational attainment, and those disparities are exacerbated as this structurally based inequality widens (Hayward, Hummer, and Sasson, 2015; Phelan and Link, 2015; Phelan, Link, and Tehranifar, 2010).

Macroeconomic Trends

Macroeconomic trends are another set of upstream structural influences on health involving broad aggregate shifts in the economy that can have population-wide effects, positive or negative. For example, the decline

of manufacturing jobs and growth of the service sector beginning in the 1970s altered the demand for a specific set of worker training and skills and caused the collapse of major industries that provided employment security. The economic and social impact of this trend, which unfolded over decades, had potentially long-term negative effects on the health, health behaviors, and longevity of workers, especially those with less education, and on their communities' ability to provide health-promoting services and resources. The economic downturn during the 2008 Great Recession affected population subgroups differentially according to age, gender, race and ethnicity, and socioeconomic status, with short- and long-term implications for health.

Technology Developments

Another set of macro-level factors comprises technology developments, which can impact mortality trends in multiple ways. Most obvious are innovations in drug development and drug potency, advanced screening for disease risks, and innovative treatments for disease. Technology, furthermore, affects individuals' work, lifestyle, and behavior. For example, machines have replaced humans in certain jobs, computers and smartphones have replaced some forms of human interaction and increased sedentary work environments, and access to the Internet has now reached most of the population. Many of these technological developments result in important cultural shifts that have implications for work, health behaviors, and health. The growth of the Internet and social media, in particular, has resulted in profound cultural changes affecting the way people communicate, learn, share information, and seek help, accelerating cultural change on a global scale.

Cultural Factors

Changing cultural factors can also be important for health and mortality risk. They can include, for example, changes in the importance of religious institutions and societal norms regarding family formation (e.g., marriage, cohabitation, divorce, nonmarital childbearing). For example, changes in the societal-wide importance of religion (i.e., secularization) could be associated with changing health and mortality patterns, given the health protection that religious belonging affords (Ellison and Hummer, 2010). Some cultural factors, including racism, sexism, xenophobia, and homophobia, can be especially influential for health and health disparities. For example, discrimination on the basis of social characteristics harms health and longevity through differential access to socioeconomic resources and through chronic stress processes, both of which are especially salient

in the meso-level structures that individuals experience on a daily basis (Williams, Lawrence, and Davis, 2019).

Meso-Level Factors

Meso-level factors encompass the local contexts in which people live their daily lives—in families, with friends and other social networks, at work, in the neighborhood, and through experiences with the health care system.

Family Context

The family is one of the most important meso-level contexts relevant to health, impacting behaviors, stressors, and health care access. Family characteristics are in turn affected by the upstream factors discussed above, including policies, culture, inequality, and macroeconomic trends. For example, societal increases in educational and labor market opportunities for women led to dramatic increases in and cultural acceptance of women, even mothers of young children, working outside the home. Through work, an increasing number of women gained economic independence, eroding the traditional exchange system on which marriage was based—that the man was the breadwinner and the woman a homemaker. At the same time, macroeconomic shifts related to the decline of manufacturing jobs reduced marriage rates as men's economic position faltered, especially among those with less education. Structural changes, furthermore, led to cultural shifts in the acceptance of divorce, cohabitation, single-mother families, and nonmartial childbearing, profoundly altering the family contexts in which children are born and raised in America. These synergistic structural and cultural shifts may affect health and mortality risk in multiple ways. For example, women's greater independence, higher status, and higher incomes could lead to improved health and lower mortality risk, especially for those with high educational attainment and higher-paying, professional jobs. On the other hand, low-income women who are single parents may be especially vulnerable to poor health and mortality risk, particularly in a period like the Great Recession or the COVID-19 pandemic, given the greater stresses of being in a low-socioeconomic structural position in the context of a weakened social safety net (e.g., limited unemployment benefits and dwindling food, housing, and health care benefits) (Montez et al., 2015).

Social Networks

Social networks are another important meso-level factor undergoing change that may be associated with increases in working-age mortality.

There is a large literature on the importance of social connections for health and longevity. Social ties, embeddedness in social networks, and engagement in social life help lower mortality risks, whereas social isolation and a lack of social connections are harmful to health (e.g., Berkman et al., 2000; Cacioppo and Hawkley, 2003; Cohen and Janicki-Deverts, 2009; Holt-Lundstad et al., 2015; House, Landis, and Umberson, 1988; Yang et al., 2016). Social networks may include immediate- and extended-family relations, friends, neighbors, workmates, and coworkers, as well as engagement in local institutions, including religious, political, sports, or civic organizations. Research indicates that social networks benefit health when social ties provide social support, knowledge, and coping skills to deal with daily stresses, but can harm health when those ties cause strain or added stress. Thus, both the quantity and quality of social networks matter in relation to premature mortality risks. Social network connections may also provide valued social capital resources, such as information about training or job opportunities, community health care access, and other local resources. To the extent that engagement in social networks reinforces common goals and interests and promotes positive and supportive social interaction, feelings of social cohesion and social integration are protective from mortality risks.

Work Environment

The work environment is another important meso-level structure that is influenced by upstream economic, social, and cultural macrostructural factors and is associated with working-age mortality (Johnson, Sorlie, and Backlund, 1999). Some work environments pose risks to workers because of physical conditions that include inadequate ventilation, heights, demanding physical labor, access to harmful drugs, or hazardous chemical exposures. Other work environments pose risks to workers through lack of autonomy or control, thus increasing stress, boredom, and frustration. Employers also influence workers' access to health care services and employer-based health insurance.

Neighborhood Environment

The neighborhoods and communities in which individuals live and the resources they provide can promote health and reduce risks of premature mortality. When the neighborhood context includes high-quality schools, transportation services, access to medical care, and employment resources, mortality risks may be reduced. Similarly, neighborhoods characterized by social cohesion, social organization (e.g., environmental volunteer groups, parent–teacher organizations, local sporting or cultural clubs), low poverty rates, housing stability, and low unemployment are likely to benefit health

and reduce mortality. The same is true for health-promoting physical and built environments. The physical characteristics of neighborhoods and communities that influence health and mortality risks include air and water quality; proximity to facilities that produce or store hazardous substances; exposures to lead paint, chemicals, mold, dust, or household pest infestation; access to nutritious foods; and safe and convenient places to exercise, such as sidewalks, running trails, and parks and recreation centers.

Health Care

Availability of and access to health care within the community in which people live or work is vital to health promotion; disease prevention; and the treatment of medical conditions, mental health, and injuries—each of which can prevent premature death. Thus, access to care (the number and proximity of providers and health care facilities, and insurance coverage) and the quality of care are key environmental factors that may mediate upstream influences on downstream health behaviors and outcomes. Unlike other industrialized countries, the United States lacks universal access to health care; has fewer physicians, hospitals, and acute care beds per capita relative to comparable countries; has a larger percentage of the population that must defer or delay care because of costs; and relies on a fragmented care delivery system characterized by large disparities in quality of care (Schneider et al., 2017). Such heterogeneity in health care access and quality was on full display during the COVID-19 pandemic. The closing of medical facilities within rural areas has been a long-standing issue, not only for the lack of access to care but also because these facilities are often one of the largest employers within the region (Lindrooth et al., 2018). State variation in Medicaid expansion through the Affordable Care Act is another example of the geographic variation in health care access, especially among the low-income population across the United States.

Individual-Level Factors

Many characteristics of individuals are relevant to health and working-age mortality. Some (e.g., education and income) are the individual-level manifestations of the social and economic structures discussed above; others reflect the downstream consequences of those more distal factors. These more proximal individual-level factors include socioeconomic status, psychological characteristics, behavior, health care utilization, and biology (one's biological and genetic makeup).

The socioeconomic gradient in health and mortality has been widely documented across birth cohorts, time, and place, with consistent evidence of widening educational disparities, in particular, since the 1970s

(Case and Deaton, 2017; Hummer and Hernandez, 2013; Preston and Elo, 1995; Wolfe, Evans, and Seeman, 2012). Like neighborhood or work contexts, individual-level socioeconomic factors affect health and mortality risk through a number of more proximal individual-level processes related to stress, behaviors, and health care access and use. Extensive research has also focused on the role of behavior in mortality trends in general and in the recent rise in mortality among working-age adults in particular. Health behaviors, including tobacco use, alcohol and drug use, violence, exercise, and diet, have a direct relation to deaths from multiple causes, including drug poisoning, alcohol use, suicide, cancer, and cardiometabolic diseases. Psychological factors, such as self-esteem, confidence, conscientiousness, mastery, sense of control, depression, and anxiety, have also been linked to a number of health outcomes, including those responsible for high and increasing working-age mortality. Insured individuals are more likely than those without health insurance to have regular access to and use of health care services.

Biological factors are most proximate to the cause of death, for they define the pathophysiologic processes that cause disease and produce fatal complications. Changes in biological processes reveal how what happens outside the body (e.g., such macrostructural forces as inequality, work environments, and economic recession; exposure to environmental threats, such as pollution or lead; social relationship stresses, such as social isolation or interpersonal violence; health behaviors, such as substance use or consumption of sugary beverages and fatty foods) affects what happens inside the body (e.g., tissue damage; dysregulation of stress response systems; and pathological changes to metabolic, endocrine, neurological, cardiovascular, immune, and other body systems), leading to acute fatalities (e.g., drug overdose, suicide) or chronic illnesses (e.g., diabetes, hypertension) that can cause death (Harris and McDade, 2018).

The ascribed demographic attributes of sex, race, and ethnicity are not explicitly shown in Figure 6-1 as individual-level characteristics. As demonstrated by the committee's analysis in Part I of this report, these attributes define fundamental historical and contemporary disparities in mortality. Explanations for these disparities therefore involve the multilevel processes elaborated by the committee's conceptual framework that are unique to each subgroup defined by sex, race, and ethnicity and their combinations.

The Role of Life-Course Stage

The macro-, meso-, and individual-level factors discussed above can operate at various stages across the life course, beginning in gestation (or even the preconception period for mothers) and continuing at birth and during childhood, adolescence, and early adulthood with the potential to

shape health and mortality outcomes among working-age adults at every stage (Ben-Shlomo and Kuh, 2002; Hertzman and Boyce, 2010; Kuh and Ben-Shlomo, 2004). Key stages in individuals' lives can have particular relevance for working-age mortality. For example, research increasingly documents the importance of early life, including gestation and birth, for the emergence of chronic diseases in later life (Almond and Currie, 2011; Barker, 2004; Gluckman et al., 2008). This body of research points to exogenous shocks experienced early in life (e.g., exposure to an influenza pandemic or nutritional deficits) as having latent, long-lasting effects on health and development that heighten the risk of premature mortality. Other factors operating in childhood, such as early adverse life experiences or low socioeconomic status, can affect mental and physical health later in adulthood (Felitti et al., 1998). Behaviors established in childhood and adolescence (e.g.,smoking, diet, or physical activity) can track into adulthood, with long-term consequences for health (Harris, 2010; Harris et al., 2006). Long-term exposures operating over the life course (e.g., a lifetime history of being overweight or obese) also can affect chronic disease risk later in life.

In addition, causal pathways involving upstream and downstream factors can operate across a range of ages *within* specific life stages to affect morbidity and mortality in that life stage. For example, the upstream decision by pharmaceutical companies to flood the market with opioids was a macrostructural effect that operated through specific community contexts (areas of manufacturing and mining job losses) and affected individuals with certain socioeconomic and psychological characteristics (e.g., those experiencing long-term unemployment or physical or emotional pain), leading to drug overdose deaths. All of these multilevel processes can operate within the life stage of young or middle adulthood to affect an individual's risk of dying from drug poisoning.

CONSIDERATIONS IN DEVELOPING EXPLANATIONS FOR TRENDS IN WORKING-AGE MORTALITY

In developing possible explanations regarding the causes of changes in working-age mortality over time, the committee employed a set of considerations to help prioritize those explanations most aligned with the observed trends and also, importantly, to rule out explanations that were clearly incompatible with those trends. These considerations also highlight the types of additional data and future research that may be useful in supporting or refuting competing explanations. Of course, rigorous testing of the explanations developed on the basis of these considerations may require data that are very different from those routinely available in nationally

representative mortality studies but are necessary to draw robust causal inferences.

Single Versus Multiple Explanations

An important consideration is whether the observed mortality trends over the 1990–2017 study period (which, as described in Part I of this report, can be multifaceted) reflect a common causal process that manifested in different causes of death and across different population groups, or whether multiple causal processes happened to be operating simultaneously or in tandem to generate the observed trends. For example, one could hypothesize that decreases in stable, well-paid employment opportunities led to increased stress and related mental health consequences, with implications for drug use and overdose deaths, suicide, and the heavy alcohol consumption associated with alcohol-related deaths. Thus, a common causal process could underlie trends observed with all three of these causes of death, which, as discussed in Chapter 3, have been increasing in recent decades and for a wide range of population groups and geographic areas.

Alternatively, the drivers of increases in drug poisoning deaths, suicides, and alcohol-related deaths could be distinct, although perhaps somewhat correlated in terms of the timeframe during which they unfolded. For example, increases in drug poisoning deaths may be attributable primarily to drug supply issues, corresponding with Food and Drug Administration approval of Purdue Pharma's OxyContin® and other opioids in the mid-1990s. Increases in suicide mortality may reflect trends in depression related to economic or social trends that occurred during roughly the same period. And increasing heavy alcohol use that causes increases in alcohol-related deaths could be responsive to changing social norms regarding access to and accessibility of alcohol over the past few decades.

Although parsimony is desirable in developing potential explanations, it was important to keep in mind as the data were examined that in cases of multiple mortality trends in different subgroups, a range of correlated factors could be operating simultaneously. The data at hand may not allow full identification of the underlying processes, and it is also possible that a common process coexists with multiple additional causal processes.

Interactions or Synergies and Dynamic Relations Among Factors

In developing explanations for trends in working-age mortality, it was also important to keep in mind that various types of factors may interact or act synergistically. For example, economic disadvantage (and the psychological distress it induces) may place individuals at greater risk of drug use and addiction. But the psychological distress they experience may culminate in

drug use and addiction only in the context of easy access to drugs, amplified by the marketing efforts of pharmaceutical companies. Thus, the economic disadvantage resulting from macrostructural factors can be viewed as a vulnerability, whereas the easy access to drugs can be viewed as a trigger or precipitating factor. Both the vulnerability (economic disadvantage) and the precipitating factor (easy drug access) may be necessary for drug poisoning deaths to increase. Note that the concept of vulnerability and precipitating factors is distinct from the concept of distal and proximal factors. The distinction between distal and proximal factors emphasizes their distance from the event of death and their location in chains of causation: A leading to B leading to C. In contrast, the distinction between vulnerability and precipitating factors refers to how different factors interact: A and B together are necessary for C to occur (but A does not necessarily lead to B).

Many schematic conceptual models of population health focus primarily on long causal chains and linear relations, but fewer emphasize interactions or synergies and dynamic relations among factors. Dynamic relations encompass feedbacks and dependencies. For example, drug use may be facilitated by drug availability, and drug availability may be reinforced by increased demand, creating a reinforcing cycle (feedback). In addition, the behaviors of individuals may affect the behaviors of others in their social networks through social norms or contagion-like processes (dependencies). These types of relations characterize the complex systems that may give rise to observed mortality trends, so understanding their key features may be important to developing comprehensive explanations for those trends.

This chapter has introduced the concept of vulnerability, which may be relevant in explanations for rising working-age mortality and disparities therein. In the context of this report, vulnerability refers to physical, economic, social, or environmental factors that place an individual at heightened risk of morbidity and mortality. Such factors can operate at multiple levels. For example, individual-level characteristics (e.g., socioeconomic disadvantage, membership in a racial minority) can make one more vulnerable to working-age mortality by limiting access to social, economic, and health care resources. A particular environment (e.g., residential segregation, single-mother family) may create vulnerability through limited access to high-quality schools or green space for physical activity. Macro-level policies may create vulnerabilities for some population groups or geographic areas through regulations on the prescribing of pain medication or placement of toxic waste sites. Vulnerabilities also can make one more susceptible to working-age morbidity and mortality in particular contexts. For example, those with comorbidities (e.g., cancer, arthritis, injuries) are at greater risk of experiencing pain, which makes them more susceptible to easy access to and widespread availability of opioids, and in turn to death from drug overdose. Similarly, those with chronic diseases are

more susceptible to COVID-19 infection and death. Vulnerability is also created when macro- and meso-level contexts (e.g., regulations regarding clean air and water, obesogenic factors such as the proximity and quantity of fast food restaurants and alcohol outlets) or individual behaviors (e.g., substance use, diet) present threats to health and longevity.

Socioeconomic Inequality at All Levels

Socioeconomic inequality is defined as a social and/or economic state of unequal access to opportunity, resources, or means in a process leading to health and longevity (Braveman and Tarimo, 2002; Link and Phelan 1995; Phelan, Link, and Tehranifar, 2010). It is important to consider how socioeconomic inequality operates at the macro, meso, and individual levels in its influence on changes in mortality trends and disparities. While the committee's framework specifically identifies social and economic inequality as a key macrostructural factor and socioeconomic status as a fundamental individual-level characteristic, socioeconomic inequality is an implicit characteristic of the midstream meso-level contexts in the framework. For example, socioeconomic inequality in neighborhoods can be characterized according to neighborhood levels of social and economic disadvantage (e.g., average income, proportion in poverty, composition of adults with a college degree, proportion of families receiving public assistance). The socioeconomic status of families is indicated by household income, poverty status, wealth, and household structure. Social networks can also be characterized by socioeconomic characteristics of network ties, such as the number or proportion of social connections with college degrees, or average earnings of network members. Thus, in seeking to explain the increase and disparities in working-age mortality, it is important to consider how socioeconomic inequality is experienced at all levels of society, is correlated across levels, and flows through and reinforces inequality at separate levels.

Differences Across Social Groups

Any explanation of the drivers of the mortality trends described in this report must also consider the reasons why those drivers manifested differently by age, gender, race and ethnicity, socioeconomic status, and geographic region during the study period. As discussed in Chapters 2 and 3, U.S. trends in working-age mortality during this period did not occur uniformly across the population but sometimes evolved in different ways across groups and in different places. For example, one cannot invoke the lack of job opportunities as an explanation for increases in working-age mortality among less-educated non-Hispanic Whites (Whites) living in rural areas without explaining why similar mortality trends were

not observed among other racial groups that have experienced unfavorable economic circumstances, such as low-educated urban non-Hispanic Blacks (Blacks). Explanations that are not sensitive to such heterogeneity in trends across social and geographic groups run the risk of being too general and thus much less useful than explanations that take such heterogeneity into account.

Consideration of vulnerability and precipitating factors may also help explain differences in mortality trends across social and geographic groups. For example, structural factors related to social inequality and disadvantage may consistently make low-educated, low-income, and racial/ethnic minority groups vulnerable to adverse health impacts through stress/mental disorders or other mechanisms. But the way in which the adverse health effects of structural disadvantage manifest (via what individual and biological processes, when, and at what ages) may differ depending on other contextual (and precipitating) factors. For example, structural disadvantages linked to racism and economic downturns were associated with increasing mortality and decreasing life expectancy among urban Blacks in the 1980s, due largely to increasing mortality from HIV/AIDS and homicide (Geronimus et al., 1996; Kochanek, Mauer, and Rosenberg, 1994). On the other hand, structural disadvantages related to inequality and economic factors may have placed low-educated Whites at particular risk of opioid- and alcohol-related deaths in the mid-1990s and early 2000s. The precipitating factors could also vary over time and across population groups depending on the social and historical context, although it could be argued that the fundamental cause (racial/socioeconomic disadvantage and lack of opportunities) is the same (Link and Phelan, 1995). For example, racial differences in the prescribing practices of physicians may have been an important precipitating factor for the especially rapid escalation of opioid-related deaths among Whites in the 1990s and 2000s (Pletcher et al., 2008), even if the economic vulnerability faced by Blacks and other minority groups was similar.

Levels Versus Trends

Although it may be tempting to assume that the causes of differences in mortality levels are similar to the causes of differences in mortality trends over time, this may not be the case. The mortality level at a certain point in time may be influenced by a multiplicity of multilevel factors interacting over the life course, but these factors may or may not be the ones driving changes in mortality over a particular calendar period. For example, the factors responsible for differences in mortality levels between Blacks and Whites may or may not be similar to those explaining changes in the Black–White mortality gap over time. Although the focus of this report is largely on changes over time, it is important to view such trends in the context of

what are often very large and persistent differences in health and mortality by such factors as race and ethnicity and socioeconomic status.

Time Lags

A critical issue in explaining the drivers of trends in working-age mortality pertains to the time lags involved. For example, trends in alcohol-related deaths (due to chronic liver disease, for example) may reflect increasing exposures to alcohol occurring across long periods. In such a case, explanations for the trend must consider changes in alcohol availability and/or use that potentially date back years, if not decades. In contrast, drug poisoning deaths may be more responsive to short-term changes in drug availability, such as those occurring with the approval and aggressive marketing of Purdue Pharma's OxyContin and other opioids in the mid- to late 1990s (Van Zee, 2009). Moreover, rates of suicide may be responsive in the medium or short term to changes in social isolation and depression, while rates of cardiometabolic diseases involving obesity and diabetes may be more likely to reflect trends in long-term exposure to risk factors and their causes beginning much earlier in time and continuing over the life course. Sorting out the time lags involved in changing rates of cause-specific mortality poses serious challenges for researchers trying to explain such trends.

Attention to Period and Cohort Effects

Related to the issue of time lags, a final important consideration in identifying the drivers of mortality trends is attention to period and cohort effects. Researchers studying mortality trends often distinguish between period-based and cohort-based explanations of the trends. Period-based influences affect mortality change simultaneously for all age groups in a population in the same time period. For example, the obesity epidemic is a period-based phenomenon that began in the early 1980s and continues to the present day and has affected all ages in the U.S. population. The prevalence of obesity increased across the 1980s, 1990s, 2000s, and 2010s, and this upward trend likely reflects period-based changes in exposure to obesogenic environments and behaviors. In another example, the rapid increase in the supply of opioids in the mid-1990s may have led to increased mortality rates for all ages of a specific population, even if some age, social, or geographic groups were more vulnerable than others. On the other hand, cohort-based influences affect mortality change by influencing mortality risk for distinct cohort groups and in so doing, tend to affect change in certain age groups more than others. For example, persons born within a particular timeframe (e.g., 5 years or 10 years) constitute a "birth cohort,"

while persons who graduated high school and/or college and/or entered the job market at the same time might constitute an "education cohort" or "employment cohort," respectively. An exposure that is specific to one or a few birth cohorts, such as exposure to a famine or cumulative socioeconomic disadvantage that originates at birth, may thus lead to cohort explanations for trends in working-age mortality. Differentiating period-based from cohort-based variation in U.S. mortality rates is therefore an important consideration for the committee in assessing the evidence for possible explanations for the recent trends in working-age mortality.

There are well-established descriptive approaches for evaluating whether and how period- and cohort-based factors are likely responsible for working-age mortality trends. Plots of age-specific mortality rates can indicate whether cohort-based variation in the rates likely exists beyond age- and period-based variation. A common approach plots all-cause or cause-specific mortality rates on the vertical axis by age (horizontal axis) for a set of either 5- or 10-year birth cohorts. To the extent that the age pattern of mortality differs across birth cohorts, one would conclude that there are different cohort patterns of mortality that change with age. However, evidence for cohort-based *differences* in an outcome does not constitute evidence for cohort-based *mechanisms* driving that outcome. Identifying cohort-based variation in mortality trends—and, by extension, cohort effects on mortality trends—is difficult. Cohort differences in the age pattern of mortality may be due to several mechanisms or a combination of mechanisms, and sorting out the simultaneous effects of age, period, and cohort influences is challenging because of the exact linear dependency among these influences, known as the identification problem (O'Brien, 2014; Yang and Land, 2013). A preferred descriptive approach for detecting cohort-based variation in mortality trends entails plotting age-specific mortality rates across time periods to observe the degree to which the period-based trends in mortality rates are parallel (Kupper et al., 1985). If the period-based trends in the age-specific death rates appear parallel to each other, the trends most likely reflect period-based sources of change. Conversely, if the period-based trends in the age-specific death rates appear to be nonparallel, the age-based variation in the period-based trends may reflect cohort-based sources of change.

The annex at the end of this chapter provides a more detailed description of these tools for exploring and interpreting period- and cohort-based variation in working-age mortality, including examples from simulated data. Some of the research reviewed by the committee in Part II of this report includes findings on cohort and period differences in patterns of working-age mortality and identifies specific period- and/or cohort-based explanations responsible for recent trends in U.S. working-age mortality. Within this report, the committee is therefore careful to indicate the method

used to analyze cohort- and period-based influences on working-age mortality trends in the research reviewed.

SUMMARY

Levels and trends in mortality are the manifestations of a large set of interacting processes, often occurring over long periods of time. Nevertheless, it is sometimes possible to identify the likely key drivers of the observed patterns, which is especially important for identifying the most effective policies and programs to promote health and reduce disparities in both health and mortality. The multilevel broad life-course framework proposed in this chapter and the set of considerations presented can be useful in this regard. In subsequent chapters, these ideas are applied in discussing potential explanations for recent mortality trends and in identifying research gaps in understanding the prominent mortality trends highlighted in Part I of this report.

ANNEX 6-1

Period- and Cohort-Based Examination of Trends in U.S. Working-Age Mortality

This annex provides a detailed description of how differences in age-specific mortality rates can be examined to evaluate whether and how period- and cohort-based factors are likely responsible for the trends in working-age mortality documented in this report. As noted in this chapter, evidence for cohort-based *differences* in an outcome is not evidence for cohort-based *mechanisms* driving that outcome. Thus, cohort differences in age-specific mortality rates do not necessarily mean that the differences arose from cohort effects on mortality. To illustrate this point, the committee generated hypothetical data (shown in Annex Table 6-1) on mortality trends from three data-generating processes (DGPs) involving age, period, and cohort effects. To simplify matters, the same age effects are assumed for all three datasets (i.e., M_x is elevated during midlife relative to early life, but lower than in old age). Any differences in the mortality rates thus entirely reflect the different period and/or cohort processes in the three scenarios:

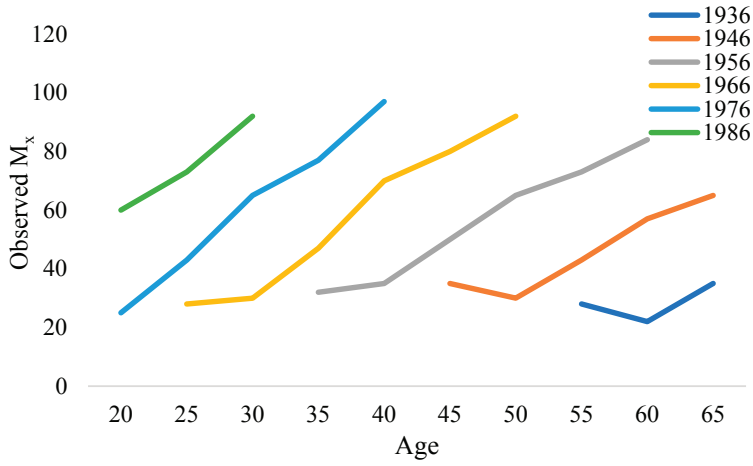
DGP_1: Large Age Variation + Large Period Variation + No Cohort Variation
DGP_2: Large Age Variation + No Period Variation + Large Cohort Variation
DGP_3: Large Age Variation + Large Period Variation + Large Cohort Variation

ANNEX TABLE 6-1 Simulated Data Used in Annex Figures 6-1 to 6-6

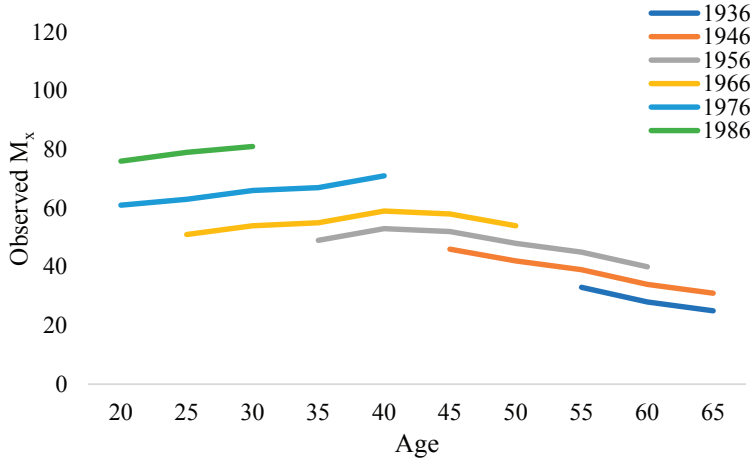
	DGP_1		DGP_2		DGP_3	
	b	σ	b	σ	b	σ
Age 1	-10	1	-10	1	-10	1
Age 2	-10	1	-10	1	-10	1
Age 3	-8	1	-8	1	-8	1
Age 4	-2	1	-2	1	-2	1
Age 5	0	0.5	0	0.5	0	0.5
Age 6	0	0.5	0	0.5	0	0.5
Age 7	-6	1	-6	1	-6	1
Age 8	-12	1	-12	1	-12	1
Age 9	-18	1.5	-18	1.5	-18	1.5
Age 10	-20	1.5	-20	1.5	-20	1.5
Period 1	-35	1.5	1	1	-35	1.5
Period 2	-35	1	0	1	-35	1
Period 3	-20	1	1	1	-20	1
Period 4	0	0.5	0	1	0	0.5
Period 5	10	1	1	1	10	1
Period 6	27	1	0	1	27	1
Cohort 1	0	3	-25	3	-25	3
Cohort 2	0	2	-25	2	-25	2
Cohort 3	0	2	-23	2	-23	2
Cohort 4	0	2	-20	2	-20	2
Cohort 5	0	1	-13	1	-13	1
Cohort 6	0	1	-6	1	-6	1
Cohort 7	0	1	-2	1	-2	1
Cohort 8	0	1	0	1	0	1
Cohort 9	0	1	2	1	2	1
Cohort 10	0	1	0	1	0	1
Cohort 11	0	1	-2	1	-2	1
Cohort 12	0	1	-6	1	-6	1
Cohort 13	0	1	-8	1	-8	1
Cohort 14	0	2	-8	2	-8	2
Cohort 15	0	2	-6	2	-6	2
Intercept	45	5	38	5	73	
Random Component	0	5	0	5	0	
N		100,000		100,000		100,000
Simulations		100		100		100

The cohort-based differences observed in M_x for each of the three DGPs are plotted in Annex Figures 6-1 through 6-3.

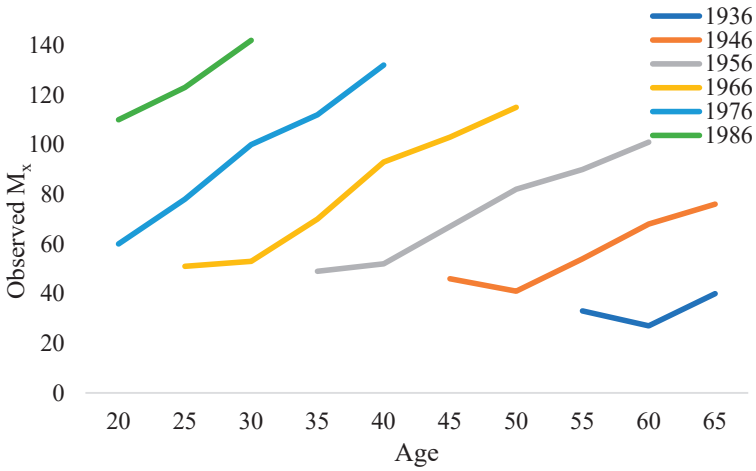
In DGP_1 (Annex Figure 6-1), the age-specific mortality rates differ considerably by birth cohort, and the age patterns increase across cohorts. For example, the M_{40} rates in DGP_1 are observed to be 35 deaths per 100,000 population for birth cohort 1956, 70 for birth cohort 1966, and 97 for birth cohort 1976. These large cohort differences in M_x reflect entirely the cohorts' differential exposures to period effects in DGP_1. That is, because no cohort-based variation exists in DGP_1, the observed differences in M_x



ANNEX FIGURE 6-1 DGP_1 M_x by birth cohort.



ANNEX FIGURE 6-2 DGP_2 M_x by birth cohort.

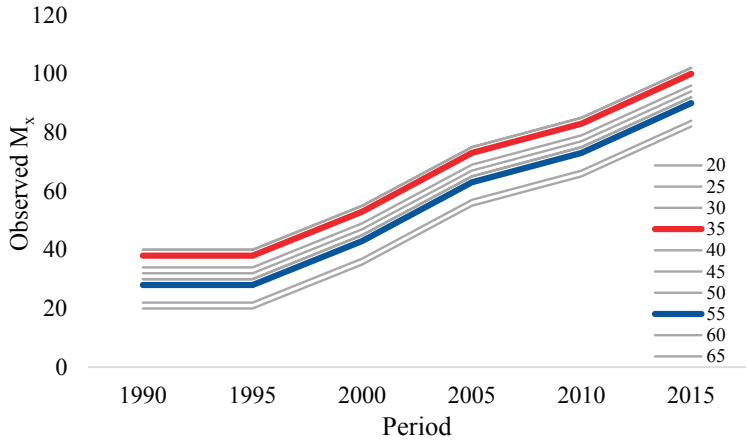


ANNEX FIGURE 6-3 DGP_3 M_x by birth cohort.

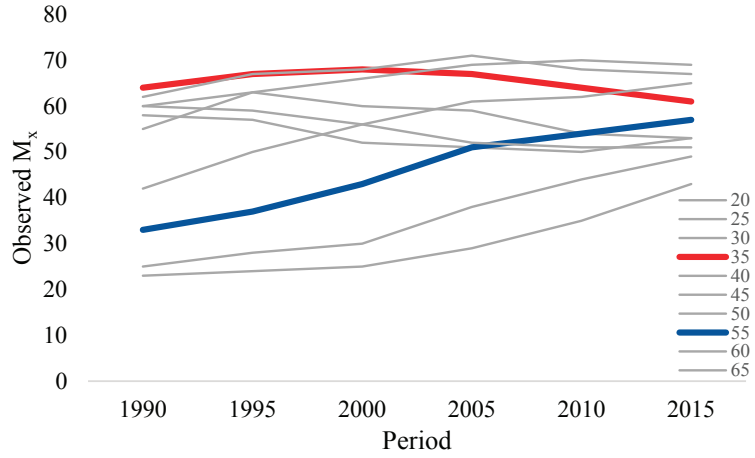
across the cohorts are due entirely to cohorts' differing exposure to the period effects. In contrast, in DGP_2 (Annex Figure 6-2), M_x differs by birth cohort, but the age patterns do not increase across cohorts. Rather, the level of M_x simply shifts up or down by cohort. As a result, the cohort-based differences are not as great as the differences observed in DGP_1, and the cohorts' age-based trends do not differ from each other. Finally, in DPG_3 (Annex Figure 6-3), cohort patterns in M_x are similar to those in DGP_1 (i.e., cohort-based differences are large, and the age patterns appear to increase across cohorts), but the differences in DGP_3 are larger across cohorts because of both the cohort and period effects on mortality trends.

Thus, this descriptive approach of contrasting different cohorts' age-specific death rates does not help detect cohort-based variation in mortality trends. As highlighted by Kupper and colleagues (1985), evidence for cohort effects in descriptive mortality plots is best detected via "nonparallelism" in the age-specific mortality curves. To find evidence for such "nonparallelism," researchers plot age-specific mortality rates (M_x) across time *periods*. If the period-based trends in M_x appear to parallel each other, the trends most likely reflect period-based sources of change. Conversely, if the period-based trends in the age-specific death rates appear to be nonparallel, the age-based variation in the period-based trends may reflect cohort-based sources of change.

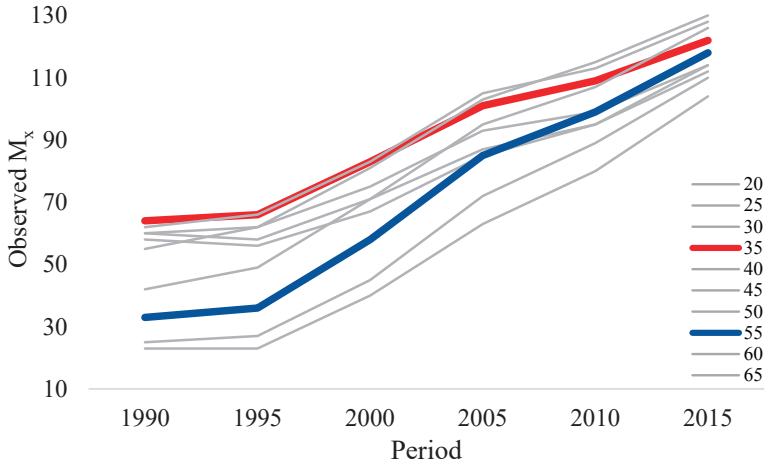
Annex Figures 6-4 to 6-6 plot the age-specific death rates generated from DGP_1, DGP_2, and DGP_3 across time periods to depict the extent to which the mortality curves exhibit "nonparallelism." M_{35} (red) and M_{55}



ANNEX FIGURE 6-4 DGP_1 M_x by period.



ANNEX FIGURE 6-5 DGP_2 M_x by period.



ANNEX FIGURE 6-6 DGP_3 M_x by period.

(blue) are highlighted to contrast clearly the variation in M_x trends across the three DGPs.

The M_x levels in Annex Figure 6-4 (DGP_1) differ by age, and the values increase over time periods. However, the perfectly parallel trends strongly indicate that the age groups share the same mortality trends. Because the patterns exhibit no "nonparallelism," the plots do not suggest cohort-based mortality effects in DGP_1. This conclusion is consistent with what is known, in that all mortality variation in DGP_1 was generated to be age- and period-based. In contrast, the cohort-based variation in DGP_2 is strongly evident in the M_x curves in Annex Figure 6-5. The age-based differences in M_x vary considerably across time periods. For example, mortality among the 35–39 age group was the highest mortality in 1990, changed little between 1990 and 2005, and then declined slightly between 2005 and 2015. In contrast, mortality among the 55–59 age group was low in 1990 and increased steadily across the entire time span of 1990–2015. Because of the disparate mortality trends between these age groups, their M_x levels were comparable in 2015. And because the age groups experienced strikingly different trends across time periods, the "nonparallelism" in the plots suggests strong cohort-based variation in their mortality rates. This conclusion is consistent with what is known, in that all mortality variation in DGP_2 was generated to be age- and cohort-based.

Finally, in DGP_3 (Annex Figure 6-6), there is evidence for strong period-based increases in M_x that are similar to the period-based increases in DGP_1 but also exhibit some key differences from DGP_1 trends. The separate age groups all experience similar mortality increases, from 15–60 deaths

per 100,000 population in 1990 to about 90–125 deaths in 2015. However, some differences in the age groups' increases are also evident. For example, age group 35–39 starts with relatively high mortality rates in 1990 (~60 deaths per 100,000 population) and increases across the time periods to about 110 in 2015. Age group 55–59 starts with relatively low mortality rates in 1990 (~30 deaths per 100,000 population) and increases rapidly across the time periods to about 110 in 2015. The age groups' differential rates of mortality increases suggest cohort-based variation in addition to the strong period-based trends observed in M_x. This conclusion is consistent with what is known, in that mortality variation in DGP_3 was generated to be age-, period-, and cohort-based.

Annex Table 6-1 shows the simulated data used in Annex Figures 6-1 through 6-6 above. Provided next are three examples of how the use of period and cohort analyses can shed light on the drivers of recent trends in U.S. working-age mortality. Age-specific death rates are plotted across time periods to explore period- and cohort-based variation in those trends. Cause-specific mortality rates are examined (1) for drug use among U.S. non-Hispanic Black (Black) and non-Hispanic White (White) men to illustrate Black–White differences in drug-related mortality trends; (2) for cardiometabolic diseases among U.S. White women and men to illustrate similar cohort-based trends among men and women; and (3) for alcohol use among U.S. Black and White men and women to illustrate race- and gender-based differences in period and cohort trends in U.S. mortality. Each example demonstrates how considering and possibly identifying the period- and cohort-based sources of U.S. mortality trends can help inform possible explanations for those trends.

With respect to U.S. men's drug-related mortality rates, evidence overwhelmingly indicates that trends in the White male population likely reflect period-based effects. In contrast, evidence strongly indicates that both period- and cohort-based effects shaped trends in the U.S. Black male population. With respect to mortality rates for cardiometabolic diseases, the patterns of trends among the working-age U.S. White population suggest strong cohort-based variation that is especially pronounced among White women. And with respect to mortality rates for alcohol use, the evidence suggests that the trends among U.S. White and Black men and women were strongly affected by the Great Recession but also that cohort-based effects influenced deaths among both White men and women.

These varying patterns in the mortality trends among working-age Americans are worth considering when evaluating possible explanations for the recent mortality trends in the United States. Any explanation for rising mortality among U.S. adults would need to attend to the Black–White differences in period-based trends in U.S. men's drug-related death rates, the similarities in the cohort-based mortality trends for cardiometabolic

diseases among White men and women, and how recent mortality trends for alcohol use appear to be both similar and different by race and ethnicity and gender.

DRUG-RELATED DEATH RATES AMONG U.S. BLACK AND WHITE MEN

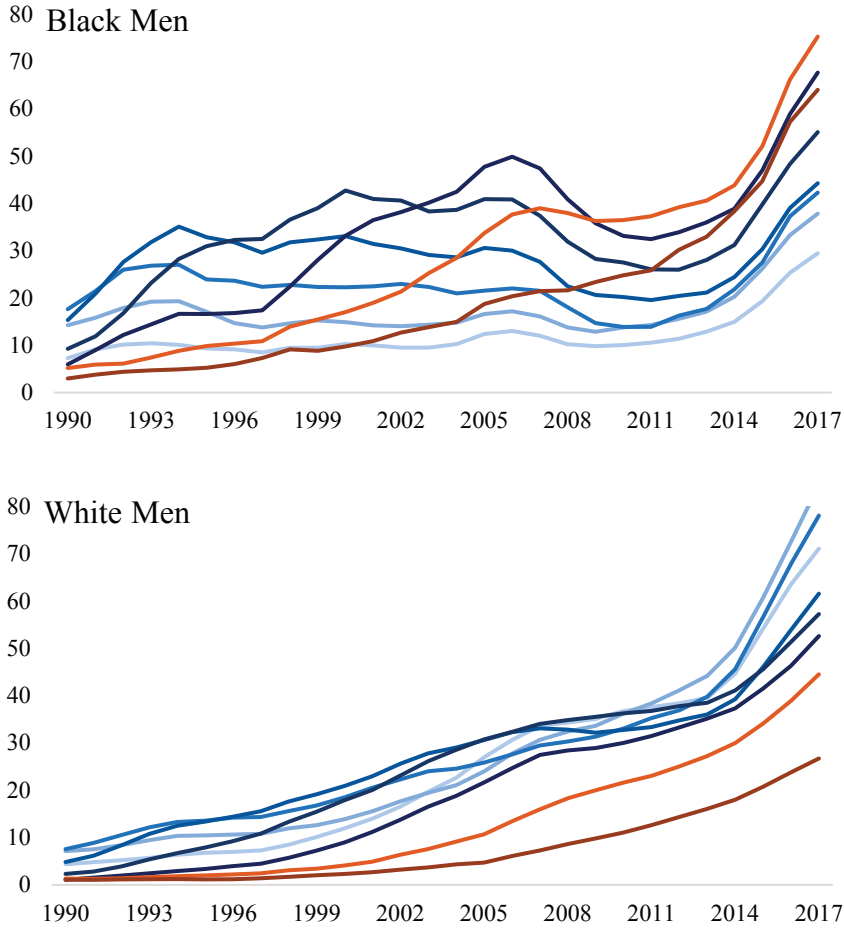
Annex Figure 6-7 plots age-specific mortality rates for drug-related deaths among Black men (top) and White men (bottom) ages 25–64. Blue lines indicate drug-related M_x for ages 25–54, and orange lines indicate M_x for ages 55–64. These ages are differentiated to highlight important race-based differences in drug-related mortality trends among U.S. men.

Drug-related mortality patterns among White men indicate strong period-based trends. There are no substantive differences in drug-related mortality trends for the age groups 25–54, with death rates increasing across time periods in nearly identical ways. Although death rates from drug use are lower among older White men, their trends are similar to those of younger White men. Trends in drug-related mortality rates among Black men exhibit strong period-based variation, but their rates differ from those of White men in two important ways. First, levels of drug-related mortality among U.S. Black men differ considerably by age. Second, the increases in death rates appear to differ by age group as well, indicating possible cohort-based variation in drug-related mortality among Black men. This can be seen most clearly by contrasting the mortality trends (orange lines) for older ages with the average mortality trends for ages 25–54 (blue lines) in Annex Figure 6-8.

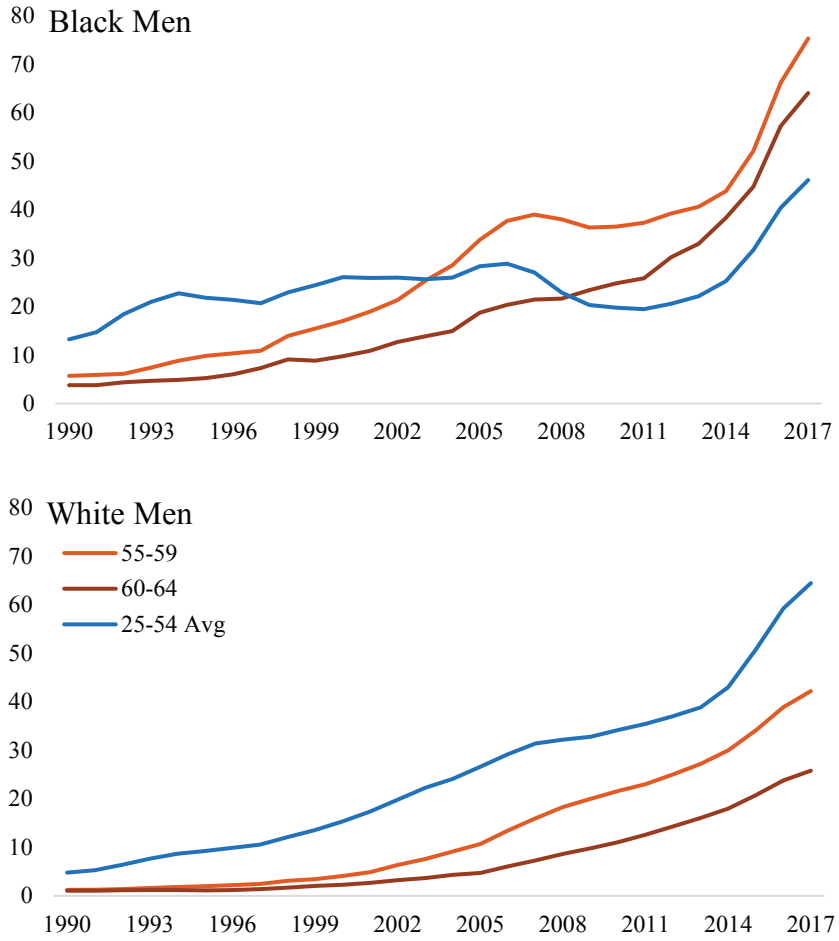
The parallel increases in drug-related M_x for White men provide strong evidence that the rising death rates from drug use among this population are predominantly a period-based phenomenon. In contrast, the disparate increases in drug-related M_x for Black men provide evidence that drug-related deaths among this population reflect both period and cohort effects, whereby older Black men (i.e., earlier birth cohorts) experienced much more rapid increases in death from drug use relative to more recent birth cohorts. In fact, the rates of increase among older Black men are more similar to the large increases observed among all White men than to the trends among younger Black men.

DEATH RATES FROM CARDIOMETABOLIC DISEASES AMONG U.S. WHITE MEN AND WOMEN

Changes in rates of mortality from cardiometabolic diseases (i.e., deaths from all heart diseases, hypertension, and diabetes) among U.S. White men and women exemplify evidence suggesting cohort-based variation in U.S.



ANNEX FIGURE 6-7 Drug-related mortality rates by 5-year age group, 25–29 to 60–64, between 1990 and 2017, U.S. Black and White men.
NOTE: Blue lines indicate M_x for age groups 25–29, and 50–54, and orange lines indicate M_x for age groups 55–59 and 60–64.
SOURCE: Data from https://www.cdc.gov/nchs/nvss/nvss-restricted-data.htm.



ANNEX FIGURE 6-8 Drug-related mortality rates for U.S. Black and White men ages 55–59 and 60–64 versus 25–54 average, 1990–2017.
SOURCE: Data from https://www.cdc.gov/nchs/nvss/nvss-restricted-data.htm.

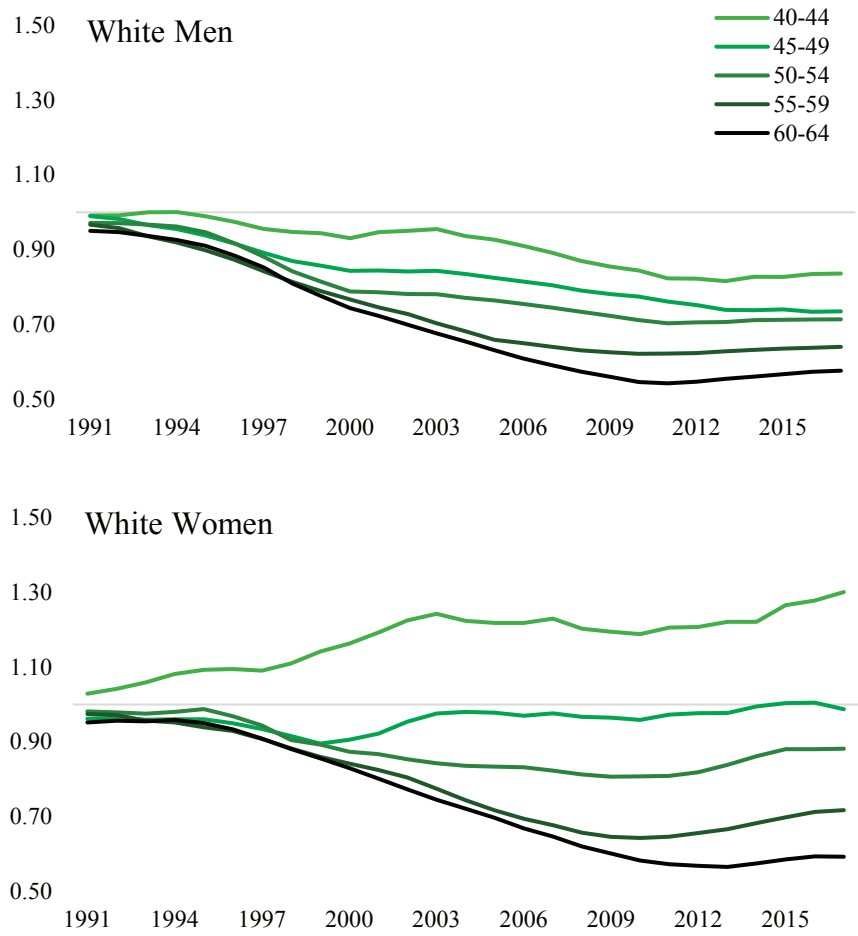
mortality trends. The period-based trends in M_x are plotted on the relative scale because the absolute differences in mortality rates among the age groups and between White men and women are too large to compare on an absolute scale. Specifically, Annex Figure 6-9 plots the M_x from cardiometabolic diseases in each year 1991–2017 to the M_x in 1990.

Among both White men and women, steady relative reductions can be observed in mortality from cardiometabolic diseases among those ages 55–59 and 60–64 until a flattening and reversal occurs in the 2010s. Across the younger age groups, the reductions are less pronounced or even absent among White women. The disparate period-based trends in the age groups' M_x provide clear "nonparallelism" and strong evidence for cohort-based variation in cardiometabolic mortality trends among U.S Whites. The patterns indicate a slowing or even a reversal of reductions in mortality from cardiometabolic diseases across young birth cohorts of White Americans.

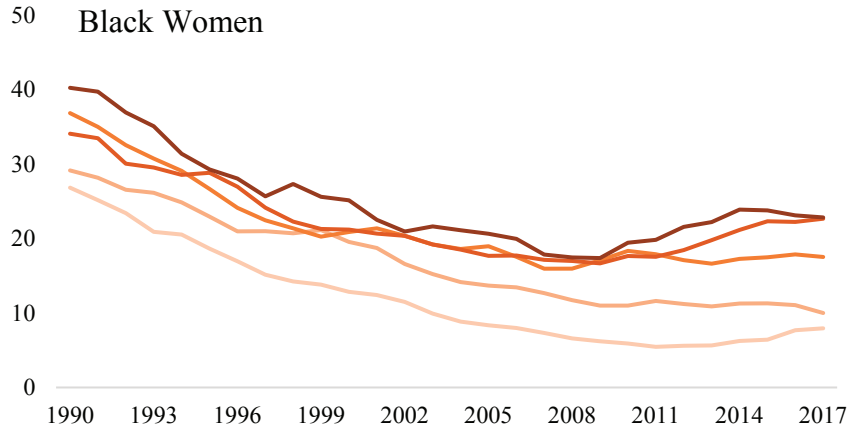
DEATH RATES FROM ALCOHOL USE AMONG U.S. BLACK AND WHITE MEN AND WOMEN

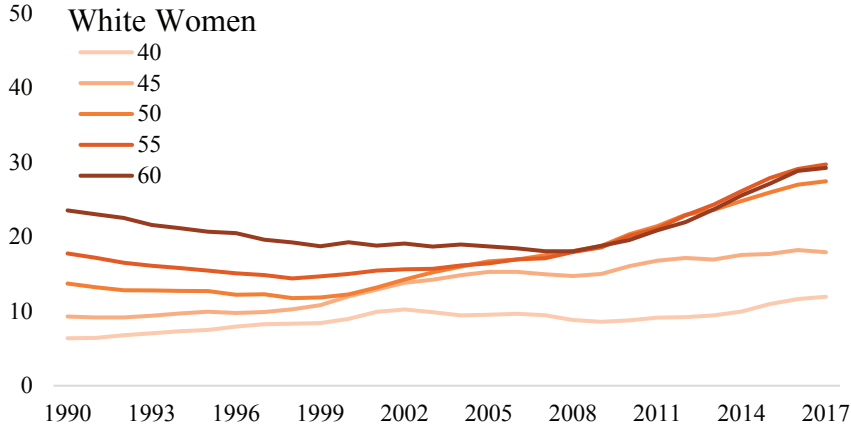
As a final example, evidence for both period- and cohort-based variation in U.S. mortality trends can be seen by plotting rates of mortality from alcohol-related deaths among U.S. Black and White men and women ages 40–64. Annex Figure 6-10 plots M_x from alcohol-related deaths across time periods to illustrate important racial/ethnic- and gender-based differences in the mortality trends.

Across the 1990s and 2000s, death rates from alcohol use declined substantially among the U.S. Black population. Among White men and women, the rates were much lower and relatively stable, albeit with some notable exceptions. Among White men and women ages 50–54 and 55–59, alcohol-related deaths began to increase in the mid-2000s. Corresponding with the Great Recession, reductions in alcohol-related deaths among Black men and women stalled and reversed for older Blacks. Likewise, alcohol-related deaths increased dramatically in the 2010s for White men and women over 50. The recent trends in mortality from alcohol use exhibit a strong period-based pattern for U.S. Black and White men and women, but variation in the trends by race and ethnicity, gender, and age indicates possible cohort-based patterns that are distinct among the U.S. White population.



ANNEX FIGURE 6-9 Mortality rates for cardiometabolic diseases by 5-year age group, 40–44 to 60–64, between 1990 and 2017, U.S. White men and women.
NOTE: Lines indicate the ratio between M_x in each year and M_x in 1990.
SOURCE: Data from https://www.cdc.gov/nchs/nvss/nvss-restricted-data.htm.


Black Women
50
40
30
20
10
0
1990
1993
1996
1999
2002
2005
2008
2011
2014
2017


White Women
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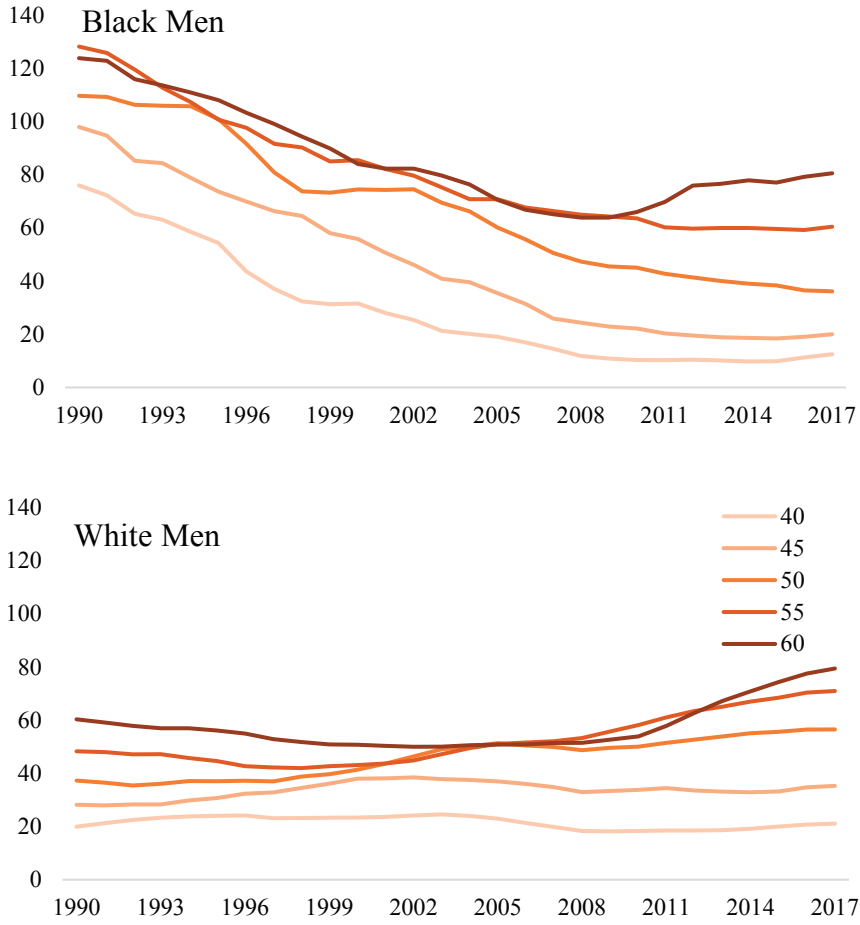

ANNEX FIGURE 6-10 Alcohol-related mortality rate by 5-year age group, 40–44 to 60–64, between 1990 and 2017, U.S. Black and White women and men. SOURCE: Data from https://www.cdc.gov/nchs/nvss/nvss-restricted-data.htm.

7

Opioids, Other Drugs, and Alcohol

Collectively, drugs and alcohol were responsible for more than 1.3 million deaths among the U.S. working-age (ages 25–64) population between 1990 and 2018 (Centers for Disease Control and Prevention [CDC], 2020b).[1,2] Drug poisoning accounted for 756,160 deaths,[3] while 374,197 deaths were alcohol-induced.[4] Mental and behavioral disorders[5]

[1]This figure is based on the International Classification of Diseases, 10th Revision (ICD-10) codes for underlying cause of death. The underlying cause of death is defined by the World Health Organization (WHO, 2011, p. 31) as "the disease or injury which initiated the train of morbid events leading directly to death, or the circumstances of the accident or violence which produced the fatal injury." In this chapter, "other drugs" refers to both illicit and prescription drugs.

[2]Note that the data presented in Chapters 3 and 4 cover the period 1990–2017 because those were the most recent data available at the time the committee conducted the analysis (2019). As this report was being written, data for 2018 were released, so the most up-to-date death counts for deaths due to drugs and alcohol are presented here.

[3]This figure includes intentional drug poisonings (suicides).

[4]In the committee's analyses, alcohol-induced deaths included deaths with an ICD-9 or ICD-10 code representing the following: alcohol-induced pseudo-Cushing syndrome, degeneration of nervous system due to alcohol, alcoholic polyneuropathy, alcoholic myopathy, alcoholic cardiomyopathy, alcoholic gastritis, alcoholic fatty liver disease, alcoholic hepatitis, alcoholic fibrosis and sclerosis of the liver, alcoholic cirrhosis of the liver, alcoholic hepatic failure, alcoholic liver damage, excessive blood level of alcohol, and alcohol poisoning.

[5]Mental and behavioral disorders represent a wide array of causes affecting the brain and associated behaviors, including cognitive diseases (e.g., dementia), mood disorders, disorders related to mental illness (e.g., schizophrenia, bipolar disorder, depression, anxiety), eating disorders, developmental disabilities and mental retardation, and disorders due to alcohol and drugs. Alzheimer's disease is not included in this category but in diseases of the nervous system. In 2018, 198,318 individuals had an ICD-10 code of F10–F19 indicated as their underlying cause of death (mental and behavioral disorder due to psychoactive substances).

due to psychoactive substances (drugs and alcohol) accounted for an additional 198,318 deaths. These substance-related deaths are major contributors to the rise in working-age mortality. Drug poisoning deaths have been rising for almost three decades, primarily among non-Hispanic Whites (Whites) but also among non-Hispanic Blacks (Blacks) and Hispanics. Rates of alcohol-induced death increased among Whites throughout the entire period. Declines in these rates occurred among Blacks and Hispanics throughout the 1990s and early 2000s but leveled off during the late 2000s and increased in the 2010s.

The trend in substance-induced deaths is not abating, and the prevalence of substance use disorders (SUDs) remains high. A 2017 study found that 7.7 million Americans (2.9% of the total population) had a drug use disorder, while 15.7 million (5.9%) had an alcohol use disorder (Segal et al., 2017). A recent study showed that increases in clinical diagnoses related to alcohol misuse, substance misuse, and suicide ideation/behavior between 2009 and 2018 largely mirror the broader mortality trends from these three causes (Brignone et al., 2020). The rise in drug poisoning deaths is well studied, and scholars have offered plausible explanations for this phenomenon. Explanations for recent trends in alcohol-related deaths have been less extensively debated; however, the factors that influence both sets of trends are similar.

This chapter first reviews and summarizes the trends in drug poisoning and alcohol-induced deaths, highlighting the timing, geographic, and racial/ethnic variations in these trends—details essential to any comprehensive explanation of these trends. For example, why were Whites more impacted than Hispanics and Blacks, particularly during the first wave of opioid deaths that was characterized by a major increase in overdoses due to prescription opioids? And why were residents of the Appalachia region impacted more and earlier relative to residents of other areas of the country? The chapter examines possible explanations offered in the literature in light of these trend variations. Ultimately, the committee believes the overall explanation is a combination of increased availability of and access to alcohol and highly lethal drugs (supply), and both underlying long-term vulnerability and increased vulnerability of certain segments of the U.S. population (demand).

TRENDS IN MORTALITY DUE TO DRUG POISONING AND ALCOHOL

Drug Poisoning Mortality

The increase in mortality from drug poisoning over the past three decades has been alarming. Nationally, the drug poisoning mortality

rate increased from 3.4 to 21.7 deaths per 100,000 population (a 538% increase) between 1990 and 2017 (National Center for Health Statistics [NCHS], 2019b). During this period, mortality due to drug poisoning rose more than mortality from any other cause (see Chapter 4). This phenomenon affected all racial/ethnic groups, both men and women, and all U.S. states. Mortality from drug poisoning began to increase in the early 1990s, but these increases accelerated between the late 1990s and mid-2000s and then surged in the 2010s (Figure 7-1). Although these trends were seen among all racial/ethnic groups, their exact timing and pattern varied by sex, age, and race and ethnicity, suggesting that not all working-age adults were equally impacted by each phase.

Drug mortality rates increased throughout the entire study period for White males and females in all three age groups (Figure 7-1). Increases were especially pronounced starting in the early 2010s. Among Black males and females ages 25–44, rates remained relatively low and stable throughout the 1990s and 2000s, followed by an uptick in the 2010s. Rates increased among Black males and females ages 45–54 throughout the 1990s and early 2000s, then declined among Black males and leveled off among Black females during the mid-2000s before again increasing in the 2010s. Rates increased steadily throughout the study period among Black males and females ages 55–64 and then surged in the 2010s. Black males ages 55–64 were the only group to maintain higher drug mortality rates than Whites throughout the entire study period. Among Hispanic males and females ages 25–44, rates remained relatively low and stable until the 2010s, when these groups experienced increases similar to those among the other groups. Rates among Hispanic males ages 45–54 also remained comparatively low and stable until the 2010s, when they increased. Hispanic females ages 45–54 had stable rates throughout the 1990s and early 2000s but saw small but consistent increases starting in the mid-2000s. Finally, rates among Hispanic males and females ages 55–64 were low and stable throughout the 1990s and early 2000s but began to increase in the mid-2000s.

Although the committee did not perform independent analyses of mortality by education or other measures of socioeconomic status (SES), several previous studies examined SES differences in drug-related mortality and found large and growing disparities among working-age White adults (Case and Deaton, 2015, 2017, 2020; Geronimus et al., 2019; Ho, 2017). Case and Deaton's (2015) seminal study of increasing midlife mortality from "drugs, alcohol, and suicide" showed that the death rate among U.S. adults ages 45–54 due to poisoning—which included prescription and illicit drug poisoning and alcohol-related deaths, both unintentional and of undetermined intentionality—increased for White adults of all educational levels, as well as for Black and Hispanic adults, between 1999 and 2013. However, the increase was especially pronounced for Whites with a high

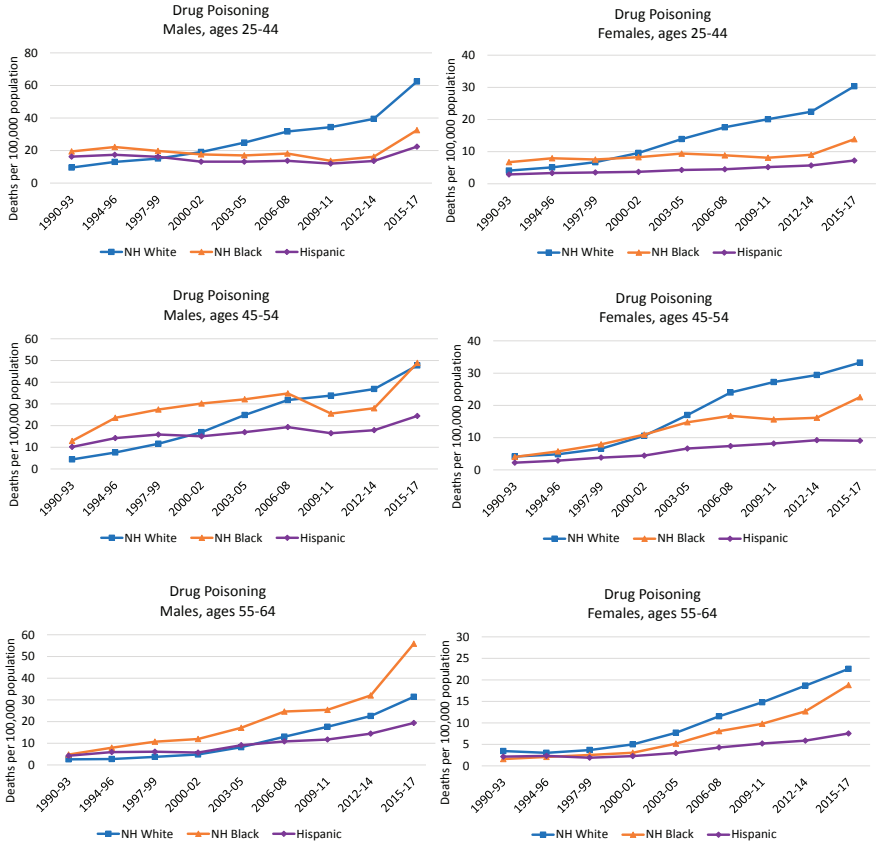

FIGURE 7-1 Mortality rates among U.S. working-age adults (ages 25–64) (deaths per 100,000 population) from drug poisoning by sex, age, and race and ethnicity.
NOTE: Each panel of the figure shows mortality rates for non-Hispanic (NH) Whites (blue line), NH Blacks (orange line), and Hispanics (purple line). Mortality rates for males are shown in the lefthand panels, while those for females are shown in the righthand panels. Mortality rates are shown for three age groups: 25–44 (top panels), 45–54 (middle panels), and 55–64 (bottom panels). Rates are age-adjusted to reflect a standard population age distribution.
SOURCE: Data from National Vital Statistics System Detailed Mortality Files, https://www.cdc.gov/nchs/nvss/deaths.htm.

school degree or less, among whom the death rate for poisoning increased more than four-fold over the period.

Those findings provided the first clear evidence that working-age drug poisoning mortality was increasing more rapidly among less-educated than among more highly educated White adults. Unfortunately, Case and Deaton (2015) did not break down the figures for Black or Hispanic adults by educational attainment; notably, though, both groups exhibited increases in poisoning mortality at ages 45–54 between 1999 and 2013.

In a subsequent study, Ho (2017) conducted a thorough analysis of changes in U.S. death rates due to drug poisoning between 1992 and 2011, stratified by educational attainment. Drug poisoning death rates increased among White adults (both males and females) at all levels of education, even during the earliest period of 1992–1996, before the emergence of OxyContin. The increases were, however, especially steep in the 2000s compared with the 1990s among those ages 30–60 compared with older adults, among White adults compared with the population as a whole, and among those with a high school education or less compared with those with a college degree or more. Thus, Ho (2017) concluded that the increased death rate from drug poisoning from the early 1990s to the 2010s was especially steep among the less educated and accounted for large shares (~70% for men and ~44% for women) of increasing educational disparities in working-age mortality over this period.

Most recently, Geronimus and colleagues (2019) documented changes in educational disparities in working-age (and older) mortality between 1990 and 2015 for Black and White women and men. This study measured educational attainment using population quartiles to help account for the effects of increasing educational attainment across time. Similar to Ho (2017), Geronimus and colleagues (2019) demonstrated that increasing drug-related mortality was especially concentrated among lower-educated White adults and accounted for 73 percent and 44 percent of the increasing educational disparity in working-age mortality for White men and White women, respectively. The authors concluded that one-half (White women) to 80 percent (White men) of the increasing educational disparity in working-age mortality over the 1990–2015 period was due to drugs, alcohol, or suicide, with educational differences in drug poisoning mortality particularly important for understanding widening educational disparities in working-age mortality among White women and men since 1990. For Black women and men, however, the findings differed. Increases in drug-related mortality among Blacks differed only very modestly by educational attainment and thus had very little influence on changing educational disparities in working-age mortality.

There are also important geographic differences in the trends in drug poisoning mortality rates. Rates increased among all racial/ethnic groups

in all metro status categories between 1990 and 2017, but the increases were steeper for some groups than others and varied in their timing (Figure 7-2). In general, differences by metropolitan status among working-age White adults are small. The gap between large metropolitan areas (hereafter referred to as "large metros") and other areas grew the most among White males, but nonmetropolitan areas (hereafter referred to as "nonmetros") and small and medium metropolitan areas (hereafter referred to as "small/medium metros") experienced slower increases in mortality from drug poisoning, particularly during the 2010s. This finding suggests that mortality due to drug poisoning is not responsible for the growing gap in all-cause mortality between large central metros and nonmetros among working-age White adults. However, Monnat (2020a) notes substantial variation in drug poisoning mortality across different rural areas in the United States, and while some rural areas have among the lowest drug mortality rates in the country, others have the highest. Combining all rural areas into one composite rate averages out these wide divergences and masks the reality that drug poisonings accounted for a large share of the widening rural mortality penalty in certain regions (e.g., Appalachia, New England) and economic contexts (e.g., mining counties) (Monnat, 2020b).

Among working-age Black adults, drug mortality rates were highest in large central metros and lowest in nonmetros throughout the period. Younger Black males and females in large central metros experienced a decline in drug mortality during the 2000s, but their rates increased again in the 2010s. Among Hispanic males in both age groups, rates were highest in large fringe metros and generally lowest in nonmetros. Among Hispanic females, rates were lowest in large central metros.

In some ways, the drug overdose crisis can be considered a national crisis, as drug poisoning mortality rates increased in every U.S. state over the study period (Figure 7-3). However, drug mortality rates were disproportionately higher and increased more in some parts of the country than others, with the highest rates concentrated in Appalachia, New England, Florida, eastern Oklahoma, and the desert Southwest (Monnat, 2018, 2019, 2020b; Monnat et al., 2019; Rigg et al., 2019; Rossen et al., 2017). The committee's analysis showed that working-age drug mortality rates increased for both males and females in all states from 1990 to 2017, but the increases were most pronounced in West Virginia (more than 2000% for both males and females). Among males, the other top-ranked states for increases were Maine, Ohio, Vermont, Kentucky, and New Hampshire. For females, the other top-ranked states for increases were New Jersey, Ohio, Kentucky, and Maine.

Most fatal drug overdoses involve opioids (see Figure 7-4). The surge in fatal drug overdose rates among all groups in the 2010s was due primarily to fentanyl, a synthetic and highly potent illicit opioid with high overdose

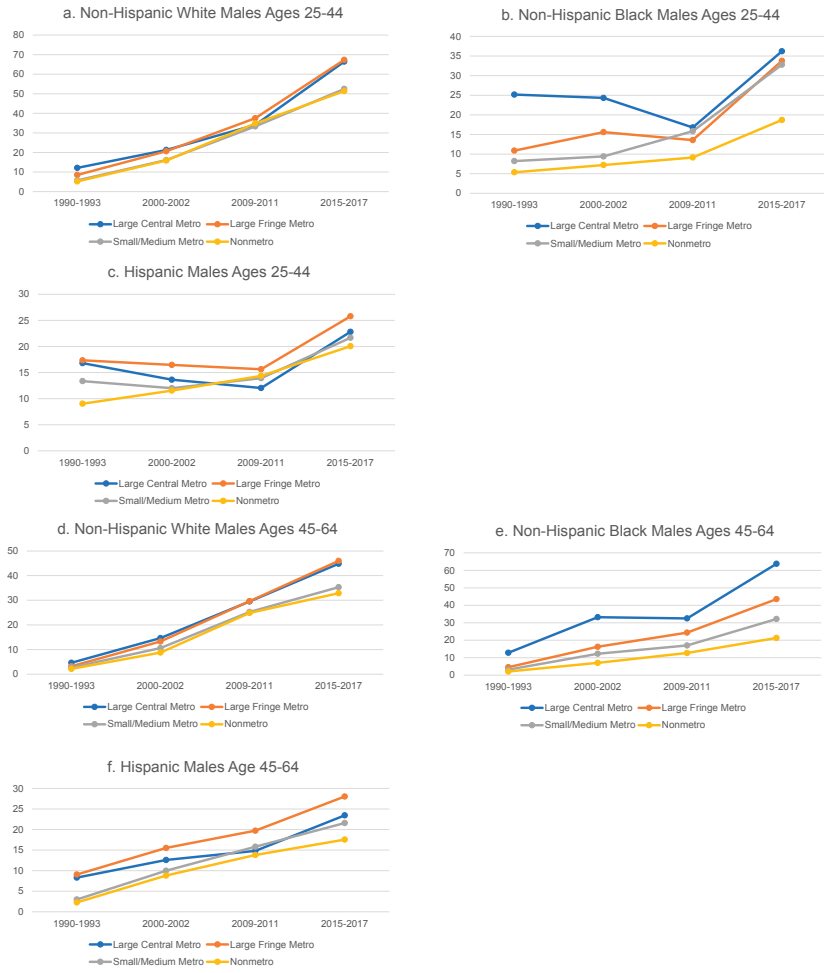

FIGURE 7-2 Drug poisoning mortality rates (deaths per 100,000 population) for U.S. working-age males and females (ages 25–64) by metropolitan status, 1990–1993 through 2015–2017.

NOTE: Drug poisoning mortality rates are shown for ages 25–44 (panels a-c and g-i) and 45–64 (panels d-f and j-l) across four levels of metropolitan status: (1) large central metropolitan areas (blue lines); (2) large fringe metropolitan areas (orange lines); (3) small or medium metropolitan areas (gray lines); and (4) nonmetropolitan areas (yellow lines). Trends in these four groups are presented separately by sex (males in panels a-f, females in panels g-l) and for non-Hispanic (NH) Whites (panels a, d, g, and j), NH Blacks (panels b, e, h, and k), and Hispanics (c, f, i, and l). Rates are age-adjusted by 10-year age group.

SOURCE: Data from National Vital Statistics System Detailed Mortality Files, https://www.cdc.gov/nchs/nvss/deaths.htm.

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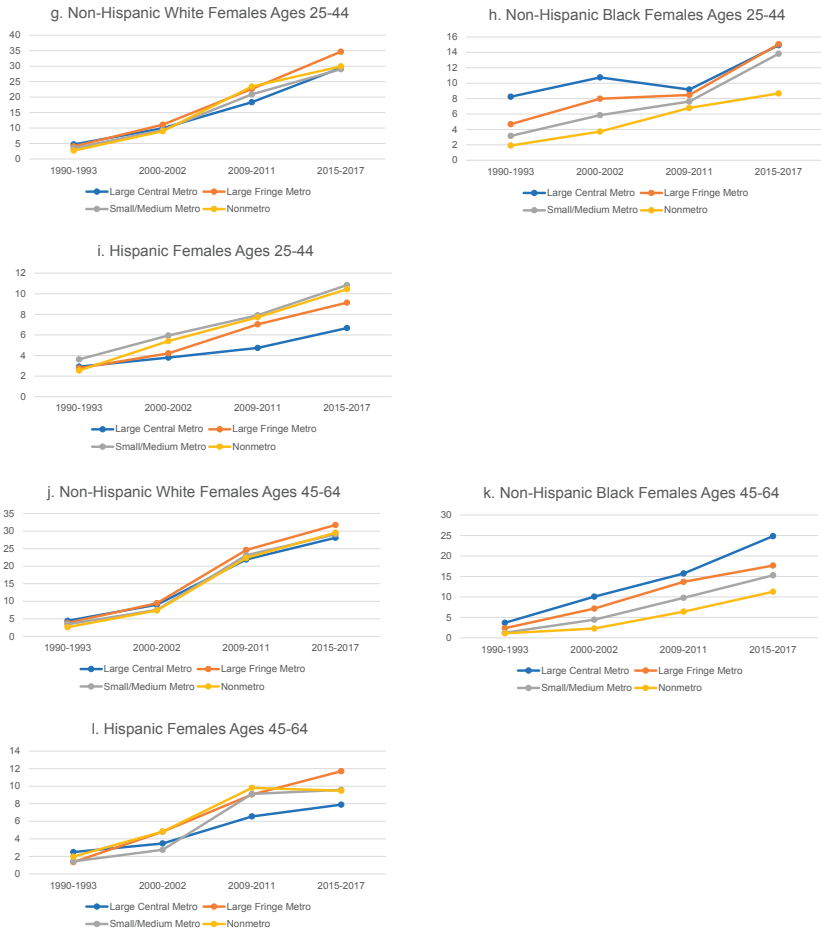

FIGURE 7-2 Continued

risk (Kiang et al., 2019; Monnat et al., 2019; Peters et al., 2020). Fentanyl was the primary contributor to overdoses among all racial/ethnic groups starting in the mid-2010s, while Whites continued to have higher rates of overdose from prescription opioids relative to other racial/ethnic groups. In state- and county-level analyses, prescription opioids, heroin, and fentanyl were found to be differentially implicated in overdoses across different parts of the United States. For example, synthetic opioid deaths were strongly concentrated throughout the East, whereas heroin overdoses were highest in the industrial Midwest and New Mexico (Kiang et al., 2019; Monnat, 2019; Peters et al., 2020; Ruhm, 2017).

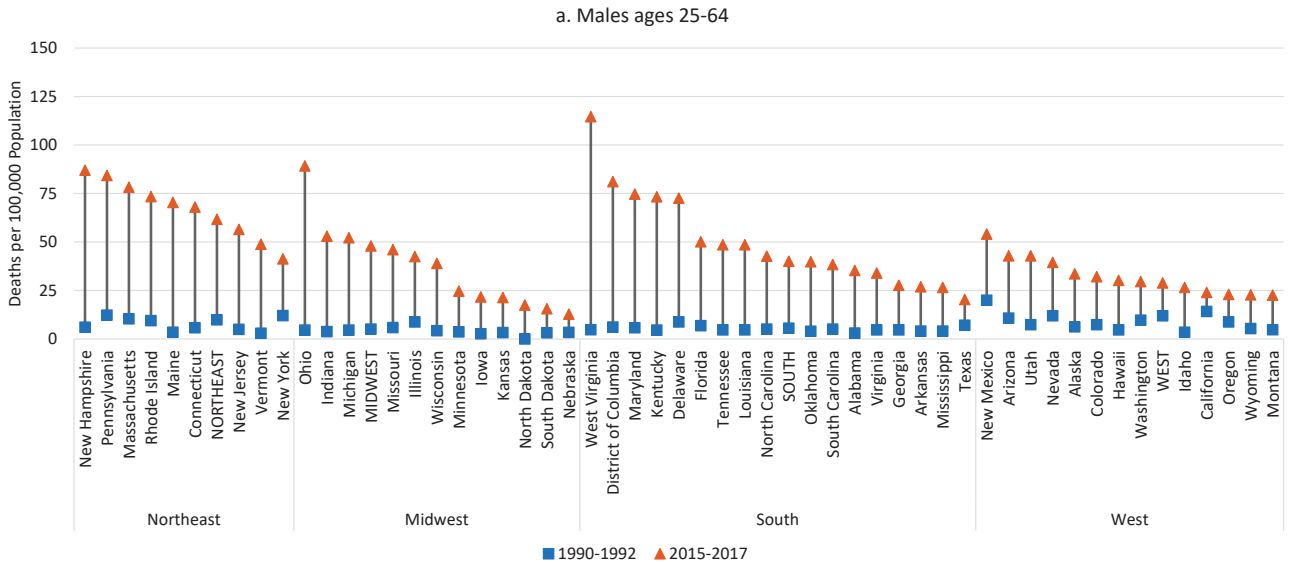

FIGURE 7-3 Drug poisoning mortality rates (deaths per 100,000 population) for U.S. working-age males and females (ages 25–64) by region and state, 1990–1992 and 2015–2017.

NOTE: Drug poisoning mortality rates are shown for 1990–1992 (blue squares) and 2015–2017 (orange triangles) along with the changes over time (black connecting lines). Mortality for males is shown in panel a, while mortality for females is shown in panel b. Rates are age-adjusted by 10-year age group. For males, the 1990–1992 rate for Alaska represents 1991 and 1992 only; the rate was suppressed for 1990. North Dakota is excluded to comply with Centers for Disease Control and Prevention (CDC) suppression criteria (fewer than 10 deaths in 1990–1992). The District of Columbia is excluded for females for the same reason. States are ordered from highest to lowest mortality rate in 2015–2017 within region.

SOURCE: Data from CDC WONDER Online Database, https://wonder.cdc.gov.

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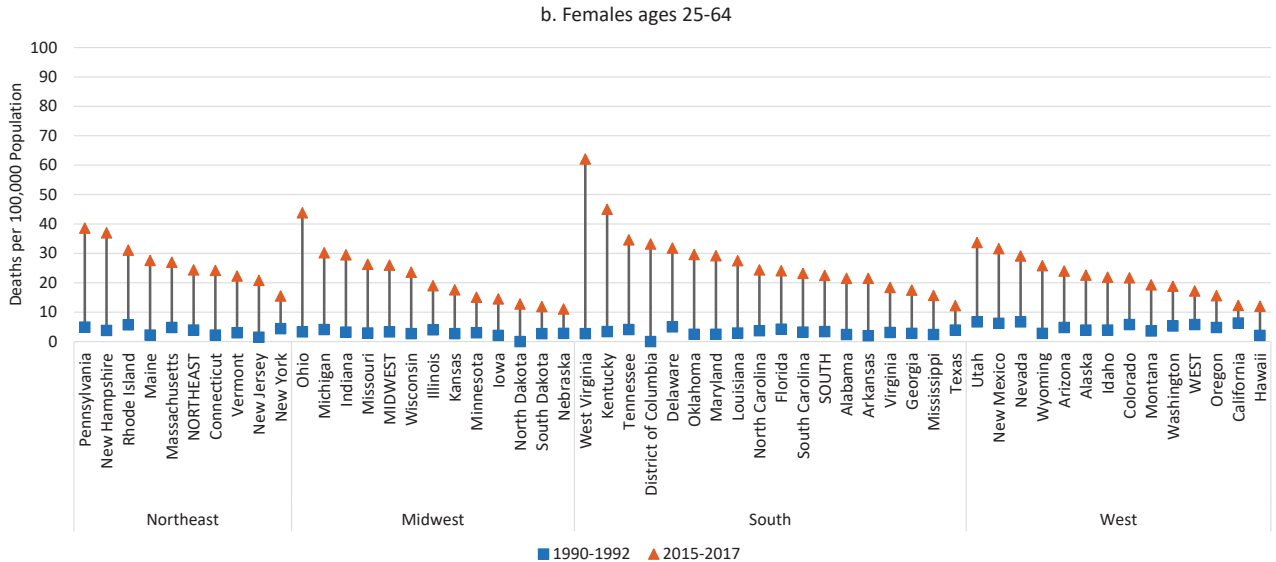

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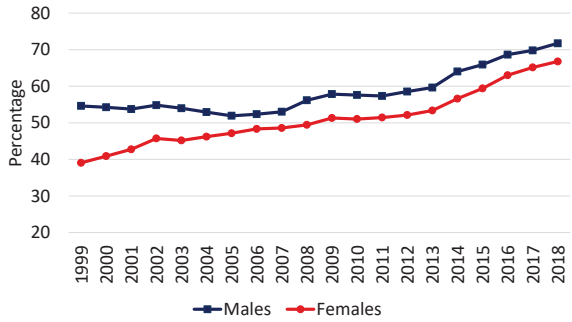


FIGURE 7-4 Percentage of all fatal drug poisonings among U.S. working-age adults (ages 25–64) that involved opioids, by sex, 1999–2018.
NOTE: The percentage of all fatal drug poisonings that involved opioids is shown for males (blue squares) and females (red circles). Drug poisonings that involve opioids are those for which opioids are recorded as the underlying cause of death (International Classification of Diseases [ICD]-10 code of X40–X44, X60–X64, X85, or Y10–14) and/or a multiple cause-of-death code signifies opioid involvement (T40.0, T40.1, T40.2, T40.3, T40.4, and T40.6).
SOURCE: Data from CDC WONDER Online Database (https://wonder.cdc.gov/).

Between 1999 and 2018, the share of drug poisonings involving opioids increased from 54.6 to 71.7 percent among working-age males and from 39.1 to 66.8 percent among working-age females. Based on death certificates only, opioids were involved in more than 386,000 working-age deaths between 1999 and 2018 (see Figure 7-4). However, nearly a quarter of death certificates indicating drug poisoning do not specify the drug involved (Ruhm, 2018a). As a result, opioid deaths are underreported on death certificates by as much as 20–35 percent, depending on the year (Ruhm, 2018a). Philadelphia County, Pennsylvania, offers a good example. There were 1,047 fatal drug overdoses in 2017 in that county, but an opioid-specific International Classification of Diseases (ICD) code was included on only 45 death certificates (CDC, 2020b).

Figure 7-5 shows trends in opioid-involved drug poisoning deaths from 1979 to 2018 for working-age (ages 25–64) males and females. The figure displays a break between 1998 and 1999 due to changes in ICD coding between those years, and readers should compare trends separately for 1979 to 1998 and 1999 to 2018. Even with this break, however, one can see that opioid deaths began increasing among males in the early 1990s, prior to the release of OxyContin in 1996. Opioid-involved deaths rose precipitously between 1999 and 2017. Over that period, the opioid-involved drug poisoning death rate rose from 7.5 to 34.6 deaths per 100,000 population among working-age males and from 2.5 to 15.3 deaths per 100,000 population among working-age females. For the first time in more than two

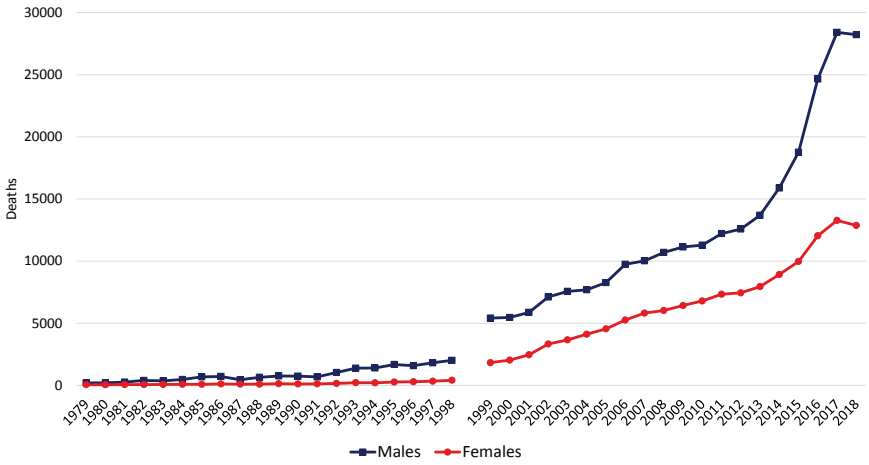


FIGURE 7-5 Drug poisonings involving opioids among U.S. working-age adults (ages 25–64) by sex, 1979–2018.
NOTE: The number of fatal drug poisonings involving opioids is shown for working-age males (blue squares) and females (red circles). A break is shown between 1998 and 1999 because of changes in International Classification of Diseases (ICD) codes between those years. Opioid overdoses are likely undercounted prior to 1999. For 1979–1998, deaths were classified as a drug poisoning involving opioids if they were assigned an underlying cause-of-death ICD-9 code of E850.0. For 1999–2018, deaths were classified as a drug poisoning involving opioids if they were assigned an underlying cause-of-death ICD-10 code for drug poisoning (X40–44, X60–64, X85, and Y10–Y14) or a multiple cause-of-death code for an opioid (T40.0, T40.1, T40.2, T40.3, T40.4, and T40.6).
SOURCE: Data from CDC WONDER Online Database, https://wonder.cdc.gov.

decades, there was a slight decline in fatal opioid poisonings among both males and females in 2018. However, the most recent drug mortality data available to the committee, covering 2019 and early 2020, suggest that this small decline may have been temporary and not a reversal of the long-term increase in opioid deaths (Ahmad, Rossen, and Sutton, 2020).

Figure 7-6 presents trends in drug poisoning by specific drug among working-age (ages 25–64) males and females, 1999–2018. The figure does not present trends prior to 1999 because of the lack of comparability in drug-specific ICD codes pre- versus post-1999. The contemporary drug overdose crisis has been described as a triple-wave epidemic (Ciccarone, 2019). Wave 1 (1990s to late 2000s) was characterized by an increase in overdoses due to prescription opioids (e.g., oxycodone, hydrocodone). Wave 2 (mid-2000s to early 2010s) was characterized by a surge in heroin overdoses, which increased as prescription opioids became more difficult

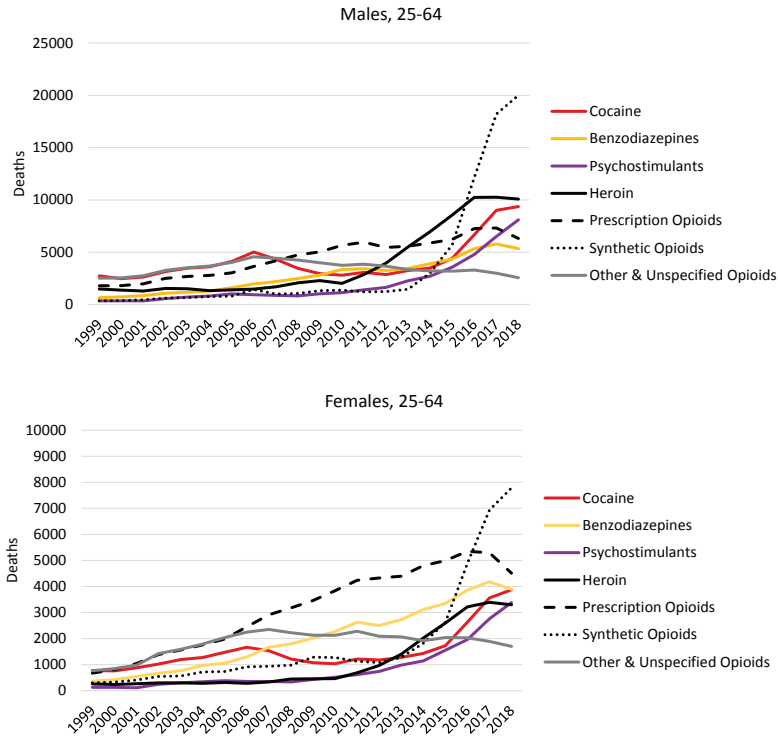


FIGURE 7-6 Specific drug involvement in drug poisonings among U.S. working-age adults (ages 25–64) by sex, 1999–2018.
NOTE: The number of deaths is shown for deaths involving cocaine (red line), benzodiazepines (yellow line), psychostimulants (purple line), heroin (solid black line), prescription opioids (dashed line), synthetic opioids (dotted line), and other & unspecified opioids (grey line), separately for working-age males (upper panel) and females (lower panel). Deaths are not mutually exclusive; more than one drug can be involved in a single death. Therefore, deaths are counted under each drug that was involved. Deaths were classified as drug poisonings if the underlying cause of death was a drug poisoning ICD-10 code (X40–44, X60–64, X85, and Y10–Y14). Specific drugs were identified using the multiple cause-of-death codes: cocaine (T40.5); benzodiazepines (T42.4); psychostimulants, including methamphetamine (T43.6); heroin (T40.1); prescription opioids (T40.2); synthetic opioids (T40.4); and other & unspecified opioids (T40.0, T40.3, and T40.6)
SOURCE: Data from CDC WONDER Online Database, https://wonder.cdc.gov.

and costly to procure and as drug cartels caught on to the demand for opioids in the United States and increased the supply of heroin (Quinones, 2015). Wave 3 (2010s) was characterized by a massive increase in overdoses due to synthetic opioids, including fentanyl and fentanyl-related compounds and derivatives. As overdoses from prescription opioids and heroin began to level off, fentanyl overdoses surged to become the primary contributor to overdose deaths.

The media, scholarly, and political focus on opioids is well deserved, but it is important to note that more than half of drug overdoses involve multiple substances, and overdoses from other drugs, including benzodiazepines, cocaine, and methamphetamine, also increased over this period (Gladden et al., 2019) (see Figure 7-6). Many people who misuse prescription opioids also use illegal drugs (Rigg and Monnat, 2015a, 2015b; Rigg et al., 2019). In 2015, among those reporting misuse of prescription opioids, 72 percent reported using heroin; 52 percent methamphetamine; and approximately a third cocaine, LSD, or ecstasy. In what has been described as the fourth wave (Cano and Huang, 2020) of the drug overdose crisis, overdoses from stimulants surpassed those from prescription opioids to compete with heroin (see Figure 7-6).

Alcohol-Induced Mortality

Mortality from alcohol-induced causes followed different trends from those involving drug poisoning, although here, too, working-age Whites experienced larger increases in mortality relative to working-age Blacks or Hispanics (Figure 7-7). In fact, Whites were the only racial/ethnic group to experience an overall increase in the alcohol-induced mortality rate between 1990 and 2017.[6] Among White males ages 45–54, the alcohol-induced mortality rate increased from 13.3 to 21.8 per 100,000 population, representing 7.4 percent of the overall increase in mortality for this population. Among White males in both the younger (25–44) and older (55–64) working-age groups, the alcohol-induced mortality rate increased from 4.8 to 6.7 and 20.7 to 31.6 per 100,000 population, respectively.

For many working-age adults, alcohol-induced mortality followed a similar pattern over the period, with declines in the 1990s, followed by increases that began in the mid-2000s and continued into the 2010s. This pattern held among White males in the younger working-age group (25–44); White, Black, and Hispanic males and females in the oldest working-age group (55–64); and Hispanic females ages 45–54. The primary

[6]Trends are not presented for Asians or American Indians. There were slight increases among Hispanic women in all three age groups, but the increases were less than 1 death per 100,000 population.

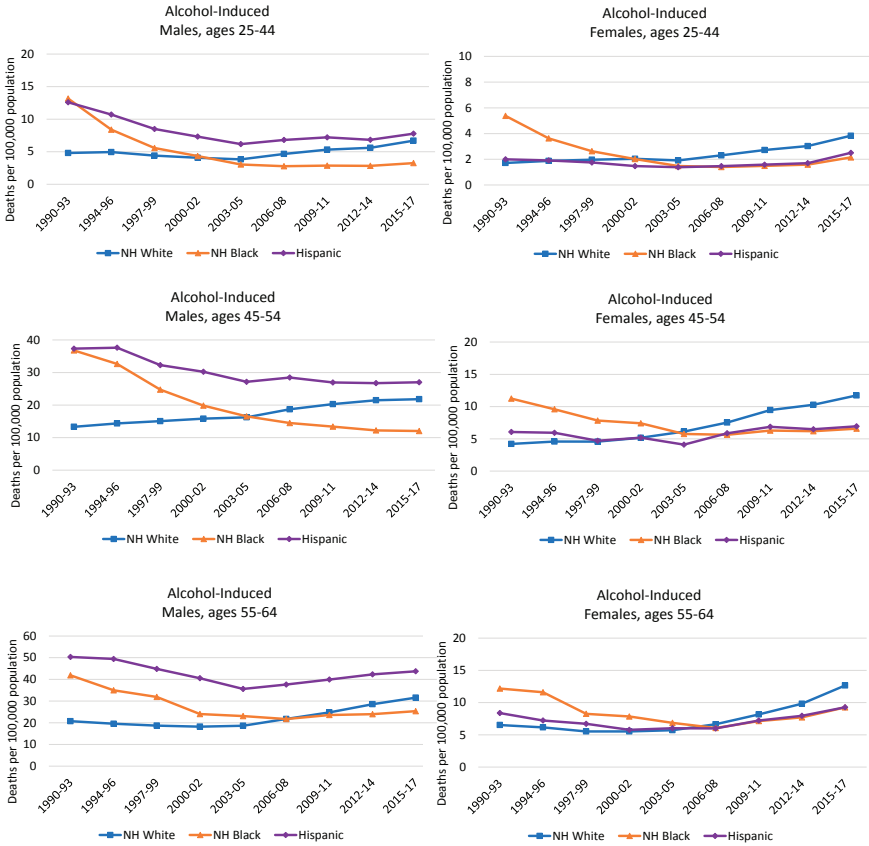


FIGURE 7-7 Mortality rates (deaths per 100,000 population) for U.S. working-age adults for alcohol-induced causes by sex, age, and race and ethnicity.
NOTE: Each panel shows mortality rates for non-Hispanic (NH) Whites (blue line), NH Blacks (orange line), and Hispanics (purple line). Mortality rates for males are shown in the lefthand panels, while those for females are shown in the righthand panels. Mortality rates are shown for three age groups: 25–44 (top panels), 45–54 (middle panels), and 55–64 (bottom panels). Rates are age-adjusted to reflect a standard population age distribution.
SOURCE: Data from National Vital Statistics System Detailed Mortality Files, https://www.cdc.gov/nchs/nvss/deaths.htm.

difference between White adults and Hispanic or Black adults was that decreases were larger among Hispanic and Black adults in the initial period, while the increases after the mid-2000s were larger among White than among Hispanic and Black adults, leading to larger overall increases among White adults. Black and Hispanic males and Black females in the 25–44 and 45–54 age groups initially followed a similar pattern of decreasing alcohol-induced mortality until the mid-2000s; however, alcohol-induced mortality among these groups stagnated throughout the remainder of the period rather than increasing. In contrast, White females ages 25–54 and White males ages 45–54 experienced an increase in alcohol-induced mortality throughout the period, with the largest increases generally occurring after the mid-2000s. Despite this steady increase, White females maintained lower alcohol-induced mortality rates relative to White males throughout the period. Rates remained constant throughout most of the period among Hispanic females ages 25–44, only increasing slightly in the most recent period, between 2012–2014 and 2015–2017. These mortality trends are consistent with those identified by Kerr and colleagues (2009), who found a significantly lower volume of alcohol consumption among Hispanic and Black relative to White respondents in six U.S. national alcohol surveys conducted between 1979 and 2005.

Vierboom, Preston, and Hendi (2019) examined trends in educational differences in alcohol-related mortality between 2000 and 2017 using a comprehensive definition of alcohol-attributable mortality based on the Centers for Disease Control and Prevention's (CDC's) Alcohol Related Disease Impact Classification; the latter includes 48 alcohol-related causes of death and causes that are influenced indirectly by alcohol use. They found that at ages 45–49, alcohol-related mortality increased at all educational levels over this period, with the smallest increases among men and women with a college degree and the largest increases among those with less than a high school education.

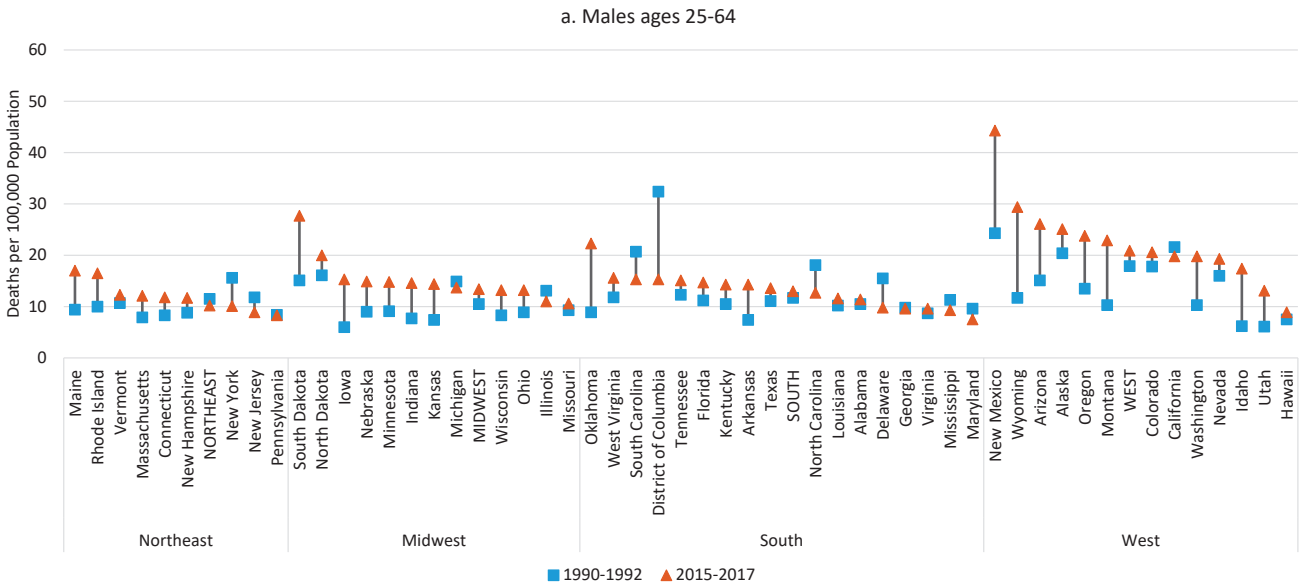
Our analysis shows that the increase in alcohol-induced deaths from 1990 to 2017 was mostly a national phenomenon (Figure 7-8). Differences across metro status categories were generally small, with somewhat more favorable trends within large central metros.[7] Working-age alcohol-induced deaths increased in all but 13 states among males and in all but 5 states among females. In keeping with the racial/ethnic dimension of this trend, the largest percentage increases occurred in Idaho, Iowa, Wyoming, Oklahoma, and Montana for males and in Iowa, North Dakota, Wyoming, Oklahoma, and Arkansas for females. States with

[7]The most notable differences by metro status occurred for Hispanic females, among whom alcohol-induced mortality was much lower in nonmetros than in other areas in the early 1990s, but higher than in other areas by the end of the period. See Appendix A for full results.

the largest percentage declines were Delaware, New York, North Carolina, and South Carolina plus the District of Columbia (DC) for males and Maryland, New York, New Jersey, and South Carolina plus DC for females. States with the largest percentage increases had relatively high proportions of White residents, while states where the steepest percentage declines occurred had relatively high proportions of Black residents.

The increases in alcohol-induced mortality reported above likely reflect much earlier changes in consumption patterns. The temporal trends observed in alcohol-induced mortality align with temporal trends in per capita alcohol consumption (Kerr et al., 2009, 2013a). The majority of alcohol-induced deaths are due to chronic liver disease and liver cirrhosis. For most people, it takes many years of heavy drinking to develop and succumb to these diseases. Peak alcohol consumption in the United States occurred during the mid-1970s to early 1980s (see Figure 7-4; Haughwout and Slater, 2018). There was then a sharp drop in consumption between the early 1980s and 1997, followed by a slow rise starting in 1998. Assuming that the main cohort of drinkers during the peak consumption period (mid-1970s to early 1980s) were ages 20–40, most of them would have been ages 35–55 at the start of the study period (1990).

Using data from six U.S. National Alcohol Surveys conducted between 1979 and 2005, Kerr and colleagues (2009) conducted age–period–cohort (APC) analysis and found a strong cohort effect for increased alcohol consumption among those born after 1975. In another APC study, using data from seven cross-sectional studies of the United States representing more than 36,000 adults (ages 18 and over), Kerr and colleagues (2013a) found that age effects for drinking beer typically peak in the early 20s and then decline, whereas wine and spirits have relatively flat age profiles. They also found that men born between 1976 and 1985 and women born between 1981 and 1985 have higher alcohol consumption relative to earlier or later birth cohorts. Among women, however, they noted an additional pattern for the 1956–1960 birth cohort, which stands out from adjacent cohorts as having high alcohol consumption and binge drinking. The authors note that members of this cohort were in their early 20s during the peak of U.S. per capita alcohol consumption (early 1980s), and this fact, along with relaxed social sanctions on female alcohol use, may have led these women to develop heavier drinking habits at that time. They conclude that "it is especially important to try to understand characteristics of the 1976-1985 birth cohorts, and the conditions between 1995 and 2005 when their drinking habits were being developed" (Kerr et al., 2013a, p. 1041). Based on a systematic review of 68 studies, Slade and colleagues (2016) similarly found a closing of the gender gap in alcohol consumption between the early 1990s and late 1990s birth cohorts.


a. Males ages 25-64
Deaths per 100,000 Population
60
50
40
30
20
10
0
Maine
Rhode Island
Vermont
Massachusetts
Connecticut
New Hampshire
NORTHEAST
New York
New Jersey
Pennsylvania
South Dakota
North Dakota
Iowa
Nebraska
Minnesota
Indiana
Kansas
Michigan
MIDWEST
Wisconsin
Ohio
Illinois
Missouri
Oklahoma
West Virginia
South Carolina
District of Columbia
Tennessee
Florida
Kentucky
Arkansas
Texas
SOUTH
North Carolina
Louisiana
Alabama
Delaware
Georgia
Virginia
Mississippi
Maryland
New Mexico
Wyoming
Arizona
Alaska
Oregon
Montana
WEST
Colorado
California
Washington
Nevada
Idaho
Utah
Hawaii
Northeast
Midwest
South
West
1990-1992
2015-2017

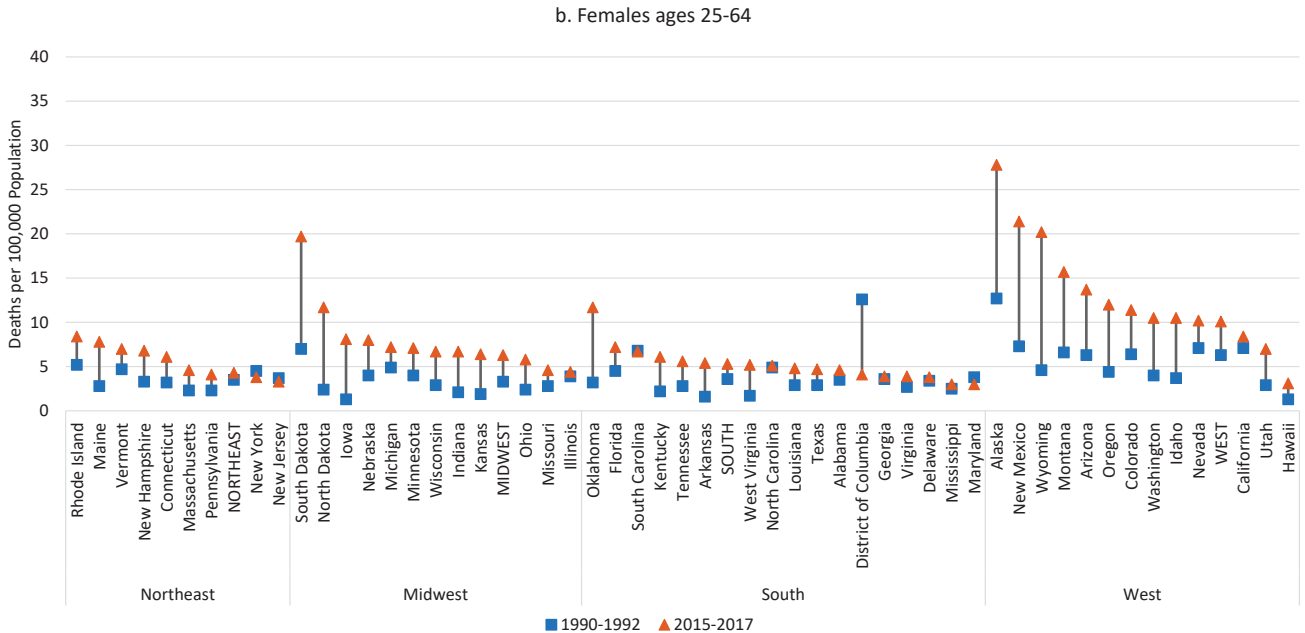


FIGURE 7-8 Alcohol-induced mortality rates (deaths per 100,000 population) for U.S. working-age males and females (ages 25–64) by region and state, 1990–1992 and 2015–2017.
NOTE: Mortality rates for alcohol-induced deaths are shown for 1990–1992 (blue squares) and 2015–2017 (orange triangles), along with the changes over time (black connecting lines). The rates for males are shown in panel a, while the rates for females are shown in panel b. Rates are age-adjusted by 10-year age group. States are ordered from highest to lowest mortality rate in 2015–2017 within region.
SOURCE: Data from CDC WONDER Online Database, https://wonder.cdc.gov.

Between the early 1980s and late 1990s, per capita consumption of alcohol (particularly beer and spirits) declined in all four major regions of the United States. Beer consumption continued to decline throughout the 2010s, but consumption of wine and spirits began to rise in the late 1990s, driving an overall increase in alcohol consumption since that time (see Figure 7-9; Haughwout and Slater, 2018).

Summary of Trends in Mortality from Drugs and Alcohol

In some ways, the trends in mortality due to drug poisoning and alcohol-related causes suggest similarities in the affected populations.[8] Both causes of death increased steadily over the 1990–2017 study period among working-age Whites. Although more research on socioeconomic disparities in mortality has examined differences in mortality due to drug poisoning rather than alcohol-induced mortality, the research collectively suggests that among working-age Whites, particularly men, increased mortality from both causes was greater among those with a high school degree or less than among those with a college degree. Mortality due to substance use generally (drug and alcohol use) explains most of the growth in the socioeconomic gap in mortality among men and about half of the growth in the gap among women. Differences in mortality due to drug poisoning and alcohol-induced causes between metro and nonmetro areas followed similar trends, even as regional trends differed. Neither cause of death contributed to the growing mortality gap between metro and nonmetro areas.

Despite these similarities, there are also important differences in drug mortality and alcohol-induced mortality trends. In particular, the timing, racial/ethnic and age profiles, and geography of these trends vary. For example, drug mortality increased among older working-age Black males during the 1990s; however, mortality from alcohol-induced causes decreased substantially among this group. And although both drug and alcohol-induced mortality rates increased among working-age Whites, younger working-age Whites experienced larger increases in drug poisoning mortality relative to their older counterparts, who experienced larger increases in mortality from alcohol-induced causes. With respect to geography, Western states experienced the smallest increase in drug mortality but the largest increases in alcohol-induced mortality.

[8]The committee also examined trends in mortality from mental and behavioral disorders. These trends largely mirrored the trends for alcohol-related mortality; therefore, they are not presented here. Appendix A presents the full set of cause-specific mortality trends by sex, age group, and race and ethnicity, as well as the cause-specific mortality trends by sex, age group, race and ethnicity, and metropolitan status.

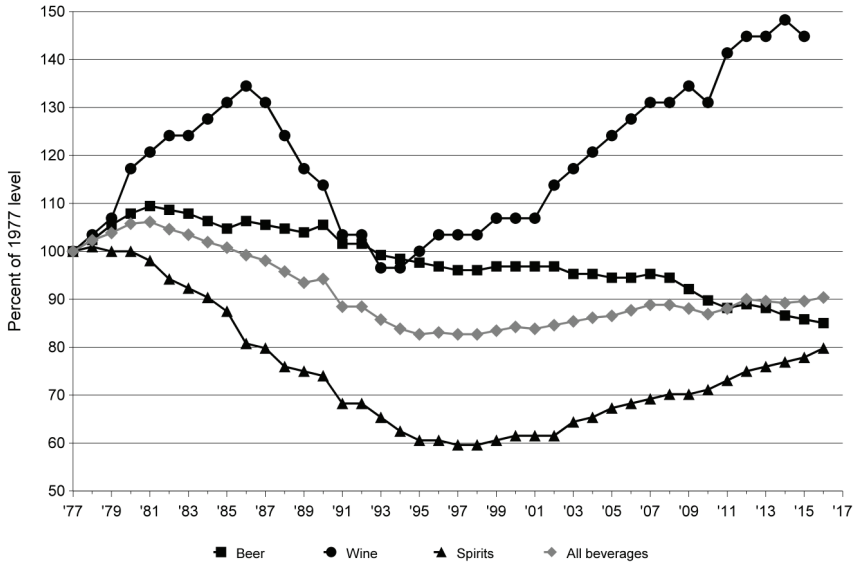


FIGURE 7-9 Percentage change in per capita ethanol consumption by beverage type, United States, 1977–2016.
NOTE: Beverage types include beer (black squares), wine (black circles), spirits (black triangles), and all beverages combined (grey diamonds).
SOURCE: Data from National Institute on Alcohol Abuse and Alcoholism, https://pubs.niaaa.nih.gov/publications/surveillance110/CONS16.pdf.

These differences in drug and alcohol-induced mortality trends could be the result of differences in the etiology of mortality from these causes. Unlike drug poisonings, which are often acute and due to overdose, most alcohol-induced deaths are chronic and are the result of many years of steady alcohol consumption. With the exception of fatal injuries caused by alcohol intoxication, drugs (particularly opioids) kill people more quickly than does alcohol. This complicates the ability to draw a clear link between cohort-based trends in the availability and consumption of alcohol among younger adults and subsequent period changes in mortality among older adults. Relative to drug poisoning, the extended period of consumption before the onset of many diseases caused by alcohol provides greater opportunity for intervention before alcohol-induced mortality occurs, as well as greater opportunity for deaths from other causes. In contrast, drug poisoning mortality may be more likely to track contemporaneous trends in the supply of particularly lethal drugs. For these reasons, although the overall trends in mortality from these causes of death differ, it is possible that these

trends are the result of common underlying vulnerabilities to drug and alcohol use within certain population groups and geographic areas.

EXPLANATIONS FOR THE RISE IN WORKING-AGE MORTALITY FROM DRUG POISONING AND ALCOHOL-INDUCED CAUSES

Scholars have debated whether the rise in drug poisonings is due to the increased availability of drugs (supply side) or the increased vulnerability of certain population groups (demand side). Scholars who support the former explanation point to the actions of legal and illegal drug suppliers and regulatory failures of government agencies, primarily the Food and Drug Administration (FDA) and Drug Enforcement Administration (DEA) (Kolodny et al., 2015). These scholars chronicle the increases in the availability of inexpensive highly addictive and lethal drugs. Scholars who point to increases in the vulnerability of population groups reference the social determinants of health that put some individuals and communities at risk of addiction and substance misuse (Dasgupta, Beletsky, and Ciccarone, 2018). These scholars chronicle an increase in socially at-risk populations that amplified the impact of supply shifts such that life expectancy in the United States began to decline during the period. Yet these are not competing explanations for the nation's overdose crisis; rather, the increase in the availability of drugs and both the long-term and increasing vulnerability of these population groups combined to create and fuel the rising trend in drug poisoning deaths. The country's drug overdose crisis represents a "perfect storm" of the flooding of the market with highly addictive yet deadly substances and underlying U.S. demand for and vulnerability to substances that temporarily numb both physical and mental pain.

Drug and alcohol addictions also create additional vulnerabilities in the population groups they impact. The addictive and destructive nature of opioids, many other drugs, and alcohol puts pressure on the social and economic fabric of families and communities, resulting in downward spirals that lead to further addiction.

Supply-Side Explanations

Prescribing Practices and the Emergence of OxyContin

On the supply side, weak government regulations and aggressive and highly effective marketing tactics on the part of the pharmaceutical industry (manufacturers, distributors, pharmacies) and pain management advocacy groups (many of which were funded by the pharmaceutical industry) and physicians sparked a massive increase in opioid prescribing in the 1990s and 2000s and the subsequent rise in prescription opioid misuse,

addiction, and overdose (see Figure 7-10; Kolodny et al., 2015). Physicians were encouraged to be more aggressive with pain management and given misleading information about the safety of opioids and their lack of addictiveness. A small group of physicians, motivated by profits, engaged in dubious schemes for prescribing large amounts of opioids or received fees to promote these drugs.

Among the most well-known culprits in the opioid overprescribing crisis is the pharmaceutical company Purdue Pharma. In 2020, Purdue reached an $8.3 billion settlement with the U.S. government and agreed to plead guilty to criminal charges that it enabled the supply of opioids "without legitimate medical purpose," conspired to defraud the United States, and violated antikickback laws in its distribution of opioids. Kickbacks included payment to health care companies and physicians to encourage opioid prescribing (Sherman, 2020). From 1996 to 2002, Purdue provided funding for more than 20,000 educational campaigns promoting the use of opioids for chronic pain in patients without cancer (a group for which opioids were

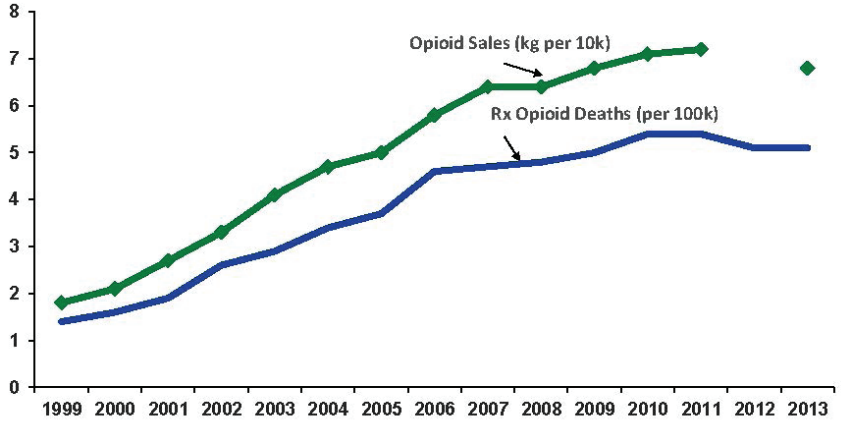


FIGURE 7-10 Prescription opioid sales and deaths, 1999–2013.
SOURCE: Baldwin, G. (2015). *Overview of the Public Health Burden of Prescription Drug and Heroin Overdoses*. Available: https://www.fda.gov/media/93249/download. Opioid sales data available from U.S. Department of Justice, Drug Enforcement Administration, Automated Reports and Consolidated Ordering System (ARCOS), https://www.deadiversion.usdoj.gov/arcos/retail_drug_summary/2013/index.html. Prescription opioid deaths data available from Centers for Disease Control and Prevention, National Center for Health Statistics, National Vital Statistics System, http://wonder.cdc.gov.

generally not indicated). In 1997, the American Academy of Pain Medicine and American Pain Society issued a joint consensus statement promoting the benefits of using opioids for chronic pain management (Haddox et al., 1997; National Academies of Sciences, Engineering, and Medicine [NASEM], 2017). These and other pain advocacy groups (many of which, as noted, were funded by Purdue and other pharmaceutical companies) contributed to opioid overprescribing by arguing that pain was the fifth vital sign and too often left untreated or poorly managed. Their representatives visited physicians' offices and medical conferences and argued that physicians were too insensitive to the effects of pain on quality of life, needed to do a better job of measuring and monitoring patients' pain, and should prescribe appropriate pain medications (Quinones, 2015). The motivation behind this movement for more aggressive treatment of pain may not have been in the best interest of patients. Prior to the mid-1990s, opioids were restricted primarily to late-stage cancer patients, palliative care, and victims of traumatic injury; however, pain advocates and pharmaceutical representatives encouraged primary care physicians to assess patients for pain along with the other vital signs and to prescribe opioids aggressively to manage a variety of chronic pain conditions, from menstrual cramps to back pain.

In 1996, Purdue introduced and began heavily marketing OxyContin—an extended-release oxycodone product. Although several other opioid products were already on the market, OxyContin is widely viewed as the product that fueled the surge in U.S. opioid addiction. U.S. Department of Justice documents show that Purdue executives and the company's owners (the Sackler family) knew that OxyContin was widely abused but lied in claiming that it was less addictive than other opioid products already on the market, intentionally misleading federal regulators, health care providers, and the public (Macy, 2019; Meier, 2018). This disinformation campaign made many physicians comfortable in prescribing the drug heavily to a wide range of patients. Purdue argued that OxyContin's new slow-release long-acting formulation not only was more effective than existing opioids but also reduced the product's ability to give users a high, and therefore its addiction potential. However, the slow-release technology worked only when the pill was consumed whole. When it was crushed and snorted or dissolved and injected, users could obtain a very intense immediate high, making OxyContin much more addictive than other prescription opioid products already on the market. Purdue also exaggerated the period of pain relief OxyContin typically provided (12 hours). Most people who used it experienced a much shorter relief period, leading them to take the pills more frequently.

When the FDA approved the use of OxyContin in 1995, the agency believed that the slow-release technology minimized the risk of addiction. Purdue (and subsequently other pharmaceutical companies) exploited the

now infamous five-sentence Porter and Jick letter, which was published in the *New England Journal of Medicine* in 1980, to convince the FDA, physicians, and the public that opioids were safe and not addictive. That letter actually stated that, among the nearly 12,000 hospitalized patients who had received at least one opioid administration in the hospital, only 4 had developed an addiction. Although Porter and Jick's study included only hospitalized patients, Purdue used the letter to state that "less than one percent of patients treated with opioids became addicted." As a result of Purdue's actions, pain specialists and other providers, especially speakers (many of whom received consulting fees from Purdue) who gave lectures to physicians about pain management, routinely cited this statistic.

In addition to misleading physicians, the FDA, and the public about OxyContin's addiction risk, Purdue used aggressive marketing tactics that were unprecedented for a Schedule II drug.[9] From 1996 to 2001, Purdue conducted more than 40 national pain management conferences; recruited and trained physicians for national speaker bureaus; compiled prescriber profiles on individual physicians to target those with already high rates of opioid prescribing; developed a lucrative bonus system for its sales representatives; distributed "patient-starter" coupons that provided patients with free prescriptions for a 7- to 30-day supply of OxyContin; and distributed numerous branded promotional items, such as tools, stuffed plush toys, and compact discs ("Get into the Swing with OxyContin," Van Zee, 2009). OxyContin sales skyrocketed from 670,000 to 6.2 million from 1997 to 2002. The global consulting firm McKinsey & Company (arguably the world's most prestigious management consulting firm) developed a plan to "turbocharge" OxyContin sales, push back against the DEA, counter emotional messages from parents whose children had overdosed, and even give Purdue's distributors a rebate for every OxyContin overdose attributable to pills they sold (Bogdanich and Forsythe, 2020; Forsythe and Bogdanich, 2019; Kristof and WuDunn, 2020).

The success of OxyContin led to the introduction of copycat extended-release medications, including Opana® ER (oxymorphone hydrochloride extended release), and several extended-release hydrocodone products (e.g., Zohydro® ER, Hysingla® ER), as well as to increased prescribing of short-release hydrocodone and oxycodone products that had been on the market long before OxyContin. By 2015, numbers of opioid prescriptions were three times higher than they had been in 1999 (from 180 to 640 morphine milligram equivalents per person) (Guy et al., 2017). Prescribing rates peaked in 2010 and have declined ever since. However, declines have not been uniform: the average number of days for which medications were

[9]Schedule II drugs are substances with a high potential for abuse but with some medicinal purpose.

prescribed continued to rise in some places, and prescribing rates remain much higher today than they were before OxyContin entered the market in 1996 (Guy et al., 2017; Schuchat, Houry, and Guy, 2017).

Purdue is hardly the only company responsible for the surge in opioid prescribing, as evidenced by the numerous city, state, and federal lawsuits (past and current) filed against opioid manufacturers, distributors, and dispensers.[10] Other major culprits include the manufacturers Mallinckrodt Pharmaceuticals (which sold more opioids in the United States than any other manufacturer during the height of the opioid crisis), Johnson & Johnson, Endo International, Teva Pharmaceuticals, and Allergan and the distributors McKesson, Cardinal Health, and AmerisourceBergen (Dwyer, 2019; Kaplan and Hoffman, 2020). Drug distributors, dispensers, and pharmacy chains (e.g., Walgreens, CVS, Rite Aid) also contributed to and profited from overprescribing through their failure to monitor and investigate suspicious opioid prescribing patterns (Cuéllar and Humphreys, 2019; Hoffman, 2020).

Despite heavy marketing efforts, some physicians remained hesitant to prescribe opioids to treat chronic pain and fearful of their addiction risk. Some unscrupulous physicians viewed the increased demand for prescription opioids as an entrepreneurial opportunity, which resulted in high-volume-prescribing pain clinics (some of which functioned as "pill mills") across the United States. At pill mills, physicians wrote prescriptions for OxyContin and other opioids, often with little diagnosis or follow-up. Several investigative books and docuseries describe how patients would line up, pay cash, and leave with prescriptions for high-dosage opioids and other drugs, which they sometimes used themselves but often sold or diverted to family and friends (Quinones, 2015; Temple, 2016; Willoughby Nason and Furst, 2020). This egregious prescribing could not have happened without the willful help of pharmaceutical distributors. In the space of just 2 years, for example, the giant pharmaceutical distributor McKesson Corporation shipped nearly 9 million opioid pills to a single pharmacy in tiny Kermit, West Virginia (population 400) (Kristof and WuDunn, 2020).

Pill mills first emerged in the most economically depressed regions of Ohio, Kentucky, West Virginia, and Florida in the late 1990s and then spread across the country (Quinones, 2015). In the late 1990s in Maine, West Virginia, eastern Kentucky, southwestern Virginia, and Alabama, hydrocodone and oxycodone (non-OxyContin) were prescribed at a rate 2.5–5 times the national average. By 2000 in these same places, OxyContin prescribing rates were 5–6 times higher than the national average (Van

[10]For a record of the various lawsuits, judgments, and settlements against pharmaceutical companies, see https://www.drugwatch.com/opioids/lawsuits.

Zee, 2009). It is no coincidence that these were the first areas of the United States to experience widespread increases in opioid misuse, diversion, and overdose and demand for SUD treatment.

Even conscientious physicians contributed to the glut of opioids on the market through opioid overprescribing after surgical procedures (Ladha et al., 2019; Neuman, Bateman, and Wunsch, 2019). In a review of the research evidence on the extent of opioid overprescribing after surgery, Neuman, Bateman, and Wunsch (2019) summarize several studies showing that U.S. physicians prescribe opioids after surgery at rates several times higher than those of their European counterparts. For example, Ladha and colleagues (2019) found that, compared with Canada or Sweden, the United States had higher average doses of opioid prescriptions for most surgical procedures. Bicket and colleagues (2017) found that 67–92 percent of U.S. patients having undergone surgery had unused opioid tablets, with the proportion of unused tablets ranging from 42 to 71 percent. Many patients store unused prescriptions improperly, often leaving them in unlocked locations such as medicine cabinets, cupboards, and wardrobes (Bicket et al., 2017; Neuman, Bateman, and Wunsch, 2019). This improper storage contributes to prescription opioid diversion through theft by relatives, friends, and strangers who enter households to provide services (e.g., repairs, cleaning, home health care) (Inciardi et al., 2007).

Collectively, the forces described above resulted in saturation of the United States with 76 billion opioid pills just between 2006 and 2012; no other country approached this level of opioid prescribing (Hingham, Horwitz, and Rich, 2019). In 2015, 97.5 million persons ages 12 and over—36.4 percent of the U.S. population (Hughes et al., 2016)—reported using prescribed pain relievers (hydrocodone, oxycodone, and morphine).

Regulatory Failure

When the FDA approved OxyContin in 1995, the drug had not been shown to be more efficacious or safe than the short-acting oxycodone that was then on the market. As noted, the claim promoted by Purdue was that OxyContin was less likely to lead to misuse and addiction because of its time-release formulation (NASEM, 2017, p. 18). In approving OxyContin, however, the agency overlooked substantial evidence against the effectiveness of this "extended-release" technology (Frydl, 2017). A recent report of the National Academies of Sciences, Engineering, and Medicine (2017, p. 264) points to limitations of the process for evaluating investigational drugs, particularly with respect to the approval of opioids:

> For example, showing that a drug has substantial evidence of efficacy does not necessarily mean that the drug is more effective than currently

> available therapies, or that the efficacy demonstrated is clinically meaningful…In addition, clinical trials sufficient to meet the FDA's efficacy standard can be conducted in a brief, highly protocolized setting and often exclude many patients who would be expected to get the drug following its approval…Clinical trials could be designed with more robust follow-up periods or be prospectively powered to ensure that well-known side effects are adequately measured. However, the FDA bases its approval decision on the data provided by the manufacturer at the time of the NDA [New Drug Application] and does not require that trials of investigational drugs be conducted with particular characteristics.

The FDA's regulatory authority continues following the initial marketing approval of a drug, and postapproval monitoring may require ongoing evaluation and timely communication with health care providers and the public. However, these actions take place against a backdrop of industry activities that promote the use of the drug to providers and patients (NASEM, 2017, pp. 364–365).

The DEA also plays an important role in regulating a large share of the country's licit drug supply, including opioids (Frydl, 2017). But the Office of the Inspector General of the U.S. Department of Justice has noted the DEA's slow response to the significant increase in the use and diversion of opioids since 2000: "DEA did not use its available resources, including its data systems and strongest administrative enforcement tools, to detect and regulate diversion effectively…[and] DEA policies and regulations did not adequately hold registrants accountable or prevent the diversion of pharmaceutical opioids" (U.S. Department of Justice [DOJ], 2019, p. i).

State licensing and monitoring boards also contributed to opioid overprescribing. In states that monitored physicians' prescribing of opioids and other Schedule II drugs, deaths due to drug poisoning were lower. Alpert and colleagues (2019) argue that Purdue viewed as a barrier to entry state requirements that physicians prescribe opioids on triplicate forms that could be used to monitor possible fraud and overprescribing. They show that OxyContin distribution was 2.5 times greater in states without versus those with this requirement, and that as a result, drug overdose deaths increased more rapidly in the former compared with the latter states.

Insurance companies also could have done more to stop opioid abuse. Public and private insurers paid for prescriptions for many years without developing adequate checks to determine whether the prescriptions were appropriate. For example, Morden and colleagues (2014) document that nearly half of disabled Medicare beneficiaries received a prescription opioid in 2010, and half of those received six or more prescriptions.

The Surge in Heroin and Fentanyl

As policy makers, state health officials, and physicians became aware of the surge in prescription opioid addiction and diversion, policies and strategies were employed to control the misuse of opioids. These measures included instituting prescribing limits, monitoring prescribing to identify excessive levels, and implementing "pill mill" laws requiring providers to submit clinical documentation from medical records to support their prescribing of opioids (Kiang et al., 2019). As a result, prescribing began declining after 2010, and prescription opioids subsequently became less available and more expressive to buy "on the street." Purdue also reformulated OxyContin into an "abuse-deterrent" formulation, but research shows that this reformulation was followed by a significant level of both residual misuse and switching to other drugs, particularly heroin (Cicero and Ellis, 2015). These forces created a "thick market" for heroin (Quinones, 2015), lowering its prices and introducing a new clientele to the drug. This new market, combined with the existing heroin client base, ushered in the second wave of the opioid overdose crisis, in which the consolidation of the heroin supply chain in Mexico and the much more widespread availability of heroin in the United States than in the past led to an increase in heroin overdose deaths.

The consolidation of the heroin supply chain in Mexico is an important part of this story. Mexican drug suppliers went from controlling 50 percent to 90 percent of the heroin market from 2005 to 2016 (Ciccarone, 2019). Quinones (2015) describes how small cells of poor rural farm boys from the tiny Mexican town of Xalisco, Nayarit, expanded their heroin business from California to small cities and towns throughout the United States in the 1990s and 2000s. Prior to 2000, U.S. heroin came primarily from four regions: southeast Asia, southwest Asia, Mexico, and Colombia. Mexico's market domination was due in part to the DEA crackdown on heroin suppliers in Colombia. The disruption of the Colombian drug cartels created an opening for Mexican suppliers to provide heroin. The heroin from Mexico was more refined and more potent than the black tar heroin that preceded it and proliferated in the Northeast and Midwest (Quinones, 2015). Heroin also became cheaper and much more widely available than at any point in recent history.

The third wave of the opioid overdose crisis began in the early 2010s, when drug suppliers and dealers began increasingly adulterating heroin and other drugs (e.g., cocaine) with fentanyl and fentanyl derivatives. Fentanyl is cheap; very potent even in small quantities (50 times more potent than heroin); and because of its potency, easy to smuggle into the United States in smaller quantities than heroin. Even when consumed in small quantities, fentanyl increases the likelihood of drug overdose compared with heroin,

resulting in higher mortality rates. In March 2015, the DEA issued a nationwide alert regarding fentanyl, noting the surging number of overdose deaths associated with its use. According to the DEA, most fentanyl entering the United States is from China (U.S. Drug Enforcement Administration [DEA], 2016) and is much cheaper and more potent than Mexican heroin (Pardo et al., 2019).

Nearly 29,000 fentanyl-related deaths occurred in 2017, more than triple the number observed in 2015, when national alarms first began to sound (NCHS, 2019a). Fentanyl deaths surpassed those involving heroin in August 2016 and have continued to climb, even as overall overdose mortality began to level off. Relative to those in other regions of the country, people who consume heroin and other narcotics in the Northeast are at greater risk of consuming a product that has been adulterated with fentanyl because of that region's proximity to shipping ports. The product may be "cut" multiple times on its way from the Northeast to Southern and Western distribution networks, thereby diminishing its potency along the way. This may explain in part why mortality associated with fentanyl shows a geographically heterogeneous pattern, concentrated in the Northeast (Monnat, 2019; Peters et al., 2020). The DEA also attributes this regional variation to the fact that black tar heroin is more popular in the Western United States, whereas white powder heroin is more popular in the East (DEA, 2016). It is easier to mix fentanyl with white powder heroin than with black tar heroin.

Attributing the rise of drug overdose deaths to misleading marketing and aggressive distribution of legal opioids and the ensuing widespread emergence of illicit opioids (heroin and fentanyl) in the United States is appealing because this explanation points to proximal factors, is conceptually plausible, and is supported by empirical evidence (and court documentation). One must ask, however, why some communities and sociodemographic groups are more vulnerable to an increase in the supply of opioids. Undoubtedly, saturation of the market with highly addictive and potent opioid painkillers was an essential spark for the massive increase in fatal drug overdoses over the past three decades. But it does not explain why rates of addiction and overdose are higher among certain population subgroups than others or why rates of overdose from other drugs, including methamphetamine, cocaine, and benzodiazepines, continue to climb.

Changes in Alcohol Supply

The rise in alcohol consumption has been linked to a relative decline in the price of alcohol; alcohol industry efforts to increase the times at which and number of places where people can consume alcohol; the development

and aggressive promotion of new alcoholic products, particularly to youth, young adults, and women; and weakening government oversight of alcohol (Freudenberg, 2014).

With the exception of the Prohibition era (1920–1933), the United States has historically had fairly moderate alcohol regulations relative to peer nations (Gruenewald, 2011). Except for raising the drinking age from 18 to 21 and enacting stricter laws regarding driving while intoxicated and the ways in which alcohol is advertised, the United States has trended toward less restrictive alcohol policies over the past several decades. As with prescription drugs, alcohol industry deregulation (e.g., relaxing days and times of sales, relaxing where alcohol can be sold, allowing home delivery and "cocktails to go") and privatization have resulted in increased availability and affordability of alcohol (Freudenberg, 2014).

Between 2007 and 2017, the number of outlets selling alcohol (including both on- and off-premise sites), grew from 528,594 to 644,647—a 22 percent increase (Nielsen, 2018). Alcohol became less expensive (in real terms) than at any time in the past 60 years (Kerr et al., 2013b). Kerr and colleagues (2013b) calculated that the cost of one drink per day of the cheapest branded spirits declined from 4.46 percent of U.S. mean per capita income in 1950 to 0.29 percent in 2011.

New alcohol products have also been developed and promoted. Beginning in the late 1990s, the alcohol industry developed and began to heavily market sweetened and flavored alcoholic beverages (FABs)—also known as "alcopops," flavored malt beverages, and "malternatives" (e.g., wine coolers, hard lemonade, alcoholic spring water)—to attract youth and women (Freudenberg, 2014). Expenditures on advertising across all FAB brands increased from $27.5 million in 2000 to $196.3 million in 2002 (Freudenberg, 2014), substantially increasing youth exposure to and consumption of these brands and products (Mosher, 2012). Members of the early 1980s birth cohort were in their late teens and early 20s during the early 2000s, so this group was beginning to drink regularly when the heaviest marketing of FABs occurred (Mosher, 2012). For a thorough review of contemporary alcohol industry marketing and lobbying practices and their association with increased alcohol consumption and alcohol-related health problems and mortality, see Freudenberg (2014).

While these supply conditions may be related to the increases in alcohol consumption that have occurred since the mid-1990s, they cannot explain why peak U.S. per capita alcohol consumption occurred during the mid-1970s to mid-1980s and was followed by a decline throughout the late 1980s and early 1990s (Haughwout and Slater, 2018). These trends raise questions about the potential role of demand.

Demand-Side Explanations

Susceptibility to substance abuse is influenced by individual/proximal factors (e.g., SES, psychological factors); community meso-level structures (e.g., family, peers, social environment); and macro-level structures (e.g., economic inequality, policies, corporate practices) (see Figure 6-1 in Chapter 6). Increases in substance-related mortality, while affecting all demographic groups and places, have been larger in some groups and places than others. Various meso- and macro-level structures have had varied impacts on different groups of people and places, making certain individuals more vulnerable to adopting harmful health behaviors and certain places more vulnerable to the infiltration of addictive opioids.

Scholars have offered a number of possible demand-related explanations for the surge in drug addiction and overdose seen over the past three decades and its particular impact on certain subpopulations and geographic areas. Some of these explanations focus on factors proximate to individuals—physical pain, mental illness, adverse childhood experiences (ACEs), and psychological distress or despair—and others on factors more structural and distal—macro-level economic and social changes. This section first provides an overview of conceptual models of addictive behaviors and then summarizes the evidence for these explanations.

Substance Use Disorders and the Underlying Causes of Drug and Alcohol Addiction

Conceptual models of addictive behaviors are useful in understanding why some individuals are more vulnerable to misusing drugs and alcohol. Underlying these models of addiction is the notion that individuals who become dependent on drugs or alcohol have lost control of their ability to use these substances appropriately. Five basic conceptual models attempt to explain addictive behaviors. These models focus on the moral, medical, psychological, sociological, and economic (rational) factors that drive those behaviors, factors that in reality often overlap (Clark, 2011).

The oldest model is the *moral model* of addiction, an archaic perspective developed by early classical theorists Cesare Beccaria and Jeremy Bentham. Under this model, an individual who becomes dependent on a substance is responsible for his or her behavior, for addiction is viewed as a rational, personal choice rooted in the morals of the individual. This choice is aimed at maximizing the individual's pleasure and can be controlled through the perception and understanding of consequences. This conscious engagement in addictive behavior makes addiction "morally wrong," and this behavior is associated with other immoral decisions and criminal activity. The

moral model thus encourages society to criminalize, regulate, and prohibit addictive behavior and to increase its consequences and costs to discourage it. This model is inconsistent with current clinical and biological thinking, and may lead to a language of addiction that stigmatizes individuals and impedes successful treatment (Fareed, 2020; Zgierska et al., 2020).

The *medical model* of addiction—the preferred model of the National Institute on Drug Abuse—defines it as a brain disease (Ozburn, Janowsky, and Crabbe, 2015). According to this model, addiction in a pathological sense stems from the genetic and neuroadaptation theory. This theory posits that addiction may be caused by certain genes that increase a person's vulnerability to addiction, or neurochemical adjustments in the brain that lead to measurable tolerance and withdrawal. The medical model therefore focuses on the impact of drugs and alcohol on certain regions of the brain and the neurocircuitry that facilitates the impulsivity and compulsivity that produce the three stages of addiction: "binge/intoxication," "withdrawal/negative affect," and "preoccupation/anticipation" (craving) (Ozburn, Janowsky, and Crabbe, 2015).

Despite substantial research on biological mechanisms, the medical model has some limitations (Koob and Volkow, 2010; Volkow and Koob, 2015). This model tends to support interventions that disrupt the brain's response to drugs and alcohol, and there have been some treatment successes. Yet despite these successes and the important evidence for genetic forces in addiction, this model fails to take into account social and cultural forces and the multiple triggering pathways that may lead to addiction, as well as the personal motivation and social support necessary for addiction recovery.

The *psychological model* focuses on addictive behaviors as a means of escape from negative emotional states caused by unmet psychological needs, implying that treatment must go beyond the addiction itself and address those needs. Unlike the moral model, the psychological model views addiction as the manifestation of motivation rather than loss of control (Khantzian, 1997). Motivation ties to self-efficacy, the belief in one's ability to stop engaging in such behavior. People who experience addiction use cognitive and behavioral self-regulatory strategies to resist cravings. Like the medical model, the psychological model does not emphasize individual choice but highlights the influence of learned reinforcement on the development of such behaviors. Applying learning theory to addiction has robust clinical implications, such as the potential for teaching strategies for reducing addictive behaviors. Still, the psychological model does not account for the social and environmental context of a person's experience with addiction.

The *sociological model* of addiction asserts that it is socially and culturally constructed through family, peers, culture, and other social influences.

Proponents of this model oppose the medicalization of social deviance, positing that it facilitates labeling of addictive behaviors based on one's social status and the agenda of social control agents. For example, someone addicted to opioids is a patient, while someone addicted to heroin is a criminal, despite the similarities between the two substances. Along with social context, the psychological context of the drug or alcohol user is salient to the process of becoming an "addict." That label becomes a core identity that furthers the performance of the associated behaviors, such that individuals behave like agents of the substance, lacking control over their own actions. Thus the sociological model suggests that effective interventions must consider the individual's social and psychological context.

The *economic or rational model* suggests that drug or alcohol addiction is an individual choice based on the level of satisfaction these substances give the user. Not only do users receive contemporary satisfaction from using drugs or alcohol but their current use raises their satisfaction for their immediate subsequent use. This reinforcing phenomenon coupled with an increasing tolerance—that is, to receive same satisfaction, the user has to use more of the substance—results in a rational addiction (Becker and Murphy, 1988).

Looking at these five models, while there is certainly a sentiment in the nation that Americans have moved away from traditional values, the evidence does not suggest that the rise in substance-related mortality is associated with a decline in morality. Indeed, some of the states with the highest levels of religiosity are also those that have experienced the greatest increases in mortality due to drug poisoning and alcohol-induced causes (Norman, 2018). Rather, the committee posits that changes in psychological needs and social context are more likely contributors to the increased vulnerability of certain U.S. subpopulations (Sudhinaraset, Wigglesworth, and Takeuchi, 2016).

In general, substances with the potential for addiction or dependence are considered to be alcohol; tobacco; and a variety of drugs, licit and illicit. However, other substances are also regularly misused, an example being hydrocarbon toxicity (e.g., glue sniffing) (Tormoehlen, Tekulve, and Nañagas, 2014). In keeping with the taxonomy of the current *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition* (DSM-5), most substance addictions that rise to the level of clinical importance meet a common set of criteria and are called SUDs. The scientific, medical, and public heath literature on SUDs overall is robust and growing. An important problem, however, is that only a few and occasional surveys—particularly the important National Survey on Drug Use and Health (NSDUH)

and the National Epidemiologic Survey on Alcohol and Related Conditions (NESARC)—address the population prevalence of these disorders.[11]

In 2015, among persons receiving treatment for substance use in the past year, 22.4 percent reported misusing prescription pain relievers (Haffajee et al., 2019; Hughes et al., 2016). It is important to note, however, that people do not immediately become addicts in adulthood when a physician prescribes opioids. National data show that nearly three-quarters of individuals ages 18–30 admitted for substance use treatment began using before age 18, and 10.2 percent began at age 11 or younger. These findings suggest that preventing initiation of substance use in childhood and adolescence is important to preventing the development of SUDs later in life (Strashny, 2014).

In 2018, an estimated 21.2 million people ages 12 or over (i.e., about 1 in 13 people in the United States) needed substance use treatment, although only about 3.7 million people received any kind of treatment (Substance Abuse and Mental Health Services Administration [SAMHSA], 2019). Medications have been shown to be effective in treating opioid use disorder and reducing the risk of dying from an overdose, yet at most 20–40 percent of people with that disorder receive treatment. Access to providers who treat opioid use disorder can vary, with deadly consequences. For example, counties with high rates of mortality due to opioid overdose have fewer primary care providers who could prescribe medications used to treat the disorder (Haffajee et al., 2019). Adoption of Medicaid expansion was associated with a 6 percent lower rate of total opioid overdose deaths compared with the rate in nonexpansion states. Counties in expansion states had an 11 percent lower rate of death involving heroin and a 10 percent lower rate of death involving synthetic opioids other than methadone compared with counties in nonexpansion states, although an 11 percent increase was observed in methadone-related overdose mortality in expansion states (Kravitz-Wirtz et al., 2020).

The Role of Physical Pain

In addition to psychological predispositions to addiction, physical pain is another potential demand-side proximal explanation for the increase in SUDs and subsequent overdose. The level of physical pain among adults in the United States is high and may be rising. According to a report of the Institute of Medicine (IOM, 2011), about 100 million adult Americans, or about 40 percent of the U.S. adult population, experience chronic pain.

[11]The most recent (and third) wave of NESARC was conducted in 2011–2012; previous waves were conducted in 2001–2002 and 2004–2005. The committee is not aware of plans to conduct a fourth wave of NESARC.

Using data from the Medical Expenditure Panel Survey, Gaskin and Richard (2012) found that in 2010, 70.3 million adults had joint pain (53.4 million had "arthritis" pain), and 22.6 million reported that this pain was severe. Millions also experience pain from injury, disease, or medical procedures. Some evidence suggests that there have been increases in physical pain over the past several decades. Case and Deaton (2015) found concurrent rising increases in reported levels of pain and declines in self-reported health and physical functioning among midlife adults (ages 45–54). Using 18 years of data from the Medical Expenditure Panel Survey, Nahin and colleagues (2019) similarly found that the proportion of adults reporting painful health conditions increased from 32.9 percent in 1997–1998 to 41.0 percent in 2013–2014. This finding may be attributable to an increase in anatomically localized physical pain syndromes per se, or to an increase in systemic conditions that have important pain manifestations.

In the most recent study of pain trends available to the committee, Zajacova, Grol-Prokopczyk, and Zimmer (forthcoming) examined the prevalence of joint, low-back, neck, migraine, and jaw/facial pain among adults ages 25–84 using the 2002–2018 National Health Interview Survey. They found a large escalation in pain prevalence among adults over this period, with overall reports of pain in at least one anatomic site increasing by 10 percent (from 49% in 2002 to 54% in 2018), representing an increase of 10.5 million adults experiencing pain. They also found that socioeconomic disparities in pain prevalence increased over this period. For example, whereas the odds of reporting any pain increased by 17 percent among those with a college degree, they increased by 40 percent among those who never attended college. Psychological distress and health behaviors were among the most important correlates of these trends.

Prior to the mid-1990s, adults with non-cancer-related pain would rarely have been prescribed opioids except for short-term needs (e.g., sickle cell crises, kidney stones, postoperative recovery). However, the prevalence of (and possible increase in) adults with painful chronic health conditions provided a new market for opioids. Among adults with severe pain, the use of strong opioids more than doubled from 11.5 percent in 2001–2002 to 24.3 percent in 2013–2014. In a recent study of adults ages 25–74 using data from the mid-1990s and early 2010s, Glei, Stokes, and Weinstein (2020) found that physical pain was linked more closely to the rise in the misuse[12] of prescription opioids relative to other drugs. Disparities in pain management experienced by Blacks and Hispanics may have blunted and protected them from the overprescribing of opioids (Mossey, 2011).

[12] "Misuse" is defined in this study as personal use in ways not prescribed or distribution of these drugs to others for whom they were not intended.

The Role of Mental Illness

Mental illnesses and SUDs are closely interrelated.[13] About 1 in 4 people with a serious mental illness (SMI) have an SUD, and about 1 in 10 people with an SUD have an SMI (National Institute on Drug Abuse [NIDA], 2018). Thus even if they are not inextricably linked, SMIs and SUDs frequently overlap. It is widely believed, with important evidence, that SMIs and SUDs each can promote the progression of the other (NIDA, 2018).

An important consideration in understanding the onset and development of mental illnesses is their early onset relative to many chronic conditions of older ages. According to the American Psychiatric Association, 50 percent of mental illnesses begin by age 14 and three-quarters by age 24 (American Psychiatric Association, 2018). Many conditions destined to become fully manifest mental illnesses may develop slowly over the first two decades of life, and they rarely appear suddenly.

It has been observed for decades that persons with mental conditions are also more likely than others to have higher rates of physical/medical conditions. This literature has confirmed, for example, higher rates of back and neck pain (Viana et al., 2018), risk of arthritis (Aguilar-Gaxiola et al., 2016), and hypertension (Stein et al., 2014) among those with chronic mental conditions. In a review of mental health surveys from 17 countries, Scott and colleagues (2016) found a variety of chronic physical conditions occurring more commonly among those with mental disorders than in control populations. These findings may be related to why people with mental illness are more likely to misuse pain relievers. Among people with mental illness, 11.2 percent reported misusing prescribed pain relievers, compared with 3 percent of those with no mental illness. People with SMI were at even greater risk, with a 15.1 percent prevalence of misuse (Hughes et al., 2016).

The high cost of psychiatric care and the related costs of mental health care insurance are important barriers to access to mental health care, resulting in substantial treatment deficiencies and high rates of unmet need (Rowan, McAlpine, and Blewett, 2013; Walker et al., 2015). A significant proportion of Americans also lack geographic access to psychiatric and other mental health services, and many U.S. counties have no psychiatric services at all (Byers et al., 2017; Kakuma et al., 2011).

The ability to relate trends in adult mental illness to other important health and functional characteristics, such as substance use, disability, employment status, and mental illness–related mortality, would be of great value. Unfortunately, ongoing population surveys and other nationwide surveillance using comprehensive indicators of adult mental illness are

[13]See also Chapter 8 for a discussion of the relationship between mental illness and suicide.

scant. The NSDUH collects data annually on substance misuse, SMI, psychological distress, and suicidal ideation. However, it does not include other commonly diagnosed mental illnesses (bipolar disorder, phobias, personality disorders, eating and gambling disorders, schizophrenia and other psychoses). Moreover, the data are cross-sectional, making it difficult to determine the direction of causality between substance use and mental health disorders. Several national surveys do include nondiagnostic indicators of mental health, such as self-reports of depressive symptoms, anxiety, depression, panic attacks, and psychological distress. Such studies with longitudinal data enable researchers to examine how changes in mental health are related to changes in substance use and suicidal behavior. However, the quality of the data is often hampered by the subjective nature of the questions and the lack of consistency in repeated measures over time. For example, a recent systematic review on this issue, covering a variety of countries, suggests that there is little evidence for substantial recent changes in rates of adult mental illness (Richter et al., 2019). Conversely, using 10 years of national health care claims data, Brignone and colleagues (2020) found increases of 37 percent, 94 percent, and 170 percent in diagnoses related to alcohol, drugs, and suicidal ideation/behavior, respectively, from 2009 to 2018. An important report on the frequency of and trends in various mental illnesses was produced for the United States for the period 1990–2016 (U.S. Burden of Disease Collaborators, 2018), but a full specification of the range of mental conditions was not available.

Adverse Childhood Experiences

ACEs include physical, sexual, and emotional abuse and parental divorce, domestic violence, incarceration, substance misuse, and mental illness. These experiences are prevalent in the U.S. population. In a study representing nearly 215,000 adults in 23 U.S. states, Merrick and colleagues (2018) found that nearly two-thirds had experienced at least one ACE, and a quarter reported three or more such experiences. Other studies using both clinic-based and national samples have found similar prevalence levels, ranging from half to 69.1 percent of the U.S. adult population (Anda et al., 2006; Brown et al., 2009; Campbell, Walker, and Egede, 2016; Choi et al., 2017; Monnat and Chandler, 2015).

Since Felitti and colleagues (1998) published their landmark ACE study (also known as the CDC-Kaiser ACE study) showing a relationship between childhood abuse and household dysfunction and the leading causes of death among U.S. adults, hundreds of studies across various samples have confirmed the relationship between ACEs and multiple adverse health outcomes across the life course (Hughes et al., 2017; Kalmakis and Chandler,

2015; Larkin, Shields, and Anda, 2012; Norman et al., 2012). Using the longitudinal data from the CDC-Kaiser ACE study, Brown and colleagues (2009) tracked 17,000 individuals who were ages 18 or older in 1995–1997 over 20 years and found that the odds of premature mortality at 20-year follow-up were significantly higher among those who had had ACEs, and that those with six or more ACEs died nearly 20 years earlier, on average, than those without ACEs. This study did not assess differences in mortality by cause. Using these same data, however, Brown and colleagues (2010) found a graded relationship between ACE score and smoking-attributable lung cancer mortality.

To the committee's knowledge, no studies have examined the relationship between ACEs and substance-related *mortality*. However, numerous studies (see systematic reviews by Hughes et al., 2017; Kalmakis and Chandler, 2015) document strong relationships between ACEs and drug and alcohol misuse, age of initiation, high-risk misuse (e.g., injection drug use), and nonfatal overdose, all of which are risk factors for fatal drug poisoning and alcohol-related death. Additional studies show strong associations between ACEs (both individually and in a dose-response relationship) and suicidal ideation and attempts in adulthood (Afifi et al., 2009; Dube et al., 2001). As a result, ACEs are now well known to be highly salient risk factors for developing mental illnesses and SUDs later in life (Anda et al., 2006; Campbell, Walker, and Egede, 2016; Choi et al., 2017; Dube et al., 2002, 2003; Loudermilk et al., 2018; Merrick et al., 2017, 2018; Stein et al., 2017). Many of these studies show strong dose-response relationships; for each additional ACE, the odds of mental illnesses and SUDs in adulthood increase. ACEs have both life-course and multigenerational effects; children with parents who misuse substances are more likely than their peers to develop mental health disorders and to misuse alcohol and drugs themselves in adulthood (Anda et al., 2002). Traumatic experiences in childhood lead to multiple changes in brain structure and function, self-regulation, and stress response that serve as pathways to risky substance use behavior throughout life (CDC, 2019a; Jones, Merrick, and Houry, 2020).

Research on temporal trends in ACE prevalence is sparse, so it is also difficult to determine whether the changes observed in working-age drug- and alcohol-related mortality can be attributed to a posited increase in ACE prevalence. Data limitations also have resulted in a paucity of research on geographic differences in the prevalence of ACEs among U.S. adults. The Behavioral Risk Factor Surveillance System (BRFSS) is the only ongoing national dataset that includes responses to questions about both ACEs and health behaviors. However, not all states include the ACE module in their annual BRFSS administration, and starting in 2015, county identifiers were no longer included in the publicly available BRFSS data.

The Role of Despair

Several scholars have appealed to an argument first made popular by Case and Deaton's (2015) research that the surge in drug poisoning in the United States stems from increased demand resulting from a rising tide of despair, particularly among less-educated working-age adults (Cherlin, 2014; Dasgupta, Beletsky, and Ciccarone, 2018; Graham and Pinto, 2019; Graham, Pinto, and Juneau, 2017; Jalal et al., 2018; Monnat, 2018, 2019; Monnat et al., 2019; Silva, 2019; Stein et al., 2017b). The term "deaths of despair" was first used by a journalist to describe Case and Deaton's findings (Khazan, 2015). Case and Deaton themselves indicate that when they proposed the term, they were choosing a label, not an explanation (Case and Deaton, 2018). The term is not a clinical diagnosis, but more a layperson's classification prompted by the fact that the rise in mortality among working-age adults was due not to the leading causes of death (i.e., heart diseases, cancer, stroke, or diabetes) but to drug use, drinking, and suicide, things that people who feel good about their lives do not tend to do. Since that article was published, some scholars have adopted the term, while others have discouraged its use because it lacks clinical justification: "despair" denotes hopelessness, a feature and a correlate of many mental health disorders but not one in itself. Beyond the label, researchers have been actively examining whether there are causal links between adverse social (e.g., economic, community, family) conditions that could result in feelings of despair and in mental health conditions and SUDs.

Examining mortality trends among Whites ages 45–54 without a college degree over the period 1999–2013, Case and Deaton (2015, 2017) show that the main causes of death driving the increase in mortality in this population were drug and alcohol poisoning, suicide, and chronic liver diseases and cirrhosis. They also provide evidence of increasing morbidity, reporting concurrent declines in self-reported health, mental health, and ability to conduct activities of daily living and increases in chronic pain and inability to work.

Case and Deaton's 2015 article resulted in massive media coverage and public attention (Cassidy, 2015; Douthat, 2015; Fox News, 2017; Krugman, 2015; Rugaber, 2017; Saslow, 2016; Tavernise, 2016), as well as commentary by scientists (Auerbach and Miller, 2018; Diez Roux, 2017; Erwin, 2017; Scutchfield and Keck, 2017). The notion that the recent rise in midlife mortality was due to increasing psychological distress among working-class Whites accorded with economic, cultural, and societal trends in the United States. In their subsequent publications (2017, 2020), Case and Deaton show that mortality and morbidity among working-age Whites without a college degree continued to climb through the late 2010s. They expand their potential explanations for these trends by describing how the

life circumstances of less-educated Whites have deteriorated over recent decades. They cite several examples, including the deterioration in wages, declining labor force participation, and declines in job quality among those without a college degree; the rise in family breakdown, including divorce, nonmarital childbearing, and single parenthood; changes in religious practices; and the decline in union representation. Many of these factors are features of largely working-class communities where manufacturing jobs disappeared long ago and where unemployment has become a permanent state. At one time, these communities, often buoyed by thriving industries and busy plants or mines, were characterized by economic and family stability; unskilled and less-educated workers could find steady work with decent benefits, and social mobility was a possibility. These same communities are now marked by disconnected families, social disorganization, and high unemployment; hopelessness and despair abound among individuals, families, and the community at large, and many youth see no future. The next section elaborates on the role of these structural changes in mortality trends.

The collapse of local economies, social institutions, and family structures experienced by working-class Whites since the 1990s appears similar to the decline experienced by their Black counterparts in the 1970s–1990s. Both declines were accompanied by lethal drug use. As alluded to earlier, the crisis among Blacks was treated primarily as a criminal justice problem, while the crisis among Whites has been treated primarily (though not exclusively) as a public health crisis—a contrast often cited as an example of systemic racism (see Chapter 11). These distinctions aside, both crises may have been fueled by despair brought on by changing economic, social, and family conditions that disproportionately impacted individuals without a college degree (Silva, 2019; Wilson, 1987).

Theoretically, "despair" or other forms of psychological distress may help to explain substance-related deaths because they reflect behaviors of individuals who are potentially depressed, distressed, and without hope for the future (Baines, Jones, and Christiansen, 2016; McLean, 2016). Of course, how to define and measure despair in research is a key challenge. A formal definition of "despair" as a noun is "the complete loss or absence of hope" or "a cause of hopelessness"; the verb "to despair" means "to lose or be without hope," and "to lose all hope or confidence" (Merriam-Webster, 2020; Lexico.com, 2020). Recent research has attempted to examine trends in psychological distress and despair-related behavior. Here the challenge of how to define "despair" is important because there are no well-developed scales or validated measures of despair or hopelessness (Goldman, Glei, and Weinstein, 2018; Muennig et al., 2018). As a result, research examining trends in despair has focused on measures of negative emotions, including sadness, hopelessness, worthlessness, and depression, and positive

emotions, such as trust, happiness, fulfillment, optimism, and life satisfaction (Gaydosh et al., 2019; Goldman, Glei, and Weinstein, 2018; Graham and Pinto, 2019; Graham, Pinto, and Juneau, 2017; Graham, Laffan, and Pinto, 2018; Muennig et al., 2018).

In the most comprehensive roadmap for examining the relationship between despair and health outcomes the committee could find, Shanahan and colleagues (2019, p. 855) propose that despair is a multidimensional concept manifesting in cognitive, emotional, behavioral, and biological domains, and that it goes beyond the individual to arise in and spread through social contexts and communities. According to these authors, *cognitive despair* includes "thoughts indicating defeat, hopelessness, guilt, worthlessness, learned helplessness, pessimism, and limited positive expectations for the future." *Emotional despair* includes "feelings of excessive sadness, irritability, hostility, loneliness, anhedonia, and apathy." Behavioral despair involves maladaptive attempts to cope with distress and consists of "risky, reckless, and unhealthy acts that are self-destructive and reflect limited consideration of the future (e.g., high-risk sexual behaviors, gambling, self-harm, reckless driving, excessive spending, criminal activity, smoking, substance use, low physical activity)." Finally, *biological despair* occurs when "the body's stress-reactive systems no longer function homeostatically and show signs of dysregulation or depletion, which constitutes a biological correlate of, and sometimes a basis for, cognitive, emotional, and behavioral despair."

The committee could find no study examining empirically the relationship between all of these domains of despair and substance use or drug-related mortality, or investigating the causal relationship between *changes* in psychological well-being and *changes* in substance use or drug-related mortality. However, there is ample empirical support for the hypothesis that proxies for despair (e.g., hopelessness, sadness, worry) are connected to drug use. For example, in a study designed to validate the relationship between various personality scales and substance use, Woicik and colleagues (2009) found that the Beck Hopelessness Scale (Beck et al., 1974) was significantly associated with drug use among adults. In a national study of college students, self-reported feelings of hopelessness, sadness, and depression were associated with significantly greater odds of nonmedical prescription opioid use (Zullig and Divin, 2012). In a qualitative study conducted in McKeesport, Pennsylvania, respondents referenced the hopelessness of the area and its lack of opportunity as drivers of the use of heroin (McLean, 2016). Among a sample of adult patients in a large emergency department in Flint, Michigan, Bohnert and colleagues (2018) found that 26 percent of patients with any history of overdose reported that they wanted to die or did not care about risks. And in research based on well-being metrics in the Gallup Healthways survey, Graham and Pinto (2019) found a strong

association among lack of hope; high levels of worry; and rising rates of drug, alcohol, and suicide mortality among lower-educated Whites.

Several studies, moreover, have found evidence of worsening psychological health among U.S. working-age adults. Using self-report data from the 1993–2019 BRFSS surveys, Blanchflower and Oswald (2020) found that the proportion of the U.S. population in extreme distress (measured as reporting major mental and emotional problems in all 30 of the past 30 days) rose from 3.6 percent in 1993 to 6.4 percent in 2019. Among low-educated middle-age Whites, the percentage more than doubled, from 4.8 percent to 11.5 percent.

Gaydosh and colleagues (2019) used data from the National Longitudinal Study of Adolescent to Adult Health (Add Health) to examine trends in a young working-age cohort (ages 32–42) who had been followed from adolescence in 1994 to the beginning of midlife in 2017. They found that developmental patterns of depressive symptoms, suicidal ideation, heavy drinking, and drug use were generally similar across all racial/ethnic, educational, and geographic subgroups, with recent rises in these adverse outcomes beyond age 30 (which the cohort reached in approximately 2010). These findings suggest that all subgroups of younger cohorts are experiencing some measure of psychological distress, and that distress appears to be arising earlier in the life course.

Goldman, Glei, and Weinstein (2018) examined changes in despair-related feelings and health among Whites from the mid-1990s to early 2010s in the Midlife in the United States (MIDUS) Study, focusing in particular on socioeconomic subgroups (they lacked sufficient data to stratify their results by race). They found a decline in psychological health in this population over the period, a decline that was steeper among those of lower SES. Given the broad age range in MIDUS (25–74), they were able to document similar declines in psychological health across age groups, including those ages 30, 40, 50, and 70. They concluded that trends of worsening psychological health are a broad-based phenomenon. In commenting on this analysis, Cherlin (2018) expresses some doubt about whether the findings support the idea of growing *despair* in the United States. He notes that among low-SES Whites, the greatest declines in psychological health occurred for indicators of "positive affect" (feeling cheerful, in good spirits, extremely happy, calm and peaceful, satisfied, full of life, life satisfaction). "Negative affect" (so sad nothing cheers you up, nervous, restlessness, hopeless, everything was an effort, worthless) showed smaller differences by SES. Moreover, scales of psychological well-being, which Cherlin argued were better measures of despair, showed weak and insignificant SES gradients. Overall, Cherlin suggests that a rising tide of despair is probably an overstatement based on the MIDUS data, concluding that trends in generic happiness, sadness, and life satisfaction differed by SES more so

than trends in despair-related psychological and social well-being. Nonetheless, he acknowledges that even if a "rising tide of despair may be an overstatement," the concentration of declining psychological health among individuals of lower SES is troubling (Cherlin, 2018, p. 7177).

Muennig and colleagues (2018) make the case for a longer-term trend of worsening psychological health in the United States since the early 1980s that spanned demographic groups. Using data from the General Social Survey 1983–2012, the authors examined trends in measures of psychological well-being in the United States, including self-reported happiness and trust in others, whether people tended to be fair, whether parents had a better standard of living, and frequency of sex. Compared with similar data from Australia on the happiness and trust measures, they found a greater decline in overall well-being in the United States, especially toward the late 1980s. They similarly noted declines in subjective ratings of physical and mental health from the BRFSS. Based on their exploratory analysis and the prevailing literature, they concluded that there has been a long-term trend of increasing mistrust and loneliness and worsening mental and physical health across all age, racial/ethnic, and SES groups (Muennig et al., 2018). While Muennig and colleagues discount the argument that *recent* changes in despair are related to the *recent* rise in midlife mortality, they do provide evidence of longer-term trends in both declining health and worsening psychological well-being in the United States, suggesting a potential long-term connection between such trends (consistent with arguments made by Case and Deaton, 2017, 2020).

In a special issue of *AJPH Rural Health* titled *The Epidemic of Despair among White Americans* (Stein et al., 2017b), researchers consider differences in drug, alcohol, and suicide deaths by metro status to determine whether these deaths were primarily a rural phenomenon linked to the despair hypothesis. They report that increased death rates from 1999 to 2015 were largely among White populations outside of large urban areas and that most increases were attributable to suicide, accidental poisoning, and liver disease. Although the study design was not causal, they conclude that the rise in mortality in these nonurban areas was caused primarily by harmful coping behaviors related to underlying social and economic factors in these communities, consistent with the despair hypothesis of Case and Deaton. As noted earlier in this chapter, drug poisoning mortality rates have risen in metro and nonmetro areas alike and across all racial/ethnic groups (to varying degrees) since the early 1990s.

There has been pushback against the argument that despair is a strong explanation for the rise in working-age mortality and against grouping drug- and alcohol-related and suicide deaths in a composite "despair" classification. Major critiques include, first, that most of the increase in working-age mortality since the 1980s was due to drug poisoning, with

suicide and alcohol-related causes contributing negligibly to either the increases for most working-age groups (Masters, Tilstra, and Simon, 2017) or the increases in educational disparities in life expectancy (Geronimus et al., 2019). A second critique is that increases in the three causes of death (drugs, alcohol, and suicide) have varied by time, geography, and demographic group, raising skepticism that they could share a single underlying cause such as despair. Third, many of the hypothesized social forces (discussed in the next section) that would lead to increases in despair predated observed mortality rate increases by several decades (Ruhm, 2021). Finally, non-Whites (particularly Blacks) in the United States have historically had much more reason than Whites to experience despair (because of economic disadvantage and systemic racism), yet racial/ethnic minorities fare better on despair-related measures and experienced comparatively smaller increases in drug poisoning during the period of observation and no or only very small increases in alcohol-induced deaths and suicides (Diez Roux, 2017; Ruhm, 2021).

Various scholars have examined data on mortality trends over time similar to the data analyzed by Case and Deaton, but taking a broader view by expanding the timeframe; the ages of death, to include both younger (25–45) and older (45–65) ages; and the causes of death, and by examining trends by gender, race, ethnicity, and education (Geronimus et al., 2019; Ho, 2017; Masters, Tilstra, and Simon, 2017, 2018; Stein et al., 2017b). Masters, Tilstra, and Simon (2017), for example, examined trends in death rates during 1980–2013 for White men and women ages 45–54 and found that recent increases in extrinsic mortality were driven by rapid increases in drug-related mortality, whereas the contributions of chronic liver disease and suicide to mortality levels had been fairly stable for the past 30 years. They also found important gender differences in which women had experienced greater increases in recent mortality compared with men. They argue that these findings are inconsistent with the despair argument as the explanation for rising White midlife mortality. In a 2018 study, these authors examined mortality rates among younger and middle-age Whites and decomposed trends into period- and cohort-based variation (Masters, Tilstra, and Simon, 2018). They document the rise in drug-related deaths for younger- and middle-age Whites as a *period-based* phenomenon consistent with increases in opioid exposure rather than a rising tide of despair among more recent birth cohorts.

It is important to note, however, that Masters and colleagues did not disaggregate trends by educational attainment, which would be essential for undermining Case and Deaton's cohort thesis. As Case and Deaton (2017, 2020) show, nearly all of the increase in drug poisoning over the prior three decades was among those without a 4-year college degree. While the rates increased slightly among those with a bachelor's degree, these increases

pale in comparison with the surge in drug overdoses and other "deaths of despair" among Whites without a 4-year college degree. It is among the less-educated group of Whites that Case and Deaton (2020) show that the risk of dying from drugs, alcohol, and suicide increased with each subsequent birth cohort. For example, they found that among those ages 45 without a bachelor's degree, the birth cohort of 1960 faced a risk 50 percent higher than that of the cohort born in 1950, and the cohort of 1970 faced a risk more than twice as high. At any given age, later birth cohorts had higher drug mortality rates than their earlier counterparts. It is only among the more highly educated (those with a bachelor's degree) that Case and Deaton found no or only very small cohort effects (with each cohort dying along the same age profile). They found a similar lack of a cohort pattern among Blacks both with and without a 4-year college degree.

Geronimus and colleagues (2019) examined years of life lost by sex and education among Whites and Blacks ages 25–84 from 1990 to 2015. They found that drug overdoses, but not suicides or alcohol-related deaths, contributed substantially to growing educational inequities in life expectancy among White males and, to a lesser extent, White females. As a result, they caution against combining drug, alcohol, and suicide deaths into a composite despair-related category and suggest that the popularization of the despair hypothesis threatens to divert attention from ongoing racial/ethnic health inequities, as well as from other causes of death that have contributed to widening educational disparities, including cardiovascular diseases, cancers, and other internal causes.

In sum, researchers have used "deaths of despair" both as a blanket term to categorize deaths from drugs, alcohol, and suicide and as a potential explanation for the trends in substance-related mortality presented in this chapter, as well as the trends in suicide presented in the next chapter. Researchers disagree on the merits of both of these uses of the concept. The hopelessness signified by despair is a feature of depression and other affective disorders but is not itself a formal mental health diagnosis. There are also challenges with measuring despair and establishing causality. Despair is the result of changes in one's long-term outlook on life that go beyond fluctuations in employment, wages, and other economic indicators. It is also about hope and expectations, about perceived negative changes in the character and nature of communities that impact all social institutions and erode individuals' outlook for themselves and their children (Blacksher, 2018; Marsh, 1987; Silva, 2019). While the committee could find no causal studies on the effects of changing psychological health on U.S. substance use and mortality trends, substantial research shows that psychological health has worsened among U.S. working-age adults. There is also empirical evidence that proxies for despair, such as hopelessness, sadness, and worry, are associated with substance use. Ultimately, measuring despair

and determining causality remain key challenges for understanding the true role of despair in recent mortality trends. Qualitative research, which provides compelling evidence for the role of increasing despair in substance misuse and overdose, can offer insights for demographers, economists, and epidemiologists who aim to develop and test strong measures of despair. Furthermore, there is a need to integrate the despair hypothesis with the supply-side story. For example, to what extent did the increase in despair "prepare the field" so that the introduction of potent and highly addictive opioids could take root? Despair may be an important part of a "perfect storm" of conditions under which drug overdose deaths rose massively in the United States during the period encompassed by this study.

Macro-Level Economic and Social Change

Macro-level economic and social changes have been posited as being among the upstream factors that have given rise to despair among working-age adults without a college degree. Slow, long-term structural changes and stressors to the U.S. economy, along with unexpected shocks (e.g., the Great Recession), have had differential effects on population subgroups and geographic areas. These trends may explain in part the geographic patterns in drug poisoning mortality discussed in Chapter 4, as well as those affecting other health outcomes discussed later in this report.

Macro-level economic trends and policy changes have resulted in prosperity in some places (e.g., high-tech and finance-dominant urban hubs) and decimation in others (Appalachia, the former Industrial Belt). The distribution of industry and occupations is uneven across the country, with some communities more vulnerable than others to particular types of downturns. In particular, industries that were traditionally the source of high-wage jobs for non-college-educated adults have been unable to sustain those jobs. Competition from lower-wage workers abroad, the introduction of labor-saving technologies at home, and decreased demand for products and services (e.g., tobacco, domestic steel) have lowered the demand for high-wage blue collar jobs (Brown and Schafft, 2018; Lichter and Schafft, 2016). What were once well-paying production jobs (mining, manufacturing) disappeared in the industrial heartland, while high-wage, high-skill service-, finance-, and technology-based employment became concentrated in a small handful of urban cores (Bailey, Jensen, and Ransom, 2014; Brown and Swanson, 2003; Lobao, 2014; Peters, 2013; Smith and Tickamyer, 2011). The declines in employment opportunities and job quality led to an outmigration of the "best and brightest" young adults from those communities seeking opportunities elsewhere (Burton et al., 2013; Carr and Kefalas, 2009; Peters, 2012; Slack, 2014). Once-vibrant communities were then left with a disproportionate share of low-wage, low-skill, and often less healthy

(or disabled) workers who found themselves with limited opportunities in the midst of closed plants and mines and empty retail establishments. The end result has been to intensify the disproportionate geographic clustering of multigenerational economic distress in many parts of the United States.

The geographic distribution of economic decline, the loss of manufacturing and mining jobs, the decline in wages for blue collar workers, and the rise in poverty in some communities all correspond to the recent surge in drug deaths (Iceland and Hernandez, 2017; Saez and Zucman, 2016; Thiede, Kim, and Valasik, 2018) and other working-age deaths in geographic areas and populations discussed in Chapter 4. The collapse of local economies can contribute to collective frustration and hopelessness, lower tax bases, community disinvestment, infrastructural decay, family disintegration, crime, and substance misuse (Brown and Swanson, 2003; Carr and Kefalas, 2009; McLean, 2016; Sampson and Groves, 1989; Smith and Tickamyer, 2011).

Moreover, rising economic distress has intersected with rising family distress and marital dissolution and long-term demographic trends of lower marriage rates, increasing single-parent families, and increasing multiple-partner fertility (Burton et al., 2013; Child Trends DataBank, 2015). Collectively, these factors may mean that growing shares of the U.S. population are feeling isolated, disconnected, unstable, and without purpose or meaning in their lives.

While the direction of causality is debatable, substantial literature shows strong associations between economic distress and poor mental health and substance misuse (Frasquilho et al., 2016; Galea, Ahern, and Vlahov, 2003; Hempstead and Phillips, 2015; Kaplan et al., 2015; Kerr et al., 2017; Monnat, 2018; Pierce and Schott, 2020). The qualitative research has been especially strong in this regard. For example, sociologist Victor Tan Chen (2015) interviewed laid-off automotive workers after the General Motors and Ford plant closures of the Great Recession. After losing their well-paying jobs on the assembly lines, which many had held for years, they found themselves in an unfriendly economy that favored high education and connections. Interviewees expressed feelings of failure, apathy, despair, and self-blame, and many had turned to substances to cope. Similarly, in her study of people with heroin addiction in the deteriorating mill city of McKeesport, Pennsylvania, McLean (2016) concluded from her interviews that deindustrialization, the lack of employment opportunities, and the outmigration of businesses and people had created an atmosphere that was vulnerable to the influx of heroin. Her interviewees cited feelings of hopelessness and social isolation related to the lack of economic activity in their communities as a motivation for their drug use (McLean, 2016). Drawing on fieldwork and in-depth interviews, Jennifer Silva explored how economic decline was experienced by working-class Whites, Blacks, and

Hispanics living in rural Pennsylvania. In a presentation to the committee in October 2019, she described how—in a context in which family ties are fragile, opportunities for mobility seem scarce, and social safety nets have diminished—her respondents often had turned to drugs, alcohol, and even food to cope with life's disappointments, the lack of economic opportunity, and hopelessness about the future.

Several quantitative studies have used objective measures of economic and social decline as indicators of place-level despair to examine associations between economic conditions and drug fatality rates. These studies have generally examined short- or medium-term economic change (i.e., since the early-2000s or Great Recession effects). Analyzing county-level mortality data for 1999–2014 and emergency department utilization data for 2000–2013 for 20 states, Hollingsworth, Ruhm, and Simon (2017) found that as the county unemployment rate increased by 1 percentage point, the opioid death rate rose by 3.6 percent, and emergency department visits for opioid overdose increased by 7.0 percent. Krueger (2017) found that, compared with working men, working-age men not in the labor force experienced notably lower levels of emotional well-being, derived relatively little meaning from their daily activities, and were more likely to feel pain and take pain medication daily (Ahmedani et al., 2017). Taking advantage of differential exposures to trade liberalization resulting from Congress's granting of Permanent Normal Trade Relations (PNTR) status to China in 2000, Pierce and Schott (2016) found an increase in mortality due to drug poisoning, alcohol, and suicide. Shifting a county from the 25th to the 75th percentile of exposure to PNTR was associated with an increase in the drug poisoning rate of 2 to 3 deaths per 100,000 population each year after the policy was instituted, a significant share of the average mortality from drug overdoses during the period 1999–2013. This increase in drug-related mortality was observed across a large portion of the working-age population (most age groups between 20 and 54). However, the association was observed only among Whites and not other racial/ethnic groups.

Using data for 1999–2016 from 112 counties in 30 commuting zones primarily in the South and Midwest, Venkataramani and colleagues (2020) found that automotive plant closings were associated with opioid mortality rates. Five years after a plant closure, opioid mortality rates had increased 85 percent more in exposed counties than in similar counties that did not experience a plant closure. The association was largest among working-age White males. Betz and Jones (2018) present evidence that growth in industries more likely to hire low-skilled workers was protective of overdose deaths, particularly for rural White males. However, they also found that the economic improvements in low-skill industries appeared to protect Blacks and women against opioid overdoses.

Very little research concomitantly examines the roles of both economic

factors and family composition in place-level variation in drug mortality. Using cross-sectional county-level data, Monnat (2018) found that various measures of economic *and* family distress (including rates of poverty, unemployment, disability, low educational attainment, public assistance, divorce/separation, and single-parent families) all were associated with higher drug mortality rates, after controlling for racial/ethnic and age composition, metropolitan status, state-level fixed effects, and opioid prescribing rates (Monnat, 2018, 2019; Monnat et al., 2019).

Other research suggests that the relationship between drug overdose rates and economic decline may vary depending on the specific drug being considered. Monnat and colleagues (2019) found high rates of prescription opioid overdoses and overdoses involving both prescription and synthetic opioids to be clustered in more economically disadvantaged counties with larger concentrations of service industry workers. Counties with high rates of heroin overdoses were more urban, had larger concentrations of professional workers, and were less economically disadvantaged. Peters and colleagues (2020) examined drug overdose rates for specific opioids in 2002–2004, 2008–2012, and 2014–2016. They identified three distinct opioid epidemics (prescription opioids, heroin, and prescription–synthetic opioid mixtures) and one syndemic[14] involving multiple opioids and other drugs. They found that counties with prescription-related epidemics had been "left behind" in the economic restructuring that occurred during the 1970s and 1980s. These communities were less populated and more remote, were older and mostly White, had a history of substance use, and were former farm and factory communities that had been in decline for several decades. Overdoses in these places exemplify the "deaths of despair" narrative. By contrast, counties with high rates of heroin overdose and those classified as "syndemic" counties tended to be more urban, connected to interstates, more racially diverse, and in general more economically secure. However, the counties with the highest drug overdose rates tended to be characterized by a dual economy in which some workers had good high-skilled and decent-paying jobs, and others had low-skilled and low-paying jobs. Blue collar employment had been declining in these counties since the 1980s. The authors conclude that the overdose crisis in these largely urban areas followed the path of previous drug epidemics, affecting the disadvantaged subpopulation that had been left behind rather than the entire community.

[14] "A syndemic, or synergistic epidemic, is more than a convenient portmanteau or a synonym for comorbidity. The hallmark of a syndemic is the presence of two or more disease states that adversely interact with each other, negatively affecting the mutual course of each disease trajectory, enhancing vulnerability, and which are made more deleterious by experienced inequities" (*The Lancet*, 2017).

Whereas the studies summarized above examined place-level objective measures of economic change and distress, Glei and Weinstein (2019) show that *subjective* measures of economic distress, such as financial strain, perceived intergenerational financial disadvantage, and current work uncertainty, are better predictors of drug misuse. They conclude that the rise in drug abuse among working-age adults may relate to perceived economic distress that is not captured by standard objective measures. This conclusion is consistent with one drawn by Jennifer Silva in her presentation to the committee, that "people's experiences of the world, whether they make sense, can have an impact on their lives because they feel their stories as true." It is also consistent with the findings of a recent cohort study by Muller and colleagues (2020). Using data from the High School and Beyond study for 11,680 males who were in high school during the 1980s and 2015 mortality data from the National Death Index and the Social Security Death Index, the authors found higher rates of suicide and drug poisoning among men who had planned to work in occupations that declined during the 1980s and 1990s. They conclude that men whose occupational expectations were unmet because of labor market declines were at higher risk of death from suicide or drug poisoning relative to men with different occupational expectations.

Some of the studies discussed above found drug-related mortality effects of economic decline/distress for Whites but not Blacks or Hispanics (Hollingsworth, Ruhm, and Simon, 2017; Pierce and Schott, 2020). This finding might call into question the explanatory power of economic decline for drug mortality trends, given that Blacks and Hispanics have long faced more precarious economic conditions relative to Whites. Alternatively, it may suggest that Whites have been less resilient in the face of the economic shifts of the past several decades, or that Blacks and Hispanics cope differently with precarious economic circumstances relative to Whites. Or it may suggest that there is a floor effect on the impact of economic decline, with Blacks and Hispanics having seen the market for their blue collar workforce decline decades earlier (Assari, 2016; Blacksher, 2019; Cherlin, 2019).

Other research has found no or only limited evidence of the relationship between short- or medium-term economic decline and drug overdoses. Currie, Jin, and Schnell (2019) concluded that the relationship between employment and opioid prescribing rates is weak and that trends in employment do not explain the rise in opioid prescribing. Dow and colleagues (2019) found no association between the minimum wage and the Earned Income Tax Credit and drug mortality. Ruhm (2018b) found that, after controlling for various demographic and geographic variables, changes in economic conditions (including changes in the unemployment rate and import exposure) explain less than one-tenth of the observed

increase in drug deaths occurring between 1999 and 2015, and even less of the growth in opioid overdose rates. In a response to Ruhm's critique, however, Case and Deaton (2018) note that Ruhm looked only at medium-term rather than long-term changes in economic conditions. Subsequently, examining the impact of medium-term economic changes on drug mortality and the correlation between county-level economic downturns and drug mortality, Ruhm (2019) found that drug mortality rates did increase more in counties experiencing relative economic decline but that this relationship was weak (explaining less than one-tenth of the rise in drug mortality rates between 1999 and 2015). Instead, he attributes most of the drug mortality increase to the "drug environment" (i.e., the cost, supply, and regulation of drugs).

Not all studies of economic change and drug mortality focus on the magnitude of the effect, although when they do, they find that the impact of short- or mid-run economic change does not explain a large share of the overall increase in drug overdose deaths. However, it is important to point out that none of these studies or the trends presented in this report explicitly test the hypothesis that *long-run* (multidecade) declines in economic, family, and social conditions contributed to the increase in drug, alcohol, and suicide mortality among working-age adults without a college degree, whether that be through the pathway of despair or some other intervening mechanism. The cumulative effects of these multidimensional long-term exposures to adverse conditions remain unexplored.

SUMMARY

Collectively, drugs and alcohol were responsible for more than 1.3 million deaths among the U.S. working-age (ages 25–64) population between 1990 and 2018. These substance-related deaths were major contributors to the rise in working-age mortality, and as of this writing, they are not abating. Drug poisoning deaths have been rising for almost three decades, primarily among Whites but also among Blacks and Hispanics. Alcohol-induced deaths also increased among Whites during the entire study period, and while alcohol-induced deaths declined among Blacks and Hispanics throughout the 1990s and early 2000s, those declines leveled off during the late 2000s and shifted to increases in the 2010s.

The rise in drug poisoning deaths has been well studied, and that research has yielded some plausible explanations for this phenomenon. The trends in alcohol-related deaths have not been studied as extensively; however, the factors that influence both trends are similar. The increased availability of drugs and alcohol (i.e., changes on the supply side) and the

high and increased vulnerability of subpopulations (i.e., the demand side) combined to create and fuel the rising trend in drug and alcohol deaths.

On the supply side, actions in the 1980s and 1990s by the pharmaceutical industry (manufacturers, distributors, pharmacies), pain management advocacy groups (often funded by pharmaceutical companies), and physicians (encouraged by pain management advocates and pharmaceutical companies), combined with weak government regulations, sparked a massive increase in opioid prescribing and the subsequent rise in prescription opioid misuse, addiction, and overdose. Pharmaceutical companies, led by Purdue and its multibillion dollar blockbuster drug OxyContin, along with distributors, pharmacies, pill mills, and some physicians, saturated the United States with prescription opioids.

As policy makers, state health officials, and physicians began to recognize the dangers of opioids and prescribing subsequently declined, prescription opioids became less available and more expensive. As a result, people who had become addicted to or dependent on them (and people with existing heroin addictions) turned increasingly to heroin. This transition introduced a new clientele and created a "thick market" for heroin, lowering its prices, and ushering in the second wave of the U.S. addiction and overdose crisis. The third wave began in the early 2010s with the infiltration of fentanyl into the U.S. drug supply. Fentanyl deaths surpassed those involving heroin in 2016 and continued to climb, even as overall overdose mortality began to level off.

As with prescription drugs, alcohol industry deregulation (e.g., relaxing days and times of sales, relaxing where alcohol can be sold, allowing home delivery and "cocktails to go") and privatization have resulted in increased availability and affordability of alcohol in the United States over the past few decades. Yet while these supply conditions may be related to the increased consumption seen since the mid-1990s, they cannot explain why peak U.S. per capita alcohol consumption occurred during the mid-1970s to mid-1980s and then declined throughout the late 1980s and early 1990s.

Demand-related explanations for the surge in substance use and overdose over the past three decades focus on why certain subpopulations and geographic areas appear to be more vulnerable than others to increased exposure to opioids and other drugs. These explanations include those that are both proximate to individuals (physical pain, mental illness, ACEs, psychological distress or despair) and those that are more structural and distal (macro-level economic, family, and social changes).

Millions of Americans experience chronic pain, and some evidence also suggests that there may have been increases in physical pain over the past several decades. Prior to the mid-1990s, adults with non-cancer-related pain would rarely have been prescribed opioids for long-term use. However, high

and possibly increasing levels of physical pain have created new markets for these drugs.

Mental illnesses and SUDs are closely intertwined. However, ongoing population surveys and other nationwide surveillance on comprehensive indicators of adult mental illness are scant, making it difficult to relate trends in those conditions to other important health and functional characteristics, such as substance use, disability, employment status, and mental illness–related mortality. Similarly, numerous studies document strong relationships between ACEs and drug and alcohol misuse, age of initiation, high-risk misuse (e.g., injection drug use), and nonfatal overdose, all of which are risk factors for fatal drug poisoning and alcohol-related death. Research on temporal trends in the prevalence of ACEs is sparse, however, making it is difficult to determine whether changes in the prevalence of these experiences are related to the changes observed in working-age drug- and alcohol-related mortality rates.

"Despair" has been among the more controversial potential explanations for the rise in substance-related deaths. Despair signifies hopelessness, which is a feature of depression and other affective disorders but is not itself a formal mental health diagnosis. The notion that the past 30-year rise in working-age mortality is due in part to increasing psychological distress among working-age adults with lower education is appealing because it accords with long-term economic, family, and social changes that have increased disconnection from the people, activities, and institutions that provide support and give people purpose and meaning. While the committee could find no causal studies on the effects of changing psychological health on U.S. substance use and mortality trends, there is ample empirical support for the hypothesis that psychological health has been worsening among U.S. working-age adults and that proxies for despair (e.g., hopelessness, sadness, worry) are connected to substance use. Ultimately, measuring despair and determining causality remain key challenges for understanding the true role of despair in recent mortality trends. Qualitative research, which provides compelling evidence for the role of increasing despair in substance use and overdose, can offer insights for demographers, economists, and epidemiologists who seek to develop and test strong measures of despair.

Finally, looking at more distal demand factors, one must consider societal factors that have made some subpopulations more vulnerable than others to the increased availability of substances. Existing research has drawn mixed conclusions about the causal relationship between objective economic factors (particularly in relation to area-level short-term changes in such economic outcomes as poverty and unemployment) and substance-related mortality. Research also shows significant relationships between subjective economic distress, unmet expectations, and drug use. It is clear as

well that economic well-being has declined among individuals without a college degree over the past several decades. The decline and transformation of industries that once provided good jobs for adults with only a high school education have resulted in the erosion of the character and nature of communities that depended on those industries. This economic decline has occurred concomitantly with declining marriage rates, increases in single-parent families, declining social safety nets, and increased disconnection from social institutions. Ultimately, there is strong observational evidence that the contexts of everyday lives and the decline in opportunities for adults without a college degree contributed to the rise in drug poisoning and alcohol-related deaths.

IMPLICATIONS FOR RESEARCH AND POLICY

Several reports and commissions have examined and offered recommendations related to the U.S. opioid crisis and the broader addiction and overdose crisis in the United States. Examples include the President's Commission on Combating Drug Addiction and the Opioid Crisis (Christie et al., 2017); *Pain in the Nation: The Drug, Alcohol and Suicide Crises and the Need for a National Resilience Strategy* (Segal et al., 2017); *Framing Opioid Prescribing Guidelines for Acute Pain: Developing the Evidence* (NASEM, 2020a); *Pain Management and the Opioid Epidemic: Balancing Societal and Individual Benefits and Risks of Prescription Opioid Use* (NASEM, 2017); and *Improving the Quality of Health Care for Mental and Substance-Use Conditions* (IOM, 2006). These reports offer detailed recommendations for programs and interventions to address the problem, and readers are encouraged to access them.

The committee's recommendations differ from those prior recommendations in that they do not focus on specific policy and practice strategies. Rather, the committee believes broad efforts are needed to address all components of the U.S. addiction and overdose crisis, on both the supply and demand sides. Such efforts would include strengthening regulatory control and monitoring of the development, marketing, distribution, and dispensing of prescription drugs; developing stronger standards, procedures, and sanctions within the pharmaceutical industry for the surveillance and prevention of activities that could result in misuse, addiction, or other harms among users of its products (for although such processes may exist on paper, they were clearly not effective with regard to the opioid epidemic); investing in programs that emphasize alternatives to arrest and incarceration and encourage entry into SUD treatment; expanding access to, improving the quality of, and learning more about the effectiveness of mental health counseling and substance use prevention, treatment, recovery, and harm

reduction programs; and revitalizing communities that have been hardest hit by the addiction and overdose crisis.

POLICY CONCLUSION 7-1: Economic policies are needed to address the larger economic and social strains and dislocations that made communities that experienced economic decline over the past four decades vulnerable to opioids and other drugs. This effort may require a holistic approach to development that involves federal, state, and local governments as well as a range of private-sector actors.

Public health policies that address SUDs have often been underutilized in favor of criminal justice policies—e.g., the "War on Drugs"—that emphasize arrests, incarceration, and punishment. Moreover, the United States annually spends much less on demand reduction than on supply reduction programs, and the gap between the two has been growing (Drug Policy Alliance, 2015; Miron and Waldock, 2010; White House Office of National Drug Control Policy, 2015). Public health policies treat substance use as a health problem, while criminal justice policies treat it as a moral failure. The criminal justice approach has been misguided and largely ineffective (Neill, 2014). It has cost the United States upwards of $1 trillion (Branson, 2012), led to extraordinary rates of incarceration that far exceed those of other industrialized nations and have disproportionately affected poor and Black communities (Mauer and King, 2007), and exacerbated tears in the social fabric of these communities (Dumont et al., 2013), all while doing little to curb either the supply of or the demand for drugs in the United States. Similarly, long-standing fear-based programs targeted at youth, such as the National Youth Anti-Drug Media campaign and Drug Abuse Resistance Education (D.A.R.E.) program, are costly and ineffective and have been found to have unintended negative consequences (Hornik and Jacobsohn, 2008; Hornik et al., 2008; Kanof, 2003; U.S. Government Accountability Office, 2006; Vincus et al., 2010; West and O'Neal, 2004). In contrast with a punitive zero-tolerance, War on Drugs approach, a public health or social determinants approach emphasizes integrating clinical care with efforts to improve structural environments and targeting both supply *and* demand factors at multiple levels (Dasgupta, Beletsky, and Ciccarone, 2018; Scutchfield and Keck, 2017).

RECOMMENDATION 7-1: Policy makers should implement policies that better address the U.S. addiction and overdose crisis and prevent future crises. In general, the most effective interventions target both risk and protective factors at multiple levels, including the individual, family, community, and society.

- **The Food and Drug Administration, the Drug Enforcement Administration, and other federal and state regulatory agencies should strengthen regulatory control and monitoring of the development, marketing, distribution, and dispensing of prescription drugs.**
- **The pharmaceutical industry (including manufacturers, distributors, dispensers, and trade associations) should develop and fund stronger internal standards, regulatory structures, and procedures for surveillance and prevention of activities that could result in misuse, addiction, or other harms among users of its products. It should also develop stronger sanctions for violation of these standards.**
- **Federal, state, and local governments should invest in programs that focus on substance use as a public health issue and pursue alternatives to arrest and incarceration. Such programs should be aimed at reducing barriers to and encouraging entry into substance use disorder treatment.**
- **Medicaid and state and local government agencies (e.g., health departments, social services, public schools) should expand access to and improve the quality of substance use prevention, treatment, recovery, and harm reduction programs, as well as mental health counseling and treatment for people with substance use disorders. Substance use prevention programs should begin early, focus on life skills training and prosocial development rather than on fear, and be targeted to children and adolescents most at risk of early initiation of drug and alcohol use (e.g., those living in neighborhoods of low socioeconomic status, those who have suffered adverse childhood experiences).**

Given the relationship between health insurance coverage and access to substance use treatment, as well as recent findings regarding largely better health and lower mortality among working-age adults who live in states that have expanded Medicaid under the Affordable Care Act (see Chapter 11), the committee also recommends that the 12 states that have not yet expanded access to Medicaid do so as soon as possible (see Chapter 11, Recommendation 11-1).

People with SUDs face numerous barriers to accessing the treatment they need, including limits on health insurance coverage, low Medicaid provider participation rates, shortages of mental health and substance use specialists, and fragmentation in care delivery ("carve-outs" and other policies that separate mental health from physical health care) (Byers et al., 2017; Huskamp and Iglehart, 2016; Kakuma et al., 2011; Rowan, McAlpine, and Blewett, 2013; Walker et al., 2015). As a result, an estimated 81.7 percent of Americans ages 12 and over with SUDs do not receive the treatment they

need (Huskamp and Iglehart, 2016). Moreover, despite their demonstrated efficacy in reducing overdose risk (Connery, 2015; Gallagher et al., 2019; Marlatt and Witkiewitz, 2010), access to harm reduction products and services, such as naloxone, medication-assisted therapies, needle exchange programs, safe injection sites, and recovery services (e.g., recovery housing, recovery groups, recovery coaches), is even scarcer (Dasgupta, Beletsky, and Ciccarone, 2018).

Addressing the control of substance addiction is an important multidisciplinary issue.

Examples of specific research gaps that require attention include improving behavioral approaches to relapse prevention, addressing the role of non-substance-related conditions in addictive behaviors, and developing better interventions to counter the adverse effects of various social groups in promoting substance use. The social and behavioral sciences can address the population affected by and clinical scope of addiction, and whether the addiction syndrome subsumes a larger set of non-substance use behaviors, such as pathological gambling (Potenza, 2006). Further research may facilitate better understanding of the biological mechanisms behind addiction and related impulse control disorders, and how those mechanisms can inform approaches to clinical management. Another important issue is the role of impulsivity in clinical substance use relapse among residents of treatment facilities. The social and behavioral sciences have yielded mindfulness strategies for deterring this relapse, and evidence suggests that the strategies can serve as the basis for viable and useful intervention (Davis et al., 2019). Social networks also play an important role in the genesis of substance use in adolescence and young adulthood, as the involvement of disadvantaged young populations with peer friends has been shown to be associated both with increased tobacco, alcohol, and other substance use and with prevention of substance use (Tucker et al., 2015).

RECOMMENDATION 7-2: Federal agencies, in partnership with private foundations and other funding entities, should support research on the effectiveness of behavioral health interventions in reducing mental illness and its consequences; on improved methods for delivering mental health and substance use treatment, harm reduction products and services (e.g., naloxone, medication-assisted therapies, needle exchange programs, safe injection sites), and recovery services; and on the extent to which inadequate access to these products and services has contributed to rising working-age mortality from substance use and suicide.

While substantial research has already focused on explaining the rise in substance-related mortality in the United States over the past three decades, this research has been limited by several research and data gaps, which are

discussed below. Addressing these gaps would aid in better understanding this rise in drug poisoning and alcohol-induced deaths and help inform policy solutions. The recommendations below address these data and research needs (see also Recommendation 7-2 above).

The existing literature offers both supply- and demand-side explanations for the observed trends in substance-related mortality over the past three decades. Supply-side explanations focus on the availability of addictive drugs and alcohol. More research on the marketing, distribution, and regulation of these legal and illegal products is warranted. It is known that variation in state regulations regarding physicians' ability to prescribe opioids influenced the magnitude of the opioid addiction problem across states. However, better understanding is needed of physicians' and patients' responses to tighter regulations and how those responses interface with the markets for heroin and fentanyl. Many believe that as regulatory measures tighten controls on prescription opioids, substance users are pushed into the markets for heroin and fentanyl. It would be useful to know whether policy makers could effectively coordinate their regulatory policies on physician prescribing and their enforcement efforts against illegal drugs.

It appears clear that the lethality of opioids (relative to other drugs) contributed in important ways to the increase in drug poisoning mortality rates (regardless of whether SUDs themselves have actually increased). It would be valuable to understand the extent to which changes in the types of alcohol consumed by Americans (e.g., greater consumption of hard liquor) or the quantities consumed during drinking sessions (e.g., binge drinking) have increased the toxicity of the behavior and contributed to rising alcohol mortality rates among Whites.

Demand-side explanations focus on the factors that make certain demographic groups more vulnerable to drug and alcohol addiction and poisoning. Qualitative researchers have interviewed individuals who are living lives plagued by drug and alcohol misuse and dependence. The testimonies of these people highlight the role of declines in communities and families triggered by changes in economic opportunities. Yet while qualitative research supports this narrative, more rigorous quantitative research has not been as convincing. It is unknown whether these stories of hardship, primarily from the Appalachia region, are generalizable to other regions of the country or other racial/ethnic groups. There is a need for both qualitative and quantitative research focused on other regions and demographic groups to provide valuable insights into why the trends in mortality due to drug poisoning and alcohol use vary so much by region and demography. As elaborated in Chapter 11 (see Recommendation 11-6), more research is needed on explanations for differences in trends (changes) in drug- and alcohol-related mortality across different individual-level demographic characteristics (including sex, race and ethnicity, and socioeconomic

status), economic and social factors (e.g., social integration, unemployment, income inequality, public policy), and various levels of geography (e.g., economic disadvantage at the state, labor market, and area levels).

It is also unclear whether drug overdose and alcohol-induced deaths (and suicide) reflect competing or mutually overlapping causes of death. Evidence of alcohol consumption is known to be common in both drug overdoses (Jones, Paulozzi, and Mack, 2014; Tori, Larochelle, and Naimi, 2020) and suicides (Kaplan et al., 2014), so alcohol use could be said to play a significant role in accidental drug poisoning and suicide. For example, alcohol is involved in about 18.5 percent of overdoses involving prescription opioids (Jones, Paulozzi, and Mack, 2014), about 15.5 percent of those involving heroin (Tori, Larochelle, and Naimi, 2020), and 27.2 percent of those involving benzodiazepines (Jones, Paulozzi, and Mack, 2014). There are studies showing that some individuals use cannabis as a substitute for alcohol and other drugs for medical purposes (e.g., symptom management, reduction of withdrawal, pain treatment) (Reiman, 2009). In a small sample of high-risk drug users undergoing substance use treatment, Shapira and colleagues (2020) found that more than three-quarters of the sample reported substituting their preferred drug for another illicit substance (e.g., substituting street methadone and transdermal prescription opioid patches for heroin). But little is known about whether people substitute drugs for alcohol based on supply-side changes and whether the effects of such substitutions vary by demographic group and geography. For example, were alcohol-related deaths and/or suicides comparatively high during the 1980s and/or 1990s in places that currently have high rates of drug overdose? Do areas with more overdose deaths have lower suicide-/alcohol-related deaths? What role does mental distress play in overdose deaths, suicides and other mental health disorders, and alcohol use? What factors drive overdose versus suicide versus alcohol-related death in the presence of mental distress?

RECOMMENDATION 7-3: The National Institutes of Health, the Substance Abuse and Mental Health Services Administration, the Centers for Disease Control and Prevention, the Food and Drug Administration, and other relevant federal agencies should support research to address the gaps in knowledge regarding the underlying causes of the rise in drug poisoning, alcohol-related death, and suicide. Specifically, this research should be focused on

- **the mechanisms underlying physicians' and patients' unintended responses to tighter regulation of drugs with a high risk of misuse and addiction, such as cases in which individuals dependent on prescription opioids were pushed to markets for heroin and fentanyl,**

and the identification of strategies for preventing those unintended consequences;

- **whether changes over time in alcohol consumption (including types of alcoholic beverages, frequency of drinking, and volume of consumption), in advertising and promotion of alcohol, in cultural acceptance of alcohol use, and in concurrent use of drugs and alcohol have contributed to increases in alcohol-related mortality rates; and**
- **whether the various multilevel mechanisms that explain demographic and geographic differences and temporal changes in drug use are the same as or different from those that drive demographic and geographic differences and temporal changes in alcohol use and suicide.**

In the absence of clinically validated measures, researchers have used various indicators to measure despair (e.g., hopelessness, optimism, happiness). As elaborated in Chapter 11 (see Recommendation 11-2), the interrelationship between mental and physical health and the implications for mortality trends also require further exploration, as some physical health morbidities (e.g., obesity, diabetes, hypertension) that have contributed to rising midlife mortality rates may also be related to mental health and/or psychological distress. Also needed is greater understanding of how trends (changes) in physical pain and psychological distress (or subjective measures of despair) vary by individual demographic group, SES, and geography.

As elaborated in Chapter 11 (see Recommendation 11-3), research on mortality trends would benefit from more analyses of multiple causes of death (MCDs). Death certificates include one underlying cause of death (UCD)—the cause the certifier has determined led directly to the death—and up to 20 contributing causes (i.e., MCDs). The conclusions one draws about the magnitude of the role played by specific causes of death in overall population mortality trends vary dramatically by whether one uses the UCD or MCD data (Redelings, Sorvillo, and Simon, 2006). Researchers should make better use of the codes for MCD in the ICD-10 in their examination, analysis, and explanation of trends in cause of death. Most research on these trends uses the ICD-10 code for UCD. But doing so misses important comorbidities and co-occurring conditions (e.g., alcohol or drug involvement in motor vehicle or pedestrian accidents, chronic substance use and heart disease, drug use and infectious disease) without which the person might not have died. An important task for future research is to consider different ways of categorizing causes of death so they shed light on multiple determinants more directly.

The committee acknowledges the important work of ongoing national surveys of mental health disorders and drug and alcohol use behaviors (e.g., NSDUH, National Epidemiologic Survey on Alcohol and Related Conditions, BRFSS), as well as administrative surveillance and vital record systems (e.g., National Center for Health Statistics vital records, National Violent Death Reporting System). However, these surveys and systems have several critical gaps that need to be addressed.

U.S. death certificates, which are compiled and made available to researchers by the National Center for Health Statistics, include decedents' educational attainment. However, the quality of the educational data varies across demographic group and states. More accurate capture of these data is needed (see Chapter 5 and Recommendation 5-1). Improvement in these data would enable better testing of hypotheses as to why drug mortality rates have increased among individuals without a 4-year college degree but remained relatively flat among those with a college degree (Case and Deaton, 2020). Several important studies have examined APC trends in drug and alcohol deaths. However, these studies have been hampered by the inability to examine those trends by decedents' educational attainment. Such studies are key to elucidating the relative contribution of increasing disadvantage among lower-educated individuals to their rising rates of drug and alcohol mortality.

More comprehensive identification of contributing causes of death on death certificates is also important (see Chapter 5 and Recommendation 5-1). The completeness of the MCD indicators on death certificates varies by certifier, and there are important differences in this regard by decedent demographic characteristics and other nonmedical factors (Wall et al., 2005). More systematic completion of the MCD section on death certificates would facilitate research on comorbid physical and mental health conditions and on the interrelationships among mental illnesses, SUDs, and suicides. Information from more complete and accurate death certificates could also be integrated into population surveillance, cohort studies, and interventional clinical trials addressing use of drugs and alcohol.

Given large and widening geographic disparities in drug- and alcohol-related mortality rates, the inclusion of geographic identifiers on the publicly accessible versions of national substance use and mental health surveillance surveys is urgently needed. The NSDUH is the only national annual surveillance survey designed explicitly to capture detailed information from individuals about both substance use behaviors and mental health conditions. Because of data privacy concerns, however, the publicly accessible version of these data includes no geographic identifiers. Researchers can apply to access the restricted-use data, which include state, county, and lower-level geographic identifiers, but the application and approval process

is time-intensive, and the only way to access the data upon approval is through a Federal Statistical Research Data Center. Few researchers can easily access these data centers. A better way is needed to balance respondents' data privacy and the release of essential information to help researchers identify and better understand trends in major causes of death, especially for data from such surveys as the NSDUH, which includes nearly 70,000 respondents (thereby greatly reducing disclosure risk).

RECOMMENDATION 7-4: The Substance Abuse and Mental Health Services Administration should add to the publicly accessible version of the National Survey on Drug Use and Health U.S. Census region or U.S. Census division categories and the nine-category U.S. Department of Agriculture Economic Research Service rural–urban continuum codes or National Center for Health Statistics urban influence codes.

The NSDUH has a large depression inventory module and a module on the use of psychiatric clinic services; the NESARC (last conducted in 2011–2012) collects information about various types of anxiety disorders (including panic disorders) that are more common in people with SUDs, and it also asks about depression. Nevertheless, there are important gaps in the availability of information on adult mental illness rates in the United States as a whole and in regional jurisdictions. More of this information is needed to understand trends in the relationship between mental health conditions and SUDs; identify the levels of unmet population need for prevention and treatment of these conditions; and assess the outcomes of these conditions, including social dysfunction, drug and alcohol use, and suicide and other related mortality.

RECOMMENDATION 7-5: The National Institute of Mental Health and other relevant federal agencies should develop a research program to identify innovative and cost-effective methods for conducting periodic or ongoing population surveys of important mental health conditions. The research agenda should include measuring access to and uptake of behavioral health care services (e.g., mental health counseling, substance use disorder treatment) and the effects of such services on mental health outcomes and other important outcomes, such as those in the social, cognitive, and functional domains. These national surveys should be linked where possible to medical record and claims data, as well as to other important sources, such as education and social service information, while carefully protecting respondent confidentiality.

Finally, research on temporal trends in the prevalence of ACEs is sparse, and better data would greatly improve the ability of researchers to examine trends in the prevalence and demographic distribution of such experiences, as well as changes in their relationship to adult health behaviors and health outcomes.

RECOMMENDATION 7-6: Questions about adverse childhood experiences should be added to the core of the Behavioral Risk Factor Surveillance System (so that the questions are asked in every state in every year), as well as to other relevant national health surveys, such as the National Health Interview Survey and the National Survey on Drug Use and Health. To advance understanding of the mechanisms and control of these experiences, this information should be improved by facilitating maximal record linkage of cohort findings to available social, military, medical, psychiatric, environmental, and law enforcement records.

8

Suicide

Suicide was among the 10 leading causes of death in the United States among working-age adults (ages 25–64) in 2015, 2016, and 2017, when recent declines in life expectancy emerged, and despite the small overall increase in U.S. life expectancy in 2018, suicide mortality continued to increase in that year (Xu et al., 2020). During the study period, 1990–2017, suicide accounted for 569,099 deaths among the working-age population (Centers for Disease Control and Prevention [CDC], 2020b). Historically, suicide mortality has been substantially higher among men than women and among non-Hispanic (NH) Whites (Whites) than NH Blacks (Blacks) and Hispanics, and this was the case among working-age adults during the study period. During this period, moreover, suicide mortality increased substantially *mainly* for Whites, with the largest absolute increases occurring among White males across the 25–64 age range.[1] Suicide is clearly a prominent preventable cause of death, particularly among working-age Whites, and an important public health concern.

This chapter focuses on the trends and disparities in and explanations for the recent rise in suicide mortality among U.S. working-age adults. For purposes of this report, suicide deaths do not include suicides by drug poisoning (which are counted as drug-related deaths in the data shown in Chapter 7) because of the difficulty of differentiating between intentional

[1]Among all racial/ethnic groups, suicide mortality is highest among American Indians/Alaska Natives (Leavitt et al., 2018). As noted in Box 4-2 in Chapter 4, because of data quality concerns, mortality trends among this population subgroup are not examined in this report.

and accidental drug poisoning.[2] First, the trends in suicide mortality are presented by age, sex, race and ethnicity, and geography for the study period (1990–2017). This is followed by a review of the research literature on factors related to suicide mortality and the degree to which such factors have changed to bring about the recent rise in suicides. Given that the rise in suicide mortality rates was driven by the rising rates among Whites, explanations for the increase in suicide mortality should focus mainly on working-age Whites. A paucity of existing research, however, examines suicide by race and ethnicity or seeks to explain the recent rise in suicide. Nevertheless, this chapter presents the committee's assessment of the literature in explaining the trends in suicide, along with its recommendation for addressing related data needs.

Research on suicide mortality tends to focus in four general areas: economic factors; social engagement, religious participation, and social support; access to lethal means; and mental, emotional, and physical health. Some of the stronger evidence has been found for the role of economic conditions. Periods of economic downturn, wage stagnation, weak safety nets, and increasing foreclosure rates are associated with rising suicide mortality in national- and state-level data. While the research literature provides some compelling evidence for links between changes in economic conditions, social integration, and psychological and physical well-being and the rise in suicide mortality among Whites, most of this evidence is suggestive and obscures the fact that these factors are interrelated and operate across the societal, community, and individual levels.

TRENDS IN SUICIDE

As noted, the increase in suicide mortality over the past three decades occurred primarily among Whites, with both the levels and absolute increase being higher among men than women (Figure 8-1). From 1990 to 2017, the contribution of suicide to the overall mortality increase among White men ranged from 12.8 percent at ages 25–44 to 8.0 percent at ages 55–64 (see Table 4-1 in Chapter 4). In 2017, suicide was the second leading cause of death among White men ages 25–44, the fourth leading cause among those ages 45–54, and the seventh leading cause among those ages 55–64 (Heron, 2019).

Although at lower levels, suicide rates began to rise among White women after 2000 (Figure 8-1), contributing to the rise in White female mortality from 1990 to 2017. The contribution of suicide to the overall

[2]As detailed later in the chapter, between 1990 and 2017, drugs contributed to 11.5–16.7 percent of all suicides, depending on the year, with their contribution being higher for women than for men.

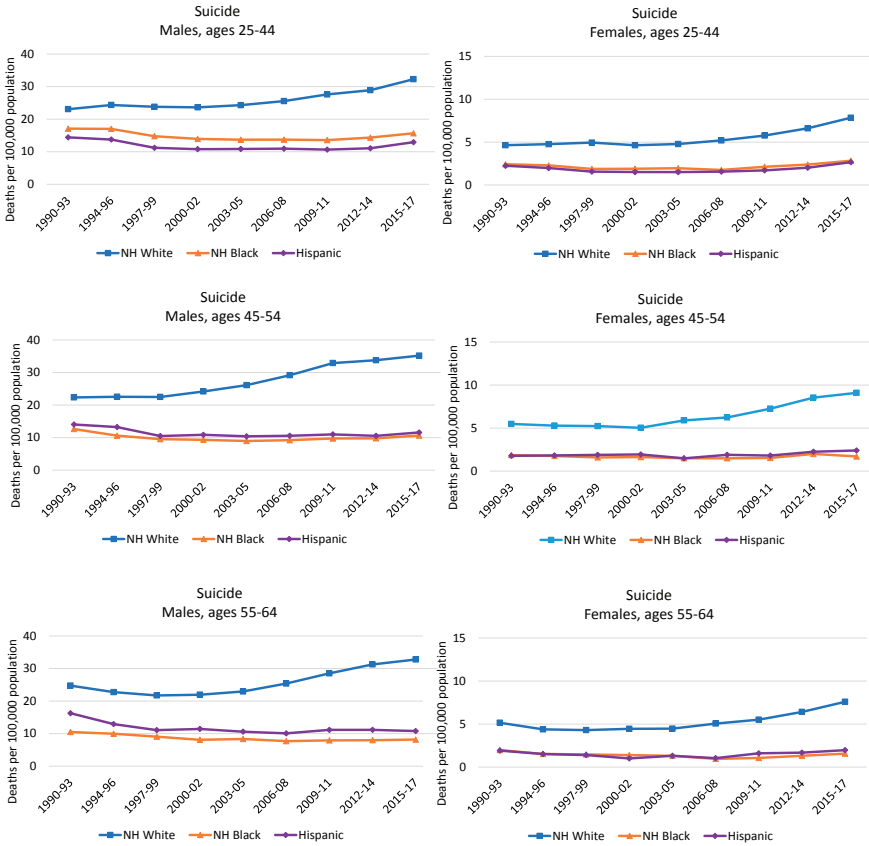


FIGURE 8-1 Suicide rates per 100,000 population among U.S. working-age adults (ages 25–64), 1990–2017, by sex, age, and race and ethnicity.
NOTE: Suicide deaths by drug poisoning are not included. Each panel shows suicide rates for non-Hispanic (NH) Whites (blue lines), NH Blacks (orange lines), and Hispanics (purple lines). Suicide rates for males are shown in the lefthand panels, while those for females are shown in the righthand panels. Suicide rates are shown for three age groups: 25–44 (top panels), 45–54 (middle panels), and 55–64 (bottom panels). Rates are age-adjusted to reflect a standard population age distribution.
SOURCE: Data from National Vital Statistics System Detailed Mortality Files, https://www.cdc.gov/nchs/nvss/deaths.htm.

mortality increase among White women ranged from 7.0 percent at ages 25–44 to 2.6 percent at ages 55–64 (see Table 4-1 in Chapter 4). In 2017, suicide was the second leading course of death among White women ages 25–34, the fourth leading cause among those ages 35–44, the fifth leading cause among those ages 45–54, and the ninth leading cause among those ages 55–64 (Heron, 2019).

Compared with the suicide rates among Whites, the rates among Blacks and Hispanics were relatively flat or declined between 1990 and 2017 (Figure 8-1). Still, suicide was among the 10 leading causes of death among Black men ages 25–64, Black women ages 25–44, Hispanic men ages 25–64, and Hispanic women ages 25–54 (Heron, 2019). During the 1990s, suicide rates among working-age Blacks and Hispanics generally decreased. Beginning in the 2000s, suicide rates increased among working-age Whites, particularly White males, while remaining steady among working-age Blacks and Hispanics. However, starting in about 2010, suicide rates also began to increase among Black and Hispanic males and females in most age groups, although these increases were smaller than those among working-age Whites.[3]

At the same time, working-age Whites experienced consistently higher suicide rates relative to working-age Blacks or Hispanics throughout the period. As the rates declined among Black and Hispanic adults in the 1990s, the gap between White adults and Black and Hispanic adults grew. This disparity widened through the remainder of the period as the rates increased steadily among White adults.

At the beginning of the study period, there was little difference in suicide rates by metropolitan status among White adults. Over time, however, suicide rates increased more slowly in large central metropolitan areas (hereafter referred to as "large central metros") than in less-populated areas, initiating a widening nonmetro suicide "penalty" in nonmetropolitan areas (hereafter referred to as "nonmetros") (Figure 8-2). The nonmetro disadvantage was largest among White males ages 25–44 and is a trend that the research literature has noted for all males (Singh and Siahpush, 2002).

In contrast, differences by metropolitan status among working-age Blacks were much smaller, although they also grew for Black women ages 45–64. Among younger working-age Hispanics, suicide rates were highest

[3]Between 2009–2011 and 2015–2017, the absolute increase in the suicide rate among younger working-age Black men (ages 25–44) was as large as the absolute increase in the rate among younger working-age White women (ages 25–44). While the absolute increase in suicide rates was the same for these groups in this most recent period, the levels of suicide are always lower for women than for men. In 2015–2017, for example, the suicide rate for younger working-age Black men ages 25–44 was twice as high (15.7) as the rate for younger working-age White women (7.9), compared with the higher suicide rate for younger working-age White men (32.3) (see Tables A-1, A-2, and A-4 in Appendix A).

FIGURE 8-2 Suicide rates per 100,000 population among U.S. working-age males and females (ages 25–64), 1990–1993 through 2015–2017, by metropolitan status.
NOTE: Suicide rates are shown for two age groups—25–44 (panels a-c and g-i) and 45–64 (panels d-f and j-l)—across four levels of metropolitan status: large central metropolitan counties (blue lines), large fringe metropolitan counties (orange lines), small or medium metropolitan counties (gray lines), and nonmetropolitan counties (yellow lines). Trends in these four groups are presented separately by sex (males in panels a-f, females in panels g-l) and for non-Hispanic (NH) Whites (panels a, d, g, and j), NH Blacks (panels b, e, h, and k), and Hispanics (panels c, f, i, and l). Rates are age-adjusted by 10-year age group.
SOURCE: Data from National Vital Statistics System Detailed Mortality Files, https://www.cdc.gov/nchs/nvss/deaths.htm.

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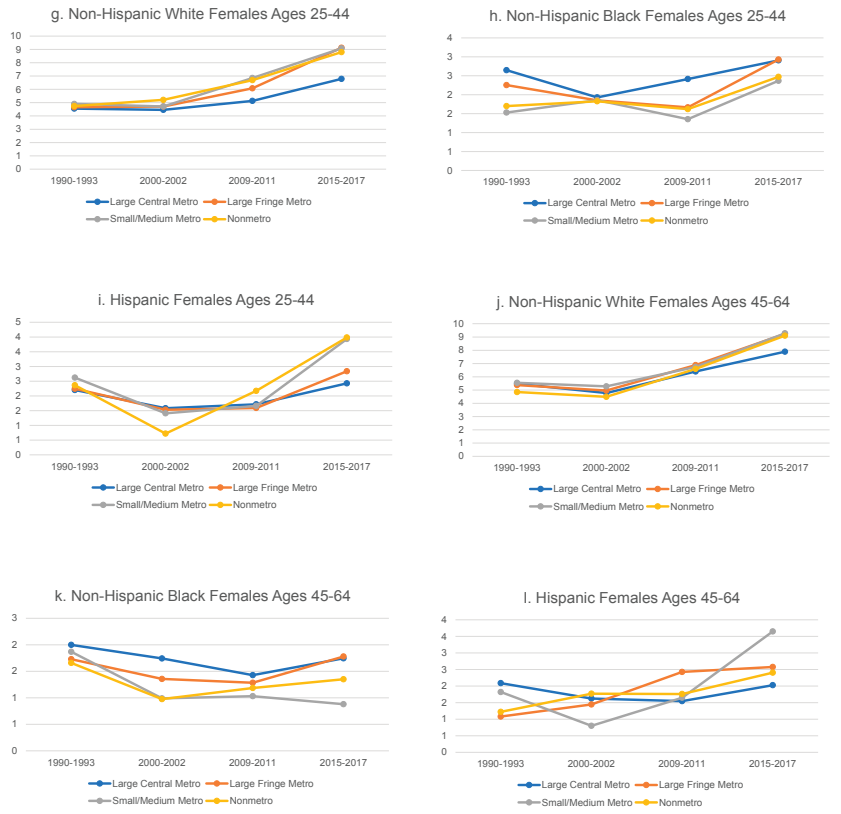

FIGURE 8-2 Continued

in nonmetro counties and lowest in large central metro counties throughout the period. Among older Hispanic males, the rates in nonmetro areas were the highest throughout the period, declined less in the 1990s, and increased more in the 2010s relative to other areas. They were lowest in large fringe metropolitan areas (hereafter referred to as "large fringe metros") in 1990–1993 but increased in these areas during the 2000s, surpassing suicide rates in large central metros and small/medium metropolitan areas (hereafter referred to as "small/medium metros") after 2010.

In the working-age (ages 25–64) population overall, male suicide rates increased by varying amounts in all but seven states between 1990 and 2017 (Figure 8-3), leading to increasing state-by-state variability in suicide mortality. This state-by-state variation in suicide mortality is not new (Miller, Azrael, and Barber 2012; Phillips, 2013), but it has widened during recent decades. The absolute increases were especially large in several Midwestern,

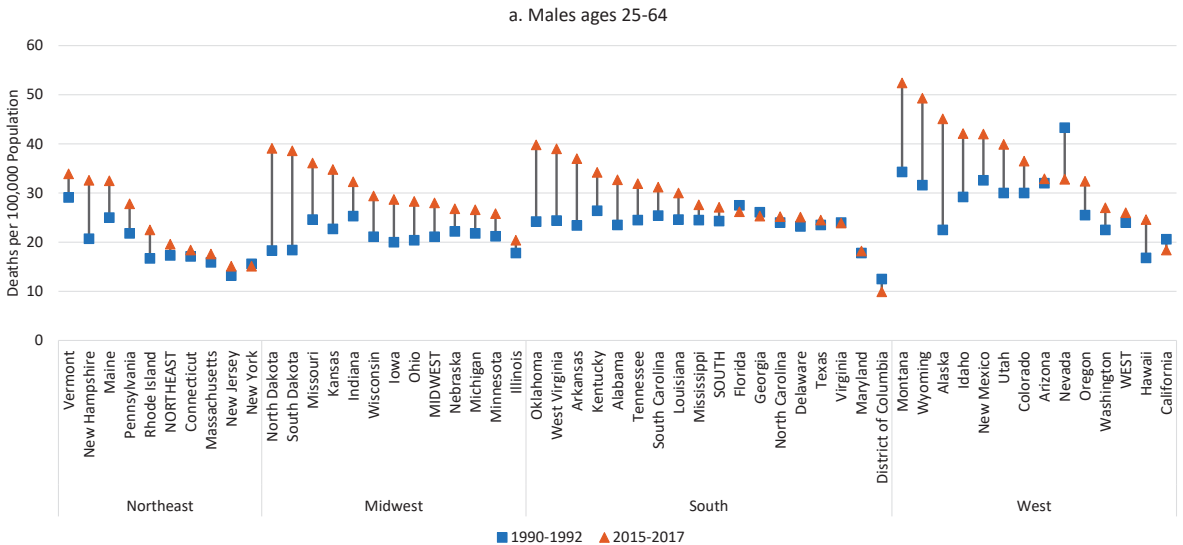

FIGURE 8-3 Suicide rates per 100,000 population among U.S. working-age males and females (ages 25–64), 1990–1992 and 2015–2017, by region and state.

NOTE: Suicide rates are shown for 1990–1992 (blue squares) and 2015–2017 (orange triangles), along with the changes over time (black connecting lines). Suicide rates for males are shown in panel a above, while those for females are shown in panel b on the next page. Rates are age-adjusted by 10-year age group. For males, the 1990–1992 rate for Alaska represents 1991 and 1992 only; the rate is suppressed for 1990. For both males and females, North Dakota is excluded to comply with Centers for Disease Control and Prevention (CDC) suppression criteria (fewer than 10 deaths in 1990–1992). For females, the District of Columbia is excluded for the same reason. States are ordered from highest to lowest mortality rate in 2015–2017 within region.

SOURCE: Data from CDC WONDER Online Database, https://wonder.cdc.gov.

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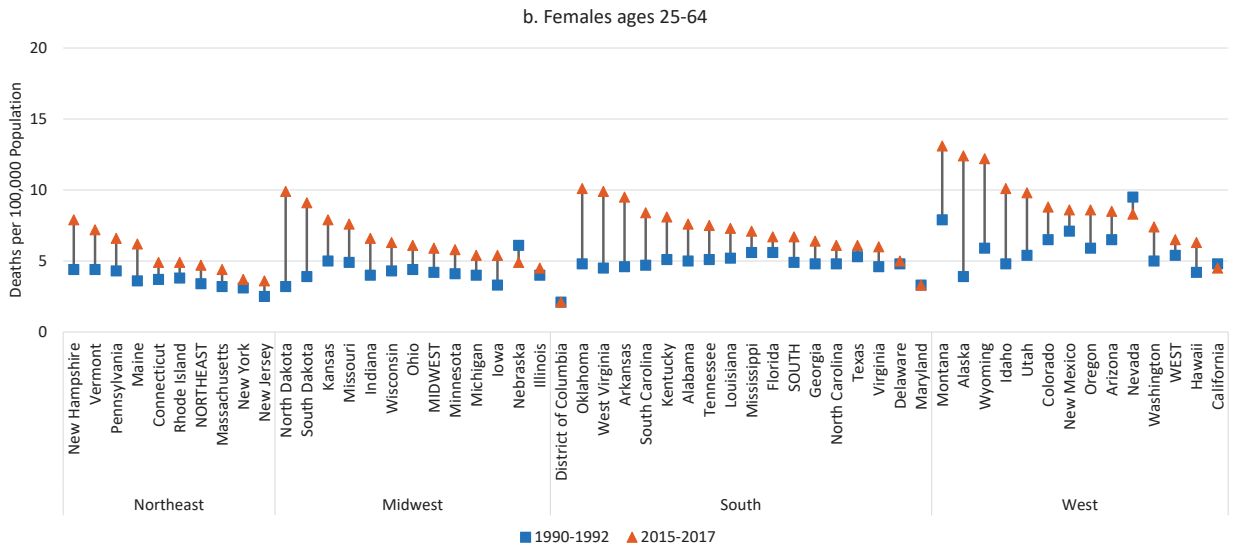

FIGURE 8-3 Continued

Southern, and Western states, whereas the percentage increases were largest in North and South Dakota, Oklahoma, West Virginia, and Arkansas. The largest percentage declines were in Nevada, the District of Columbia, and California. The highest rates in 2015–2017 were all in the West: Montana, Wyoming, Alaska, Idaho, and New Mexico. Among females ages 25–64, the rates increased in all but four states, and there were small declines in California, Nevada, and Nebraska. As with men, the absolute increases among females were most pronounced in several Midwestern, Southern, and Western states, and the largest percentage increases were in North and South Dakota, West Virginia, Oklahoma, and Idaho. The highest female suicide rates in 2015–2017 were in Montana, Alaska, Wyoming, Oklahoma, and Idaho.

Research suggests that suicide mortality began to rise for the baby boom cohorts in the late 1990s and continued to climb in succeeding birth cohorts. This pattern appears to be most evident for those without a 4-year college degree (Case and Deaton, 2017, 2020; Phillips et al., 2010). Using three different approaches to estimate an age–period–cohort model of suicide mortality, Phillips (2014) found that, although baby boom cohorts did not have higher suicide rates than previous birth cohorts, male and to a lesser extent female suicide rates began to rise and continued to do so for subsequent birth cohorts. Thus, the baby boom cohorts appear to have ushered in new cohort patterns of suicide rates over the life course. Chauvel, Leist, and Smith (2016) studied suicide mortality in 1990–2010 for birth cohorts, net of age and cohort linear time trends, and further documented sharp increases in suicide mortality for White men with low levels of education among cohorts born between 1955 and 1970.

Explanations for the Rise in Suicide Mortality

A range of societal, community, relationship, and individual factors undoubtedly contribute to suicide ideation, attempts, and mortality, as conceptualized in Figure 6-1 in Chapter 6. At the same time, the risk can be mitigated by protective factors, such as community and family supports, access to mental health and substance use services, and cultural and religious beliefs (Conejero et al., 2016; Denney et al., 2009; Pescosolido, 1990; Pescosolido and Georgianna, 1989; Wray, Colen, and Pescosolido, 2011). Durkheim's (1951) focus on the role of social integration and societal regulation in times of rapid social change in shaping suicidal behavior is informative in this regard. According to research in the Durkheim tradition, individuals' social ties within the family, with religious and other organizations, and in political domains (including participation in the labor force) reduce social isolation and buffer against suicidal behavior (Abrutyn and Mueller, 2018; Bearman, 1991; Durkheim, 1951; Pescosolido, 1994). These

social ties may be particularly critical for individuals facing stressful life events and other personal crises. The interpersonal theory of suicide mortality in turn proposes that feelings of perceived burdensomeness and thwarted belonginess can precipitate a suicidal desire and the act itself (Chu et al., 2017). The reasons for the increase in suicide mortality among Whites and its geographic variation, as well as factors that account for trends among other racial/ethnic groups, are likely to be multifactorial and thus to escape simple explanations. While it is important to consider the role of both predisposing and protective factors (Shanahan et al., 2019), their role in the increase in suicides among Whites has not been extensively studied. The review that follows includes research that has considered various factors that may have played a role: economic factors; social engagement, religious participation, and social support; access to lethal means; and mental, emotional, and physical health.

Economic Factors

The available evidence suggests that suicide mortality can be responsive to economic conditions. Employment may mitigate against suicide risk in a number of ways, including steady income, employment-based social networks, access to informational resources and health and other employment-based benefits, and a sense of self-worth. Historical data for the United States between 1928 and 2007 link trends in suicide mortality to business cycles, with rates rising during recessions and declining during economic expansions among working-age adults (Luo et al., 2011). An increase in the unemployment rate has been associated with higher death rates from suicide, although this association may depend on the time period in which unemployment occurs (Catalano et al., 2011; Modrek et al., 2013; Tapia Granados, 2005; Tapia Granados et al., 2009). Using longitudinal state-level data for the period 1972–1991 with state fixed effects and controls for state-level educational attainment, race and ethnicity, per capital income, and age, Ruhm (2000) found that suicide mortality increases by 1.3 percent for every 1 percent increase in state unemployment rates. Using similar methodology, with controls for a set of economic, demographic and social/cultural variables,[4] Phillips and Nugent (2014) found a significant positive association between unemployment and suicide mortality in midlife based on state-level data from 1997 to 2010; this association appeared stronger in states with higher female labor force participation rates. In a separate study between 1976 and 2000 (Phillips, 2013), however, unemployment was not

[4]Female labor force participation rate, percent employed in manufacturing, per capita income, sex composition, age structure, percent foreign-born, percent divorced, alcohol consumption (gallons per capita), and antidepressant drug use.

associated with elevated suicide mortality. Similarly, DeFina and Hannon (2015), using state-level data on rates of unemployment and suicide mortality with state fixed effects, found a significant positive association between the two rates from 1995 to 2010 but not between 1979 and 1995. They attribute this pattern to growing economic insecurity resulting from wage stagnation, changes in employment and hiring practices, and more stringent requirements for safety net programs in the latter period that made individuals more vulnerable to the effects of unemployment (DeFina and Hannon, 2015; Hacker et al., 2010).

That economic insecurity may play some role in the recent increase in suicide mortality is apparent from analyses of the National Violent Death Reporting System (NVDRS), which provides information on circumstances surrounding completed suicides. Hempstead and Phillips (2015) used these data to examine circumstances surrounding suicide mortality between 2005 and 2010 and found that economic troubles (e.g., job, financial, or legal problems) increased significantly among individuals ages 40–64, and that these increases were related to suicide by suffocation, a lethal method that has increased in recent years. Similarly, Kerr and colleagues (2017), who also used the NVDRS data, found poverty to be strongly associated with suicide mortality between 2005 and 2011, as were foreclosure rates at ages 45–64, while Houle and Light (2014) document a significant association between state-level foreclosure rates and suicide mortality at ages 46–64. Employment has also been tied to a lower risk of suicide in analyses of data from the National Health Interview Survey (NHIS) and the National Longitudinal Mortality Study (NLMS) (Denney et al., 2009; Kposowa, 2001), as have higher levels of education, such that those with lower levels of schooling are at higher risk of suicide (Case and Deaton, 2015, 2017; Denney et al., 2009). A study by Geronimus and colleagues (2019) documents changes in educational disparities in mortality among working-age (and older) adults between 1990 and 2015 for both Black and White women and men, demonstrating that both White women and White men exhibited small increases in educational disparities in working-age mortality due to suicide. These authors measured educational attainment in quartiles to help account for increasing educational attainment across time and showed that working-age Whites with lower levels of education experienced slightly higher increases in suicide rates relative to those with more education.

Indeed, some of the strongest evidence for a link between economic conditions and suicide mortality has been found among those with lower levels of schooling. Kaufman and colleagues (2020) used state-level monthly data to examine the effects of differences between the state and federal minimum wage on suicide mortality among adults ages 18–64 from 1990 to 2015. Their difference-in-differences model found that a $1 increase in the state minimum wage reduced the suicide rate by 3.4–5.9 percent among

those with a high school education or less, and the effects were greatest during periods of high unemployment. Similar findings are reported by Dow and colleagues (2019), who examined the effects of state variation in minimum wages and the Earned Income Tax Credit (EITC) on suicide mortality among those ages 18–64 from 1999 to 2015. They report that a 10 percent increase in the minimum wage reduced nondrug suicides among adults with a high school education or less by 3.6 percent; a 10 percent increase in the EITC reduced suicides in this group by 5.5 percent. Unfortunately, many of these studies, and much of the research on economic conditions, do not examine racial/ethnic differences in the role of economic factors in suicide mortality.

As noted, the recent increase in suicide mortality among Whites is a phenomenon seen primarily among those with a high school education or less (Case and Deaton, 2015, 2017, 2020). Since the 1970s, the wages for this population have stagnated, and their rates of labor force participation have declined (Case and Deaton, 2020). Since the late 1960s, for example, when labor force participation rates among working-age males were fairly similar across all education groups, the rates have steadily declined among those with a high school education or less (Krause and Sawhill, 2017), and they continued to decline following the Great Recession despite historically low unemployment rates in recent years. Between 2009 and 2016, the labor force participation rate among Whites ages 25–64 with less than a high school education declined from 58 percent to 53 percent; the decline for those with a high school education was from 76 percent to 72 percent. In contrast, labor force participation rates among those with a bachelor's degree or higher remained steady at 86 percent (National Center for Education Statistics [NCES], 2020a, 2020b). At the same time, the loss of manufacturing employment and the growth of service-sector jobs have changed the nature of work and compensation for those with low levels of education. Case and Deaton (2020) argue that the long-term economic trends have had a cumulative negative impact on the lives of cohorts born since the 1940s.

Although these changes are not unique for Whites, evidence suggests that the association between the changing economy and "deaths of despair," of which suicide mortality is a key component, is stronger for Whites than for other racial/ethnic groups (see Chapter 7). Pierce and Schott (2020) found this to be the case in counties more exposed to economic shocks due to trade liberalization. Likewise, a recent study by Graetz and colleagues (2020) found higher levels of manufacturing employment to be a significant predictor of lower suicide mortality among White men and women in 1999 and 2017 in 704 U.S. commuting zones (see also Phillips and Nugent, 2014). The working class is facing some of the bleakest prospects for upward mobility recorded in recent generations, and White cohorts do not appear to be any less immune to these adverse trends relative to other

racial/ethnic groups (Cherlin, 2018; Chetty et al., 2017). This erosion of the American Dream for less-educated White Americans means that they have experienced a relative loss of status in recent decades and can no longer place themselves above other racial/ethnic groups in a class-based social hierarchy (Hochschild, 2016; Silva, 2019). Changing economic conditions and the nature of work, however, are unlikely to explain all of the rise in suicide mortality among White Americans.

Social Engagement, Religious Participation, and Social Support

In the face of stagnating wages and deteriorating economic conditions for those with low levels of education, social institutions could potentially play an important supportive role (Wilcox et al., 2012). Social engagement other than through employment, whether through local civic organizations, school boards, volunteer activities, religious involvement, friendship and family networks, or other forms of community engagement, is hypothesized to reduce social isolation and feelings of loneliness and buffer against self-harm. Yet it appears that such engagement has declined during the past few decades. Although not without his critics, Putnam (2000) draws attention to the decline in several forms of civic participation in the United States in his book, *Bowling Alone*. At the same time, social capital, measured at the individual or the community or state level, has been associated with measures of health and mortality, including suicide, although these associations are likely to be sensitive to what measure of social capital is used and subject to the ecological fallacy[5] (e.g., Kawachi et al., 1997; Lee and Kim, 2013; Smith, Lucia, and Kawachi, 2014). Smith, Lucia, and Kawachi (2014) examined associations between various dimensions of social capital and suicide rates at the state level during 1999–2002, controlling for other state-level characteristics that had been associated with suicide mortality in prior studies, including state-level Gini coefficient, gun ownership, alcohol and drug use, serious mental illness, poverty and unemployment rates, suicide belt state,[6] urbanization, and population instability. They found that suicide rates for White men and women were lower in states with higher levels of social capital, controlling for other state-level characteristics, but not for Black men, the only other group that had sufficient numbers of suicides to be included in the analysis. The results are suggestive for a possible

[5]The ecological fallacy refers to an incorrect interpretation of statistical data that occurs when inferences about the nature of individuals are deduced from inferences about the group to which those individuals belong. An example of this fallacy would be to infer that if states with more Catholics have lower suicide rates, Catholics must be less likely to commit suicide.

[6]The suicide belt is a region of the Western United States where the suicide rate is particularly high compared with the national average. It comprises Arizona, Colorado, Idaho, Montana, Nevada, New Mexico, Oregon, Utah, and Wyoming (Wray, Colen, and Pescosolido, 2011).

role of social engagement in White suicide mortality, although the authors' cross-sectional analysis does not speak to whether *changes* in social capital and civic engagement are potential contributors to rising suicide mortality among White men and women.

Another important form of social engagement is participation in religious organizations. Although some of the association between more frequent attendance at religious services and lower mortality is due to health selection (i.e., unhealthy individuals are less likely to attend religious services), the association is robust to controls for health status, including status related to external causes of death such as suicide (Hummer et al., 1999). Other studies have documented a lower suicide risk in areas with a greater proportion of Catholics, with the results for other denominations being more mixed (Klugman, Condran, and Wray, 2013; Wray, Colen, and Pescosolido, 2011). Similar to the findings of Hummer and colleagues (1999), participation in religious activities predicted lower suicide rates in multivariate analysis of the 1993 National Mortality Followback Survey (Nisbet et al., 2000). The proportion of Americans who do not affiliate with any particular religion has grown over the past decade with each successive younger generation, as has the percentage who do not attend religious services regularly. In 2018–2019, more than 50 percent of White adults said they attended religious services only a few times a year or less, reflecting a growth of close to 10 percent since 2009 and proportions higher than those among Blacks and Hispanics (Pew Research Center, 2019). Based on the General Social Survey, weekly church attendance at ages 40–59 also declined more rapidly for Whites without a college degree than for those with a bachelor's degree between 1970 and 2018 (Case and Deaton, 2020, Figure 12.3). Wilcox and colleagues (2012) similarly document a decline in participation in religious services among Whites ages 25–44 without a college degree, but not among their Black and Hispanic counterparts.

Another significant type of social support is a stable marital relationship. Prior research has documented a lower risk of suicide for married than for unmarried individuals among participants in the NHIS and NLMS, with the association being stronger for men than for women (Denney et al., 2009; Kposowa, 2001). A significant positive association has also been documented between suicide risk and the proportion divorced in one's county and state of residence (Phillips, 2013; Reckera and Moore, 2016; Singh and Siahpush, 2002; Trgovac, Kedron, and Bagchi-Sen, 2015), suggesting that stronger family ties may buffer against self-harm. One of the notable changes in American family life is the decline in marriage, especially among those with lower levels of education (Cherlin, 2018; Ruggles, 2015). Since 1970, being currently married declined for most White women, with the largest declines seen among those with a high school education or less and those with some college. By 2000, White women without a college degree

were less likely to be married than in 1940 or 1970 and less likely to be married than those with a college degree (Torr, 2011). For Black women, this pattern of marital status by educational attainment had emerged by 1970 such that those with lower levels of schooling were less likely to be married than those with a college education (Torr, 2011). Thus, the decline in marriage among White women, and particularly among less-educated White women, is more recent (Cherlin, 2009; Murray, 2012), overlapping with the time period when suicide rates were increasing among Whites.

Not only are women with less than a college degree less likely to be married, but they are also more likely to be divorced or in cohabiting unions with children. Declining wages for men with less than a college degree have made these men less attractive marital partners for women, and a sharp differentiation in marriage patterns and family circumstances has emerged between men and women with and without a college education (Cherlin, 2014). Autor, Dorn, and Hanson (2019) document a link between marriage and a lack of well-paying jobs, especially in regions where automation and trade have led to reductions in such jobs. They found further that these employment shocks are associated with "male idleness and premature mortality, and raise the share of mothers who are unwed and the share of children living in below-poverty, single-headed households" (Autor, Dorn, and Hanson, 2019, p. 161).

Taken together, the above evidence suggests that social support, whether from formal institutions such as churches or other community-based social support networks or from stable interpersonal relationships within marriage, has deteriorated in the past several decades for individuals with lower levels of education, and that these trends have had a more profound impact on Whites than on other racial/ethnic groups. Research examining links between these patterns and suicide rates, however, has been mainly descriptive. To what extent these changes can explain the increase in suicide mortality among White men and women and whether such changes interact with the growing economic stratification and individual-level risk factors requires further investigation.

Access to Lethal Means

Another societal factor that can influence suicide risk is access to lethal means. Recent research has drawn attention to the role of firearms in patterns of suicide mortality by geography and gender. For example, the higher mortality from suicide among males is related to their use of more lethal means (e.g., firearms) relative to females (Miller, Azrael, and Barber, 2012) (Figure 8-4).

Rates of firearm-related suicide are higher in states with looser gun regulations and more gun ownership, and in nonmetropolitan than in

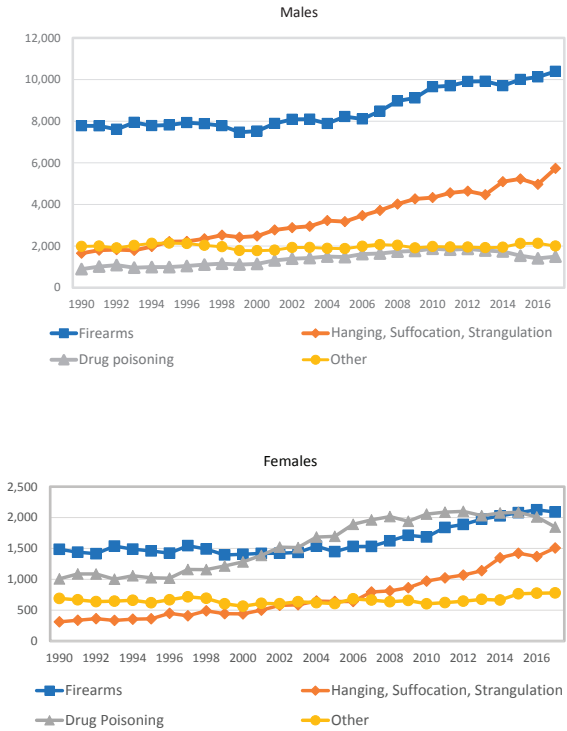

FIGURE 8-4 Number of suicides among U.S. working-age non-Hispanic Whites (ages 25–64), 1990–2017, by type and gender.
SOURCE: CDC WONDER Online Database, https://wonder.cdc.gov.

larger metropolitan counties (Anestis and Anestis, 2015; Anestis, Selby, and Butterworth, 2017; Fleegler et al., 2013; Ivey-Stephenson, Blair, and Crosby, 2018; Kaufman et al., 2018). Anestis, Selby, and Butterworth (2017) studied trends in suicide by firearms between 1999 and 2015 and found that at the state level, the absence of laws requiring universal background checks and imposing a mandatory waiting period for the purchase of firearms were associated with a more steeply rising trajectory of statewide suicide rates. Furthermore, where firearm suicide rates were declining, this decline was not offset by increases in suicide by other means. Others have documented higher suicide rates in states with higher gun ownership (Opoliner et al., 2014), and handgun ownership has been associated with higher rates of firearm-related suicide mortality (Studdert et al., 2020). These findings

suggest that strengthening state gun control laws has the potential to reduce suicide rates and possibly rural–urban differentials in these rates.

In 2017, suicide by firearms accounted for nearly half of all suicides among White men ages 25–64 but only about one-third of suicides among their female counterparts, whereas drug poisoning was a more common means of suicide among women than men. (Recall that in this report, suicides due to drug poisoning are included in the discussion of drug-related mortality [Chapter 7] and are not included in the trends shown in Figures 8-1 through 8-3 above). This gender difference in suicide by drug poisoning may explain some of the lower suicide mortality among women documented in this chapter, which considers only suicide deaths classified as "intentional." However, given that rates of male suicide are 3 to 4 times higher than female rates, the exclusion of deaths from drug poisoning, many of which are accidental, probably plays a small role in the gender differences shown in Figures 8-1 through 8-3. It is also possible that suicides due to drug poisoning are underreported for both males and females (see Rockett et al., 2018).

As seen in Figure 8-4, although the number of suicides by firearms increased among White men between 1999 and 2017, their share of all suicides declined from 63 percent in 1990 to 53 percent in 2017, during the period when suicide mortality was increasing most notably. At the same time, suicides by hanging, suffocation, and strangulation increased from 13 percent to 29 percent among White men. Similarly, among White women, suicides by firearms increased over the period, but their share of all suicides also declined, from 43 percent to 34 percent, while the percentage of suicides due to hanging, suffocation, and strangulation almost tripled, from 9 percent to 24 percent. While the levels are much lower, the distributions of suicides by type are similar for Blacks and Hispanics (not shown). Although looser state gun laws are associated with higher suicide rates by firearms and suicide by firearms contributed to the increase in suicide mortality over this period, suicide by other means made a larger contribution to the overall increase in suicide mortality. The increased contribution of hanging, suffocation, and strangulation to rising suicide rates suggests that changes in gun availability cannot be the primary reason for rising suicide mortality among White men and women.

Mental, Emotional, and Physical Health

An individual's risk of suicide is related to a family history of suicide; prior suicide attempts; psychiatric disorders, including depression and substance use; pain and other health problems; social adversity and deprivation during childhood and adolescence; and impulsivity (Conejero et al., 2016; Denney et al., 2009; Fazel and Runeson, 2020; Ilgen, 2018; Petrosky et

al., 2018). Furthermore, suicide can be precipitated by the experience of stressful life events with or without the presence of mental health problems. An analysis of the NVDRS, for example, found that in 27 states in 2015, relationship problems/loss, life stressors, and recent impending crises were more common among those without versus those with known mental health conditions, although they were common among both groups (Stone et al., 2018).

One of the most important individual risk factors for suicidal behavior and mortality is the presence of mental illness. As discussed in Chapter 7, however, the role of mental illness has been challenging to study because ongoing representative population surveys and other nationwide surveillance instruments do not include comprehensive diagnostic indicators of adult mental illness. Many such studies tend to be conducted in variously selected populations, such as military or veteran populations, prisoners, and the homeless. Several national surveys include nondiagnostic indicators of mental health, such as self-reports of depressive symptoms, anxiety, depression, panic attacks, and psychological distress, but none include the array of commonly diagnosed mental illnesses thought to be most associated with suicide risk, such as anxiety disorders, bipolar disorder, phobias, personality disorders, eating and gambling disorders, schizophrenia and other psychoses, and panic disorders. These mental conditions predict different levels of suicidal behavior (Fazel and Runeson, 2020), but while not all cases of completed suicide are among people with a history of mental illness, the presence of such illnesses is extremely common. A study of risk factors for suicide attempts among U.S. Army soldiers, for example, found that 63.7 percent had a prior history of mental illness (Ursano et al., 2018). Too and colleagues (2019) conducted an extensive meta-analysis of record linkage studies of suicide deaths and calculated the pooled rate ratio for mental illness antecedents (i.e., the rate of suicide deaths in persons with specific mental illnesses over the rate in those without those illnesses). They report a pooled rate ratio of 13.2 for psychotic disorders and 12.3 for mood disorders (e.g., depression).

An issue of concern with regard to the control and mitigation of suicide is access to professional mental health and related care. This issue has also been difficult to study, as "access to care" has varying meanings in research studies and operates at multiple levels for individuals. Variation in access may reflect the density of licensed therapists of various kinds in a geographic area; the presence of various community-based programs for treatment of general mental illness or more specific problems, such as substance use disorders; or inpatient hospital "beds" for management of more serious or immediate mental illnesses. Access may also refer to having personal or family insurance for mental health conditions. Even if such insurance

is present, however, it may have important limitations with respect to the services covered and/or a requirement for substantial copayments. Many mentally ill and suicidal patients seek care at general hospital emergency departments, where infrastructure for suicide management and prevention may not be adequate (Asarnow, Babeva, and Horstmann, 2017). Finally, access may refer to nonfiscal barriers, such as stigma or social impediments. As noted in Chapter 7, it is generally believed that personnel and facilities for adequate psychiatric care are lacking in many American regions and jurisdictions, and many counties have no mental health professionals whatsoever. The lack of access to mental health services is especially acute in the nonmetropolitan areas where male suicide mortality has been historically high (Andrilla et al., 2018).

An older review of 40 studies examined the relation between access to mental health care and suicide rates (Luoma, Martin, and Pearson, 2002). This multinational study found that only 19 percent and 32 percent of decedents, respectively, had made contact with the mental health care system in the month before death and in the year before death, highlighting the need for more attention to the role access to mental health care can play in reducing suicide mortality. Holliday (2018) conducted a more recent review and concluded that the evidence for this association was "mixed," noting that even if mental health services were proximately available, not all persons with mental illnesses might need them. However, she found that two policy-oriented studies offered more evidence that access to mental health care could yield some reduction in suicide rates. Using cross-sectional state-level data, Tondo, Albert, and Baldessarini (2006) found that states receiving more federal mental health aid had lower suicide rates, and aid was a stronger correlate of suicide rates than was the proportion of uninsured individuals in the state, density of psychiatrists or physicians, or sociodemographic variables. Lang (2013) examined the causal impact of mental health parity laws on suicide rates between 1990 and 2004; parity laws require health insurance plans to provide comparable coverage for physical and mental health. Using variation in implementation dates, Lang found that in the first year after states enacted parity laws, the suicide rate for adults ages 18–64 declined by 5 percent, and these effects were maintained 2 years or longer after the laws had been enacted, although the magnitude of the change decreased somewhat over time.

A paucity of research examines the relationship between mental illness and suicide mortality separately by race and ethnicity because of the limited data sources and measurement issues mentioned earlier. For example, race-specific suicide rates and rankings as determined by death certification may be affected differentially by different suicide methods (e.g., gunshot wound versus drug poisoning) (Warshauer and Monk, 1978) or the types of

opioids used by decedents with substance use disorders (Alexander, Kiang, and Barbieri, 2018). Nonetheless, the racial/ethnic patterns of mental health reflect the racial/ethnic patterns of suicide mortality. For example, there is evidence of higher lifetime risks of mental illness among Whites compared with Blacks and Asians (Alvarez et al., 2019). In what is known as the "minority mental health paradox," non-Whites report mental health that is equal to or somewhat better than that of Whites (Williams and Earl, 2007). The 12-month prevalence of most psychiatric disorders, including depression (Hasin et al., 2018), tends to be lower among non-Whites (Breslau et al., 2005; Miranda et al., 2008; Vilsaint et al., 2019). Once adjustments for socioeconomic status are included, Blacks almost always report better mental health on dimensional measures of depression (Barnes and Bates, 2017), while Hispanics report a lower prevalence (Breslau et al., 2005) and Asians an especially low prevalence (Takeuchi et al., 2007) of psychiatric disorders generally relative to Whites. It should be noted, however, that racial/ethnic minorities are significantly more likely than Whites to conceal their mental health symptoms and avoid treatment because of the stigma associated with mental illness (Clement et al., 2015); while findings are mixed on whether racial/ethnic minorities experience higher levels of stigma than Whites (Wong et al., 2017).

The racial gap in mental illness has sometimes been explained by differential levels of access to health care and treatment. African Americans receive fewer prescription medications, and even among insured patients with the same condition, Black and Hispanic patients use significantly fewer medications relative to White patients (Briesacher, Limcangco, and Gaskin, 2003). A recent study attempted to address the minority mental health paradox by examining a common argument that Whites compared with non-Whites have more access to and receive better health care, including for mental illness. Using data from the 2008–2013 Medical Expenditure Panel Survey, Schnittker and Do (2020) show that Whites consume more pharmaceuticals than non-Whites for a wide variety of medical conditions, and although these drugs are effective in treating their particular condition, depression or suicide can be a side effect. The authors report a strong relationship between the use of medications for which suicide is a potential side effect and significant distress, such that the disproportionate use of these medications by Whites partially explains the non-White advantage in mental distress. This study did not address whether the use of these medications is also associated with a higher suicide rate among Whites compared with non-Whites.

Some evidence points to a decline in mental health among individuals of low socioeconomic status since the mid-1990s. For example, Goldman, Glei, and Weinstein (2018) document a decline in life satisfaction, psychological

well-being, and positive affect[7] between the mid-1990s and 2011–2014 among middle-age and older Whites of low-socioeconomic status, the population subgroup that has experienced rising death rates from suicide (Case and Deaton, 2015, 2017, 2020; Goldman, Glei, and Weinstein, 2018). A caveat regarding the research in this area is that it tends to focus on declines in the positive manifestations of mental health, and it is not clear whether poor mental health increases in step with declining positive mental health (Cherlin, 2018). As noted in Chapter 7, Graham and Pinto (2019) found racial/ethnic differences in optimism for the period 2010–2015 using data from the Gallup Healthways Survey, which showed that lower-income Blacks and Hispanics had higher levels of optimism about their future life satisfaction[8] compared with lower-income Whites, especially Whites living in rural areas. The role of lower and declining psychological well-being among Whites of low socioeconomic status may be important given that greater psychological well-being has been associated with significantly lower overall mortality (Alimujiang et al., 2019; Keyes and Simoes, 2012; O'Connor and Graham, 2019) and with lower suicide mortality (Too et al., 2019).

Another factor that may play a role in suicide mortality is an increase in the reported prevalence of pain among U.S. adults. As noted in Chapter 7, a study using two cross-sectional waves of the Midlife in the United States (MIDUS) Study found an increase in the prevalence of pain between 1995–1996 and 2011–2014, reporting that pain accounted for an important share of the increase in drug misuse, often correlated with suicide, over this period (Glei, Stokes, and Weinstein, 2020). Additional evidence that chronic pain may have played some role in the surge in suicide mortality since the early 2000s comes from the NVDRS. Petrosky and colleagues (2018) used NVDRS data from 18 states on 123,181 decedents who had died from suicide from January 2003 through December 2014. They found that the percentage of suicides with evidence of chronic pain was up from 7.4 percent in 2003 to 10.2 percent in 2014.

With respect to physical illness, Stickley and colleagues (2020) found that persons with four or more such illnesses had 3–4 times greater odds of suicidal behavior compared with those with no such illnesses. In a multinational systematic review of antecedent physical illness and functional disability, suicidal behaviors were more common in patients with such disabling conditions as malignant diseases, liver disease, neurological disorders, male genital disorders, and arthritis. Because chronic illness and its

[7]How much of the time in past 30 days the respondent felt cheerful, in good spirits, extremely happy, calm and peaceful, satisfied, and full of life.

[8]Respondents were asked where on a ladder with a scale of 1–10 they thought their life satisfaction would be in 5 years.

comorbidities are associated with higher levels of physical and emotional pain (Fässberg et al., 2016; Racine, 2018), these findings resonate with the above trends in pain, especially for Whites, who tend to seek and receive more health care relative to Blacks and Hispanics (Institute of Medicine [IOM], 2003; Schnittker and Do, 2020). In general, however, less attention has been paid to the physical illness and multimorbidity antecedents of suicide than to pain, and it may be that further investigation would support screening of these patients for suicide risk, with subsequent management.

Increased mortality from drug- and alcohol-related causes among White Americans ages 25–64 is documented in Chapter 7. The fact that substance use is a risk factor for suicide accords with those findings, and several studies show that suicide is often associated with acute alcohol consumption (see Cherpitel, Borges, and Wilcox, 2004, for a review). Kaplan and colleagues (2015) document an increase in the percentage of suicides showing the presence of alcohol intoxication from 2005–2007 to 2010–2011 among most racial/ethnic groups, including White men and women. Acute alcohol intoxication may be a mechanism connecting worsening economic conditions to suicide (Kawohl and Nordt, 2020; Nordt et al., 2015). For example, Kaplan and colleagues (2015) and Kerr and colleagues (2017) show that alcohol ingestion itself (particularly acute alcohol intoxication) was a key risk factor for suicide during and following the Great Recession. Using data from the NVDRS, Kerr and colleagues (2017) also found a positive association between poverty and alcohol involvement in suicides in 2005–2011 for both males and females in all age groups.

SUMMARY

Trends and disparities in suicide mortality among U.S. working-age adults over the study period (1990–2017) indicate that suicide rates increased substantially mainly among Whites, especially White males. Although at lower levels, suicide rates also increased among working-age White women, especially since 2000. Although Black and Hispanic working-age adults generally experienced declining (1990s) and then steady (2000s and 2010s) trends in suicide deaths, the trends among those in the younger working-age group (25–44) showed a slight increase from 2012–2014 to the end of the period. Nevertheless, the rising trend among working-age Whites compared with the relatively flat trend among Blacks and Hispanics resulted in a widening racial/ethnic gap in suicide mortality during the study period. The nonmetro–metro gap also widened, especially among younger working-age (ages 25–44) White males, for whom suicide rates rose more slowly in the latter areas. Suicide rates also were higher in Western states, especially those with large rural populations, relative to other regions of the country.

The potential causes of rising suicide mortality among Whites are complex, involving multiple factors that operate independently and interactively across the societal, community, and individual levels to affect suicide risk. Research tends to focus in four general areas: economic factors; social engagement, religious participation, and social support; access to lethal means; and mental, emotional, and physical health. Unfortunately, little of this research examines differences by race and ethnicity, estimates causal impacts, or attempts to explain *change* in suicide mortality (mainly differential rates). Therefore, understanding of why working-age suicide rates increased among Whites during the study period is mainly inferential.

Some of the stronger evidence has been found for the role of economic conditions in suicide mortality. Periods of economic downturn, wage stagnation, weak safety nets, and increasing foreclosure rates are associated with rising suicide mortality in national- and state-level studies. While employment is protective against suicide, the negative effects of unemployment appear to be conditional on the context of economic insecurity. Because economic insecurity is more common among those with less education or household income, groups of lower socioeconomic status are especially sensitive to changes in economic conditions. On the other hand, the economic recovery from the Great Recession and low unemployment rates since 2016 have not benefited the less educated as much as those with a college degree, especially among Whites, who have not been able to rebound from periods of economic insecurity as they have in the past. Thus, deteriorating economic conditions among those without a college degree may be an important factor explaining rising suicide mortality among Whites, especially White men.

Extensive research documents the Durkheimian premise that social integration within institutions, communities, and friendship and family networks is protective against suicidal behavior and death, and descriptive evidence suggests that such social capital resources are associated with lower suicide rates among Whites. Whether levels of social engagement have changed in recent decades to bring about the rise in suicide mortality, however, is more difficult to determine. Evidence indicates that social integration has declined in recent decades so that social isolation and lack of social support have increased. Engagement in religious organizations, for example, an important factor related to lower suicide rates, declined in the past decade, especially among Whites without a college degree. Marriage, another social context that is protective against suicide risk, also declined over the past several decades for White women without a college degree in particular, following a similar trend in marriage among Blacks seen several decades earlier.

Although suicide mortality by firearms rose over the study period, its contribution to the rise in overall suicide mortality declined as suicides by

other means increased more rapidly. Thus access to lethal means of suicide can only partially explain rising suicide mortality among Whites. While there is evidence that more firearm-related suicides occur in states with looser gun regulations and greater gun ownership and are higher in nonmetropolitan versus large metropolitan areas, the proportion of all suicide deaths related to firearms declined from 1990 to 2017. Further research is needed on lethal means of suicide to better understand the increase in different suicide modalities, how they differ by sex, and what factors might precipitate the choices made. In particular, research on the role of gun control laws and gun availability in suicide mortality is warranted, with attention to the causal effect of changes in gun control laws and gun availability on trends in suicide mortality.

Within the category of mental, emotional, and physical health, factors identified as especially relevant to suicide mortality are life-course traumas and stressors, especially those that occur early in life as adverse childhood experiences, and mental illness. Not surprisingly, those with a history of mental illness have a much higher risk of suicide. Poor access to mental health care may explain this relationship, although the evidence is inconclusive given the many different forms of access that are typically not measured in the databases available for study (e.g., density of licensed therapists, community-based programs, insurance, hospital emergency rooms). The important role of mental illness in explaining the rising trend in suicide mortality among Whites is supported by the concordance between the racial/ethnic patterns of mental health and the racial/ethnic patterns of suicide mortality. Whites have higher levels of lifetime mental illness relative to Blacks, Hispanics, or Asians, and this racial gap may be explained by differential levels of access to health care and treatment. Not only does better access to health care provide more opportunity to be diagnosed with a mental illness, but it also makes Whites more likely to receive prescription medications relative to Black or Hispanic adults, medications that sometimes induce depression or even suicide as a side effect, which might partially explain Whites' higher rates of mental distress relative to non-Whites. Comorbidities related to physical illnesses, disabilities, and drug and alcohol use also contribute to levels of mental illness and pain, all of which represent important predisposing factors to suicide.

While the research literature provides some compelling evidence for links between changes in economic conditions, social integration, and psychological and physical well-being and the rise in suicide mortality among Whites, most of this evidence is suggestive and obscures the fact that these factors are interrelated and operate across the societal, community, and individual levels. For example, employment provides economic security, which reduces suicide risk, but employment also provides work-based social

networks, access to health information, health benefits, a sense of self-worth, instrumental social capital, health care, and psychological well-being, all of which are protective against suicide. Better understanding of the key factors involved and improved data explaining the recent rise in suicide mortality will require research in several areas, highlighted below.

IMPLICATIONS FOR RESEARCH AND POLICY

Because suicide rates increased from 1990 to 2017 mainly among Whites, research needs to focus on what changed for Whites, and why changes in economic conditions, social integration, or mental health and access to mental health care appear to have affected their suicidal behavior differently relative to other racial/ethnic groups. This information may be fruitful in understanding the smaller, but still concerning, recent increases in suicide among Black and Hispanic young adults. Although evidence indicates that recent trends in religious involvement, marriage rates, experience with pain, and psychological well-being coincided with rising rates of suicide among working-age Whites, especially those of low socioeconomic status, research has yet to forge explicit links between macro-, community-, family-, and individual-level trends and changes in suicide rates by sex, race and ethnicity, socioeconomic status, or geography. For example, the continued rise in suicide rates over the past several years among White Americans despite low unemployment and the prolonged economic expansion following the Great Recession points to the need to consider differential impacts of economic restructuring on population subgroups and geographic regions of the United States.

Prior studies have relied on either individual-level data (e.g., NHIS, NVDRS, NLMS) or aggregate-level data, typically at either the county or state level. Studies combining both of these types of data could help elucidate the relative importance of individual- and contextual-level factors and their possible interactions in suicide trends, with attention to the role of both protective and predisposing characteristics. For example, a multilevel study design would enable researchers to disentangle the effects of individual-level economic status, such as unemployment status and poverty, from those of upstream community-level economic conditions, such as declining manufacturing industries. Such a design could also help determine whether changes in the latter economic conditions interact with individual-level family characteristics, such as marital status and household income. These studies would benefit from considering geographic measures of social and economic factors from large social science surveys, such as the American Community Survey, and from continued linkage of the NHIS and the NLMS to the National Death Index (NDI).

A paucity of research on factors related to suicide mortality addresses differences by sex, race and ethnicity, socioeconomic status, and geography. Growing evidence suggests that the economic insecurity that characterizes the lives of population subgroups of lower socioeconomic status and with less education is linked to suicide mortality, but Whites of low socioeconomic status appear to be more vulnerable in this regard relative to their non-White counterparts. Given that the recent rise in suicide mortality has been driven mainly by Whites in nonmetro areas, understanding why the same economic, social, and geographic factors associated with rising suicide rates among Whites are not related to rising rates among other groups could provide insights into how to reduce suicide among Whites. Moreover, recent trends showing a slight rise in suicide deaths among Black and Hispanic young adults (ages 25–45) call for research on how the economic and social factors associated with suicide operate differently by sex, race and ethnicity, and geography in order to identify modifiable factors that can shed light on factors contributing to the rise among younger Blacks and Hispanics and help reduce disparities in suicide mortality. As noted in Chapter 7 (see Recommendation 7-3) and elaborated in Chapter 11 (see Recommendation 11-6), using a multilevel study design that combines aggregate- with individual-level data to uncover the individual-, meso-, and macro-level explanations for changes in mortality will advance research on suicide and the other major causes of death that this report documents as contributing to the recent rise in working-age mortality. Similarly, understanding how changes in suicide and other causes of death vary by sex, race and ethnicity, socioeconomic status, and geography is imperative for uncovering protective and predisposing factors unique to specific population subgroups to inform policies aimed at reducing disparities in mortality.

Accordingly, the committee developed a cross-cutting recommendation calling for quantitative and qualitative studies that would aid in assessing the role of upstream versus downstream factors in suicide risk and uncover the multilevel mechanisms that explain the demographic and geographic differences and temporal changes in suicide rates (see Chapter 11, Recommendation 11-6; see also Recommendation 7-3 in Chapter 7). There is a critical need for research on the role of access to mental health care in rising trends and disparities in suicide mortality (see also Chapter 7 and Recommendations 7-2 and 7-5). The lack of detail and specificity that characterizes existing research hampers the development of prevention strategies designed to improve access to mental health care and treatment for vulnerable populations, including non-Whites but also Whites of lower socioeconomic status. For example, measures and data regarding types of access and treatment are inconsistent and incomplete. In addition, research is needed on the geographic distribution and availability of mental health

services, substance use treatment programs, and emergency medical services that can play an important role in mental health treatments and suicide prevention (see Recommendations 7-2 and 7-5 in Chapter 7).

The findings presented in this chapter also point to the need for additional research on lethal means of suicide. Although rates of firearm-related suicide are higher in states with looser gun control regulations and greater gun ownership, the share of all suicides due to firearms declined during 1990–2017, while the share due to hanging, suffocation, and strangulation increased. More needs to be known about trends in different suicide modalities, variations by sex, and the causal role of access to and availability of firearms in trends in suicide mortality.

RECOMMENDATION 8-1: Federal agencies, in partnership with private foundations and other funding entities, should support research on lethal means of suicide aimed at better understanding the increase in use of different suicide modalities, how modalities differ by sex, and what factors might precipitate the choices made. Research on the role of gun control laws and gun availability is particularly warranted, with attention paid to the causal effect of changes in gun control laws and gun availability on trends in suicide mortality.

Studies taking a life-course approach to the study of mental health and suicide mortality using longitudinal data are also needed. Longitudinal data enable researchers to track predisposing and protective factors as they unfold across the life course to identify the most vulnerable life stages in the development of suicide behavior and deaths. Research taking this approach could answer such questions as the role of early-life and adolescent environments in predicting adult mental health outcomes, the extent to which those environments are moderated by adult characteristics and exposures, and whether these factors interact in their influence. Such datasets could also be used to assess childhood, adolescent, and adult precursors associated with suicide, including pain and disability. Several longitudinal studies that follow individuals over time, such as the National Longitudinal Study of Adolescent to Adult Health (Add Health), the Panel Study of Income Dynamics, the Wisconsin Longitudinal Study, MIDUS, and the Health and Retirement Study link their data to the NDI. Add Health, which has followed its respondents from adolescence into midlife, conducts ongoing death surveillance of its sample that includes the collection of death certificates to obtain primary and secondary underlying causes of death, hospital records, and interviews with next of kin for all decedents. To advance research in this area, all longitudinal studies, especially studies of early life and young adulthood, when mental health patterns are often established,

should routinely link their data to the NDI. Funders of longitudinal studies, especially the National Institutes of Health, could be instrumental in this regard by facilitating the regular linking of longitudinal study data through its administrative relationship with the NDI.

RECOMMENDATION 8-2: Directors and funders of longitudinal studies should routinely link these survey data to the National Death Index to support a life-course approach to the study of mental health and suicide mortality.

9

Cardiometabolic Diseases

Deaths due to cardiometabolic diseases include the following cause-of-death categories: *endocrine, nutritional, and metabolic (ENM) diseases* (e.g., thyroid conditions, diabetes, hyperlipidemia, obesity); *hypertensive heart disease* (e.g., heart disease caused by prolonged exposure to high blood pressure); and *ischemic heart disease and other diseases of the circulatory system* (e.g., reduced blood supply to the heart, including atherosclerosis and coronary heart disease (CHD), stroke, and other cardiovascular conditions). Collectively, cardiometabolic diseases were responsible for more than 4.8 million deaths among the U.S. working-age (ages 25–64) population between 1990 and 2017 (Centers for Disease Control and Prevention [CDC], 2020b). ENM diseases accounted for 703,247 deaths; hypertensive heart disease for 360,309 deaths; and ischemic heart disease and other diseases of the circulatory system for the largest share, 3,782,186 deaths (CDC, 2020b).

The contribution of cardiometabolic mortality to the recent rise in working-age mortality is complex and involves several countervailing trends. Death rates due to ENM diseases and hypertensive heart disease generally increased from 1990 to 2017, especially since 2010. While significant long-term reductions in mortality from ischemic heart disease and other diseases of the circulatory system had occurred since 1990, much of that progress appears to have stalled after 2010. The combination of these trends operated to increase all-cause working-age mortality after 2010 because the slowdown in declines in mortality from ischemic heart disease and other circulatory diseases no longer offset the rise in mortality from ENM diseases and hypertensive heart disease.

Within the working-age population, certain subgroups experienced greater relative increases in mortality from ENM diseases and hypertensive heart disease over the past three decades and slower declines in mortality from ischemic heart disease and other circulatory diseases in the recent decade—notably younger adults (ages 25–44) of all racial/ethnic groups, non-Hispanic (NH) White (White) males and females, NH Black (Black) males (in the recent decade), and those living in rural areas (exceptions are detailed in the next section). These troubling changes in cardiometabolic mortality have been most pronounced in the South and in nonmetropolitan areas.

Among the potential explanations for these patterns are three relevant trends: the obesity epidemic; diminishing returns of medical advances; and social, economic, and cultural changes. While all three sets of factors played some role in cardiometabolic mortality trends, the evidence suggests that rising obesity—an epidemic that has now spanned four decades—has exerted the greatest influence. The evidence also shows that younger adults, particularly those born in the late 1970s and early 1980s, who have been exposed to obesogenic environments for their entire lives, have experienced more adverse endocrine, metabolic, and cardiovascular consequences relative to older adults who averted this exposure as children and young adults. Moreover, vulnerable populations (racial/ethnic minorities, less-educated Whites, and rural populations) experienced higher rates of obesity and chronic stress due to social and economic conditions, together with less access to effective medical interventions for cardiometabolic disease, contributing to persistent disparities in cardiometabolic mortality across population subgroups and geographic areas. This chapter first summarizes the trends in cardiometabolic mortality by age, sex, race and ethnicity, and geography, and then reviews the evidence salient to explaining changes and disparities in these trends. The chapter ends with several recommendations for future research and policy evaluations.

TRENDS IN CARDIOMETABOLIC MORTALITY

Endocrine, Nutritional, and Metabolic Diseases

During the study period (1990–2017), trends in mortality from ENM diseases were largely similar among working-age males and females within each age group (Figure 9-1). Among younger working-age adults (25–44) mortality from these diseases increased slowly throughout the 2000s and 2010s, with the largest increases occurring among Black males. Among older working-age adults (45–64), Whites experienced gradual increases in mortality from ENM diseases throughout the period, while trends among Blacks and Hispanics were marked by decreasing mortality in the 2000s

and a subsequent leveling off and/or increase during the 2010s. In every age group, the largest increase in ENM mortality in the 2010s occurred among Black males.

Differences between White and Hispanic adults in ENM mortality were minimal among those ages 25–54. Among older adults (ages 55–64), Hispanics experienced higher mortality than their White counterparts in the 1990s, but this gap began to narrow in the 2000s and had disappeared by the end of the study period primarily because of declining ENM mortality among Hispanics. Although working-age Blacks experienced larger changes in mortality from ENM diseases over time, these changes preserved the sizable racial inequalities in mortality from these diseases as the rates for Blacks remained roughly twice those for Whites and Hispanics throughout the period (Figure 9-1).

Working-age adults living in large central metropolitan areas (hereafter referred to as "large central metros") generally experienced lower mortality from ENM diseases relative to those in less-populated areas throughout the study period (Figure 9-2). Mortality in nonmetropolitan areas (hereafter referred to as "nonmetros") was not always the highest, but it increased more rapidly (or decreased more slowly) than in other areas throughout the 1990s. By 2000–2002, mortality from ENM diseases was highest in nonmetros, and this continued to be the case throughout the remainder of the study period among all working-age adults except Black males ages 25–44. The largest increases among these younger Black males occurred in small and medium metropolitan areas (hereafter referred to as "small/medium metros"). The net result was that disparities across metropolitan areas widened over time, particularly among Whites and Blacks.

At the state level, mortality from ENM diseases increased among males in all but two states (New Jersey and Vermont) and the District of Columbia, with particularly large increases in several Southern and Western states and in South Dakota (Figure 9-3). Among females, the rates increased in 38 states. There was very little change in the rates across the Northeastern states, but small increases occurred across most of the Midwest and West, and comparatively large increases occurred in several Southern states, most notably West Virginia, Mississippi, Arkansas, Kentucky, and Oklahoma. Among both males and females, there were relatively large declines in the District of Columbia.

Hypertensive Heart Disease

White adults across all working ages experienced increased mortality from hypertensive heart disease during the study period, while the trends among Hispanic and Black adults differed by age and sex (Figure 9-4). Although rates of mortality due to hypertensive heart disease were lower

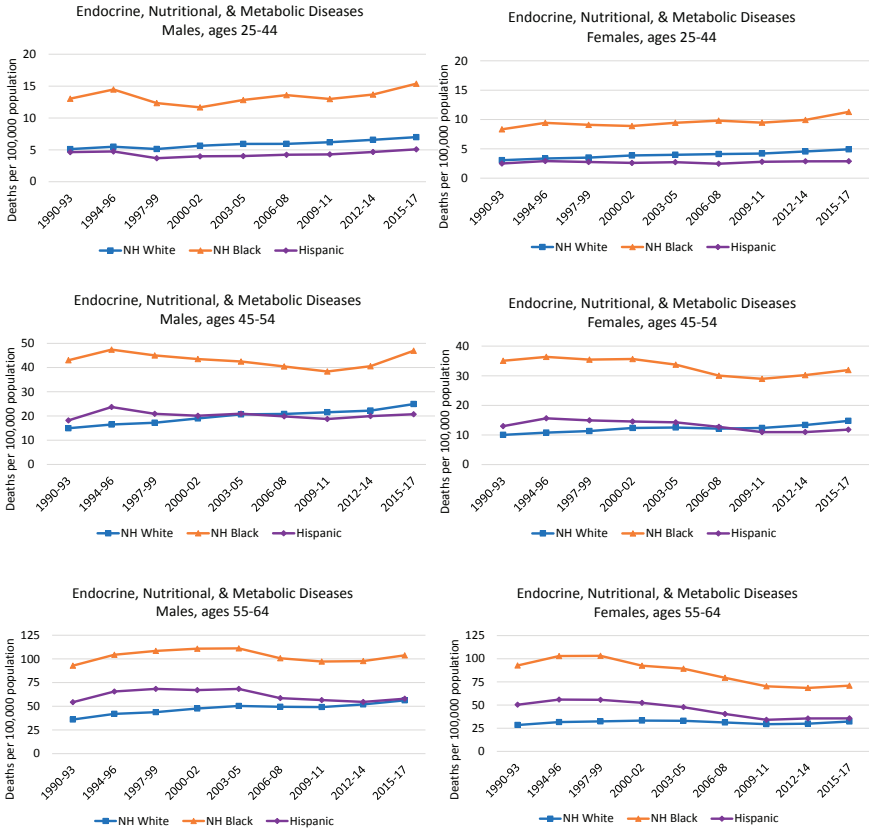

FIGURE 9-1 Mortality rates (deaths per 100,000 population) from endocrine, nutritional, and metabolic diseases among U.S. working-age males and females (ages 25–64), 1990–2017, by sex, age, and race and ethnicity.
NOTE: Each panel shows mortality rates for non-Hispanic (NH) Whites (blue line), NH Blacks (orange line), and Hispanics (purple line). Mortality rates for males are shown in the lefthand panels, while those for females are shown in the righthand panels. Mortality rates are shown for three age groups: 25–44 (top panels), 45–54 (middle panels), and 55–64 years (bottom panels). Rates are age-adjusted to reflect a standard population age distribution.
SOURCE: Data from National Vital Statistics System Detailed Mortality Files, https://www.cdc.gov/nchs/nvss/deaths.htm.

FIGURE 9-2 Mortality rates (deaths per 100,000 population) from endocrine, nutritional, and metabolic diseases among U.S. working-age males and females (ages 25–64), 1990–1993 through 2015–2017, by metropolitan status.
NOTE: Mortality rates are shown for those ages 25–44 (panels a-c and g-i) and 45–64 (panels d-f and j-l) across four levels of metropolitan status: (1) large central metropolitan areas (blue lines), (2) large fringe metropolitan areas (orange lines), (3) small or medium metropolitan areas (gray lines), and (4) nonmetropolitan areas (yellow lines). Trends in these four groups are presented separately by sex (males in panels a-f, females in panels g-l) and for non-Hispanic (NH) Whites (panels a, d, g, and j), NH Blacks (panels b, e, h, and k), and Hispanics (panels c, f, i, and l). Rates are age-adjusted by 10-year age group.
SOURCE: Data from https://www.cdc.gov/nchs/nvss/nvss-restricted-data.htm.

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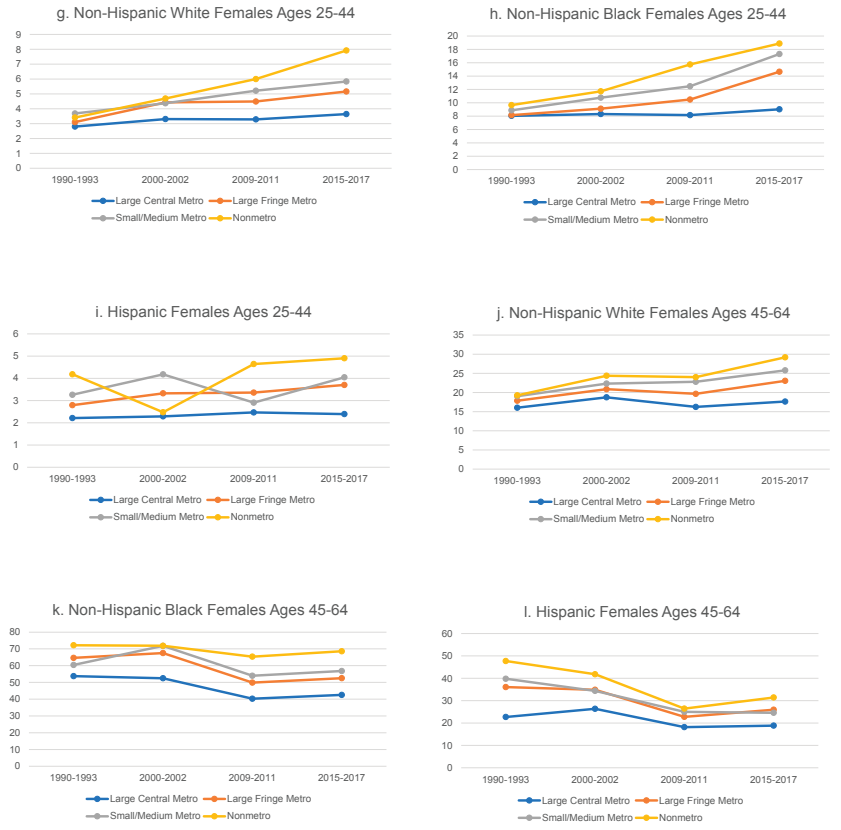

FIGURE 9-2 Continued

among younger (ages 25–44) than older (ages 45–64) adults, they increased slowly in this younger age group throughout the period among White and Black males and females and Hispanic males, with the largest increases occurring in the early 2000s and the most recent period, from 2012–2014 to 2015–2017; the increases were greater among males than females and among Blacks in this age group. Mortality rates among Hispanic females in all age groups remained stable over the study period. Older Black females ages 55–64 were the only group of working-age adults to experience declining mortality from hypertensive heart disease over the period, although these declines slowed in the 2010s and trended slightly upward after 2012. Among Black males ages 45–64 and Black females ages 45–54, mortality due to hypertensive heart disease showed small fluctuations during the 1990s and 2000s before stagnating (Black females) or increasing slightly (Black males) in the 2010s.

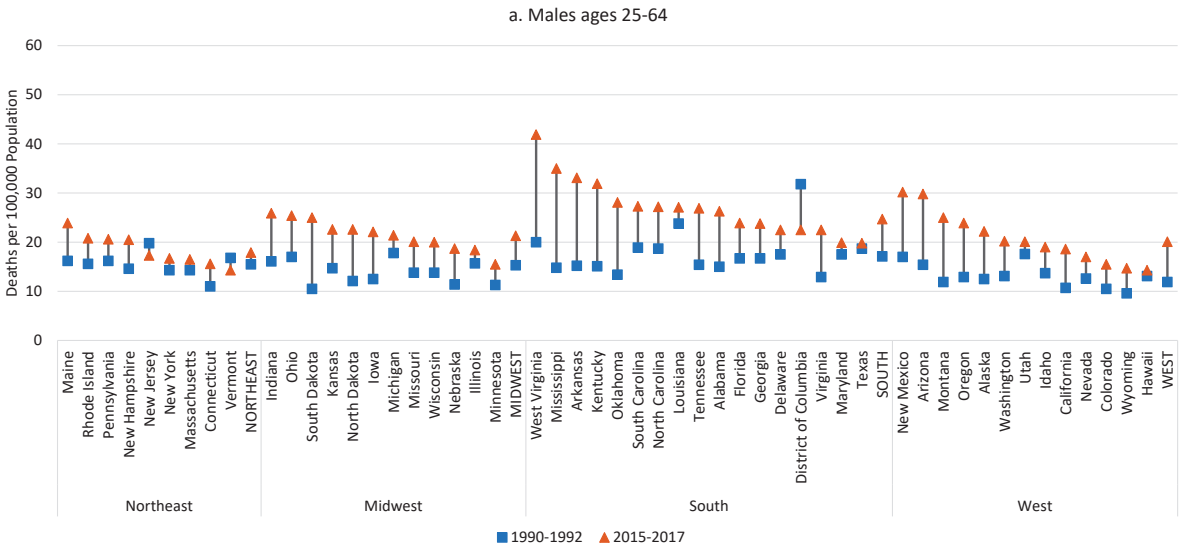

FIGURE 9-3 Mortality rates (deaths per 100,000 population) from endocrine, nutritional, and metabolic diseases among U.S. working-age males and females (ages 25–44), 1990–1992 and 2015–2017, by region and state.

NOTE: Mortality rates are shown for 1990–1992 (blue squares) and 2015–2017 (orange triangles), along with the changes over time (black connecting lines). Mortality rates for males are shown in panel a, while those for females are shown in panel b. Rates are age-adjusted by 10-year age band. For males, the 1990–1992 rate for Alaska represents 1991 and 1992 only; the rate was suppressed for 1990. The District of Columbia is excluded for females for the same reason. States are ordered from highest to lowest mortality rate in 2015–2017 within region.

SOURCE: Data from CDC WONDER Online Database, https://wonder.cdc.gov.

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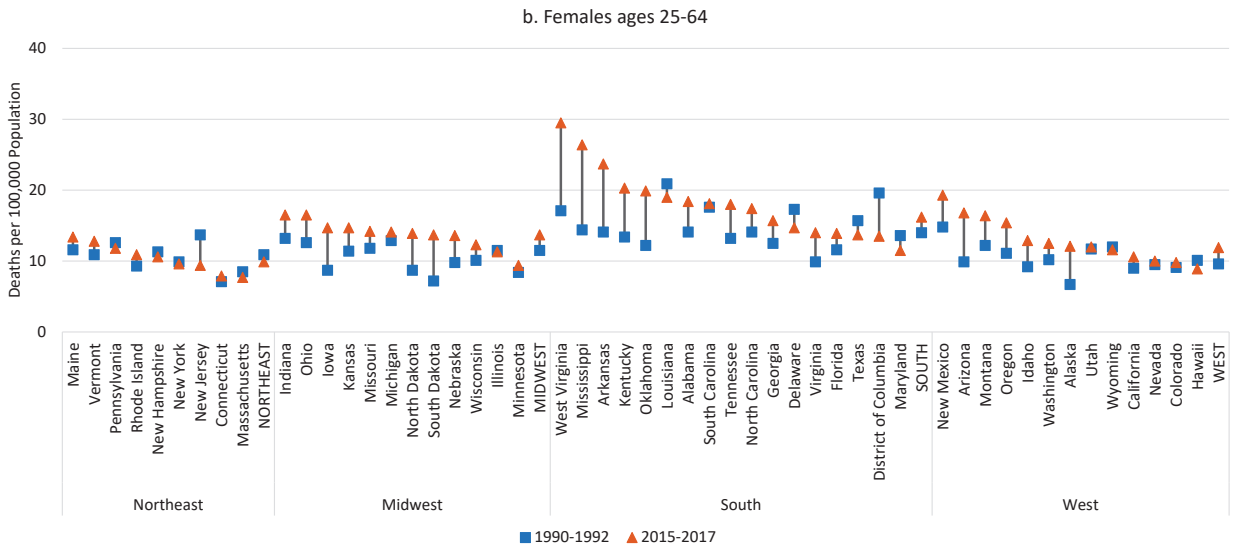

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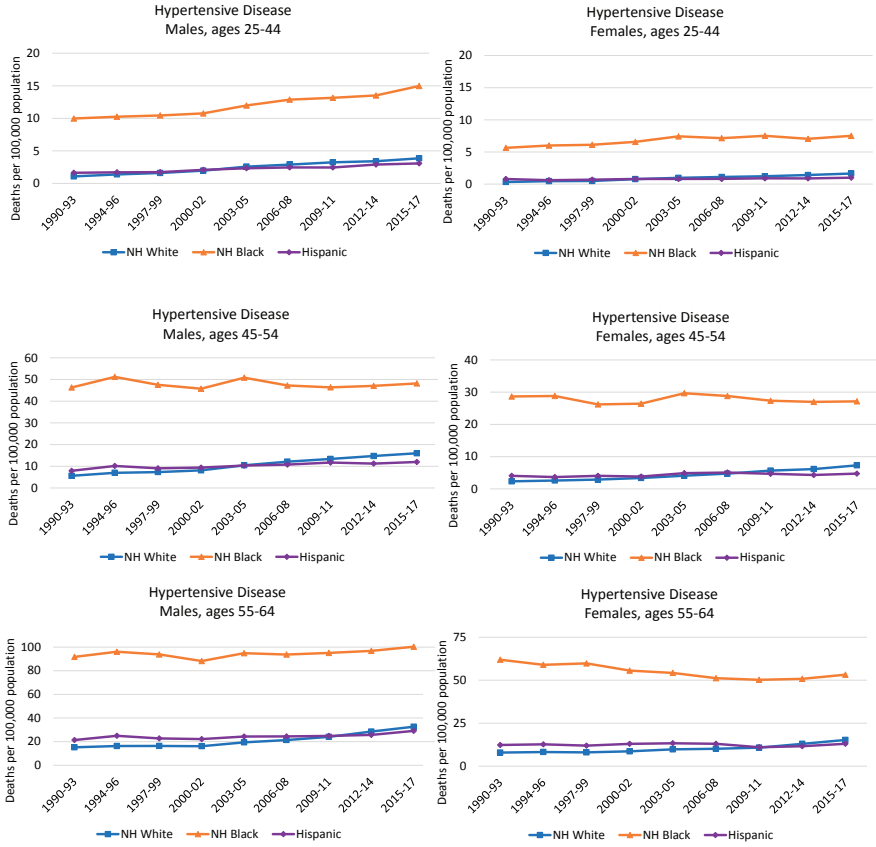

FIGURE 9-4 Mortality rates (deaths per 100,0000 population) from hypertensive heart disease among U.S. working-age males and females (ages 25–64), 1990–2017, by sex, age, and race and ethnicity.

NOTE: Each panel shows mortality rates for non-Hispanic (NH) Whites (blue line), NH Blacks (orange line), and Hispanics (purple line). Mortality rates for males are shown in the lefthand panels, while those for females are shown in the righthand panels. Mortality rates are shown for three age groups: 25–44 (top panels), 45–54 (middle panels), and 55–64 years (bottom panels). Rates are age-adjusted to reflect a standard population age distribution.

SOURCE: Data from National Vital Statistics System Detailed Mortality Files, https://www.cdc.gov/nchs/nvss/deaths.htm.

Throughout the study period, working-age mortality from hypertensive heart disease was more than three times higher among Blacks than among Whites and Hispanics. Because mortality increased more quickly among younger (ages 25–44) Black adults than among their White and Hispanic peers, the mortality gap widened in this age group. However, the Black–White mortality gap narrowed slightly among older working-age adults (ages 45–64), as mortality increased among Whites but declined or stagnated among Blacks. While working-age White and Hispanic adults experienced similar mortality rates throughout the period, the mortality rates in 1990–1993 were slightly higher among Hispanic adults in every age group, but by 2015–2017, the rates were slightly higher among White adults.

While the substantive differences are not large, interesting patterns emerge when these trends are examined separately by metropolitan status (Figure 9-5). Mortality from hypertensive heart disease in large central metros was highest in 1990–1993 but increased less than in other areas, while mortality in nonmetros increased more rapidly. In 1990–1993, large central metros had the highest mortality from hypertensive heart disease among White adults and nonmetros the lowest, the exception being White females ages 25–44, among whom mortality did not differ by metropolitan status. During the 2010s, however, mortality from hypertensive heart disease increased sharply in nonmetro areas, while large central metros experienced much smaller increases. By 2015–2017, mortality from hypertensive heart disease among working-age Whites was highest in nonmetros and lowest in large central metros. The exception was mortality among older White males (ages 45–64), which ended the period with little difference across metro status because mortality in nonmetros did not increase as sharply in this population.

The same trends occurred among working-age Black males (higher mortality in large central metros in 1990–1993 and smaller increases, or larger decreases, over time than in other areas), reducing the gap in mortality by metro size. Among older Black females, the overall declines in mortality from hypertensive heart disease that occurred in the 1990s and 2000s were driven primarily by decreasing mortality in large central metros; mortality stagnated or increased elsewhere. Despite the favorable trends in large central metros; mortality remained highest in these areas for most working-age Blacks. The exception was younger working-age Black females, among whom mortality from hypertensive heart disease increased within small/medium metros and large fringe metropolitan areas (hereafter referred to as "large fringe metros"), but decreased within both nonmetros and large central metros in the 2010s.

Mortality from hypertensive heart disease among older working-age Hispanics (ages 45–64) deviated from the overall trend among older males and females. Among older Hispanic males, mortality increased most rapidly

FIGURE 9-5 Mortality rates (deaths per 100,000 population) from hypertensive heart disease among U.S. working-age males and females (ages 25–64), 1990–1993 through 2015–2017, by metropolitan status.

NOTE: Mortality rates are shown for those ages 25–44 (panels a-c and g-i) and 45–64 (d-f and j-l) across four levels of metropolitan status: (1) large central metropolitan areas (blue lines), (2) large fringe metropolitan areas (orange lines), (3) small or medium metropolitan areas (gray lines), and (4) nonmetropolitan areas (yellow lines). Trends in these four groups are presented separately by sex (males in panels a-f, females in panels g-l) and for non-Hispanic (NH) Whites (panels a, d, g, and j), NH Blacks (panels b, e, h, k), and Hispanics (panels c, f, i, and l). Rates are age-adjusted by 10-year age group.

SOURCE: Data from https://www.cdc.gov/nchs/nvss/nvss-restricted-data.htm.

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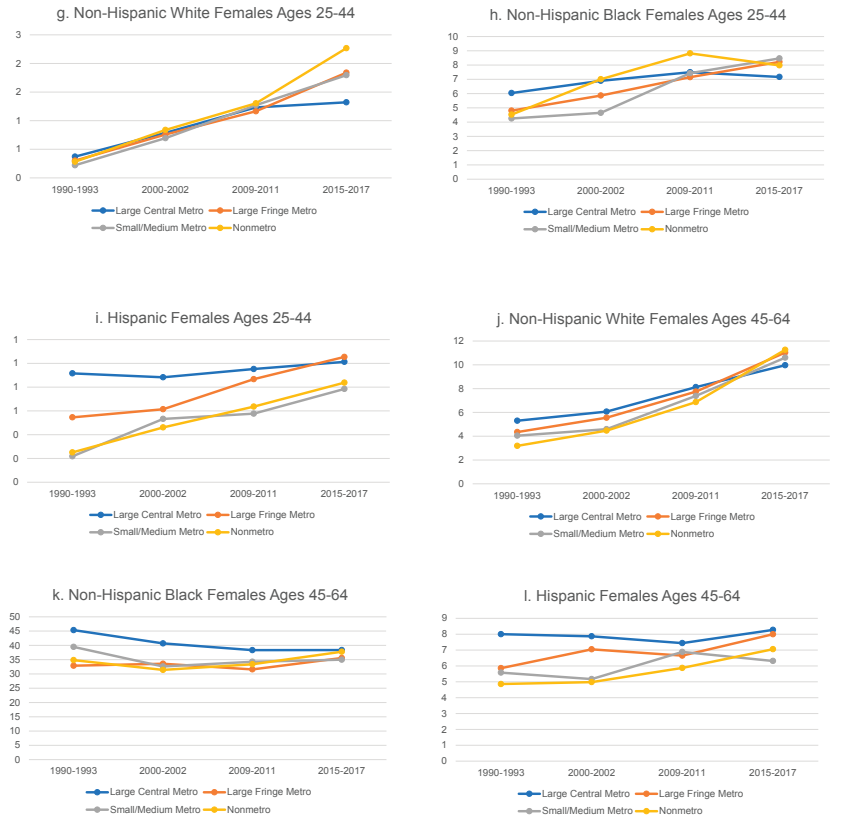

FIGURE 9-5 Continued

in large fringe metros; by 2015–2017, the mortality rates were similar in large central metros and large fringe metros and lower in nonmetros and small/medium metros. Among older Hispanic women, mortality from hypertensive heart disease remained steady throughout the study period, a pattern driven by mortality trends in large central metros. Outside of large central metros, mortality in this population increased between 1990 and 2017. Even with these increases, however, mortality from hypertensive heart disease remained relatively low among Hispanic men and women.

At the state level, mortality from hypertensive heart disease increased among males in every state (Figure 9-6), a pattern of state-level consistency found for only two other causes of death (drug poisoning and liver cancer). Mortality among females increased in every state but New York (where the rate of mortality from hypertensive heart disease, the highest in the

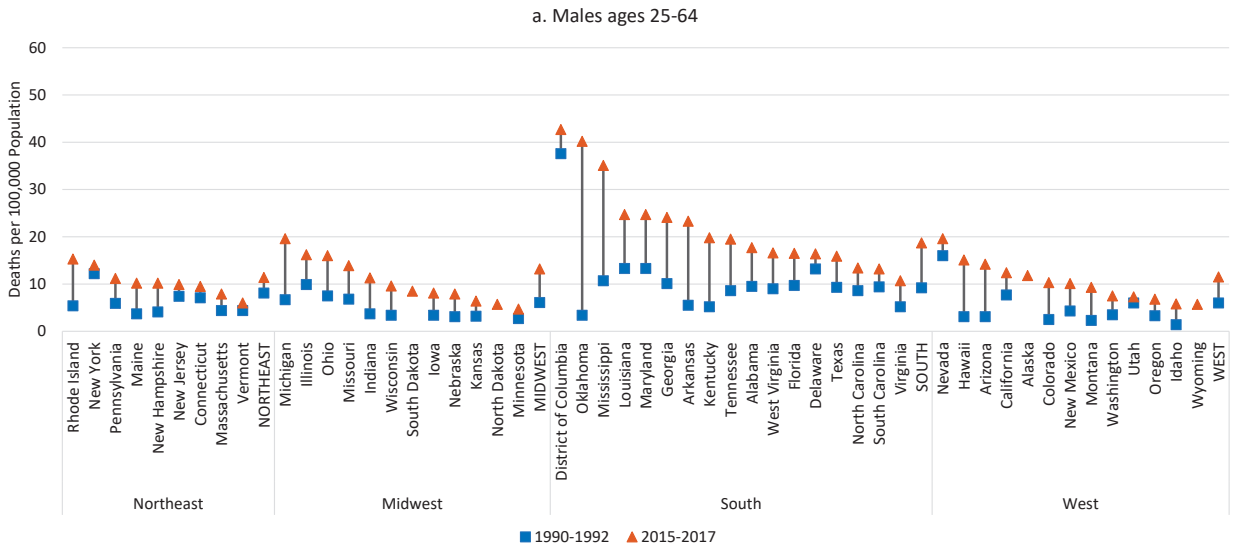

FIGURE 9-6 Mortality rates (deaths per 100,000 population) from hypertensive heart disease among U.S. working-age males and females (ages 25–44), 1990–1992 and 2015–2017, by region and state.

NOTE: Mortality rates are shown for 1990–1992 (blue squares) and 2015–2017 (orange triangles), along with changes over time (black connecting lines). Mortality rates for males are shown in panel a, while those for females are shown in panel b. Rates are age-adjusted by 10-year age band. For males, the 1990–1992 rates were suppressed for North Dakota, South Dakota, Alaska, and Wyoming. For females, the 1990–1992 rates were suppressed for North Dakota, South Dakota, Alaska, and Wyoming, Montana, and Idaho. Rates for females in both 1990–1992 and 2015–2017 were suppressed for Vermont. States are ordered from highest to lowest mortality rate in 2015–2017 within region.

SOURCE: Data from CDC WONDER Online Database, https://www.cdc.gov/nchs/nvss/deaths.htm.

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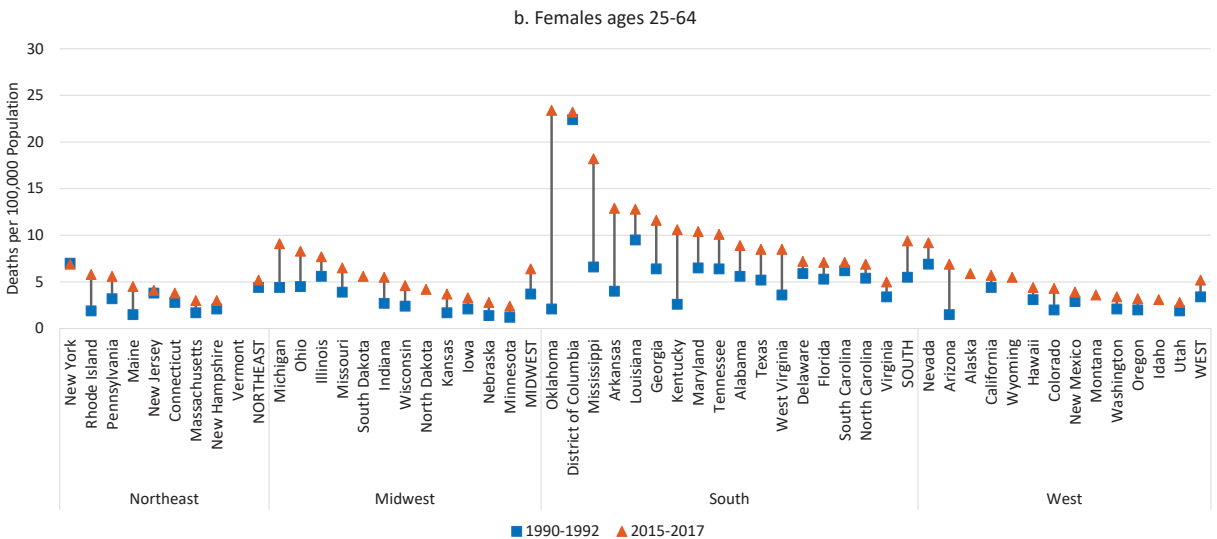

FIGURE 9-6 Continued

Northeast region, remained virtually unchanged). The largest increases and highest mortality rates occurred in several Southern states (notably Oklahoma, Mississippi, Arkansas, and Kentucky). In contrast, mortality from hypertensive heart disease changed comparatively little in the Northeast. Although increases among both males and females were small in the District of Columbia, it maintained the highest rate of mortality from hypertensive disease for males and the second highest for females (after Oklahoma) in 2015–2017.

Ischemic Heart Disease and Other Circulatory System Diseases

By far the largest contributor to the recent plateau in all-cause mortality among working-age adults was the slowing pace of improvements in mortality from ischemic heart disease and other circulatory diseases. The remarkable declines in mortality from these diseases seen throughout the 1990s and 2000s stalled in the 2010s (Figure 9-7).[1] Working-age Blacks had much higher rates of mortality from ischemic heart disease and other circulatory diseases relative to their White and Hispanic counterparts, although they also experienced the largest reductions between 1990 and 2017. The more rapidly declining death rates among Blacks reduced the mortality gap between them and both Hispanics and Whites over time. Although Hispanic adults had lower mortality from these diseases relative to White or Black adults, the trends among Whites and Hispanics were similar over time. The flattening of the mortality trend in ischemic heart disease and other circulatory diseases after 2010 was similar across all working-age adults.

Perhaps because ischemic heart disease and other circulatory diseases are a leading cause of death, differences in mortality from this category of causes by metropolitan status (Figure 9-8) largely mirror the patterns noted earlier for all-cause mortality. That is, mortality from ischemic heart disease and other circulatory diseases was highest in nonmetros and lowest in large central metros throughout the study period. Among working-age

[1]The plateau in mortality from ischemic heart disease and other circulatory system diseases obscures continued progress in lowering the death rate for ischemic heart disease—progress that was offset by increasing death rates from other circulatory diseases. As detailed in Chapter 4, this latter category of miscellaneous causes of death from circulatory diseases spans International Classification of Diseases (ICD)-10 codes I00–I09, I26–I28, I30–I51, I60–I69, I70–I78, I80–I89, and I95–I99 and includes deaths from arrhythmias, cardiomyopathy, heart failure, cardiac arrest, myocarditis, and valvular and pericardial disease. Whereas age-adjusted working-age mortality from these causes increased between 2011 and 2018 (from 29.9 to 32.2 per 100,000 population), mortality from ischemic heart disease decreased (from 38.6 to 35.2 per 100,000 population) (CDC, 2018). The combination of these trends produced what appears to be a plateau and belies a mixed picture of progress and setbacks for this category of cardiometabolic mortality (Shah et al., 2020).

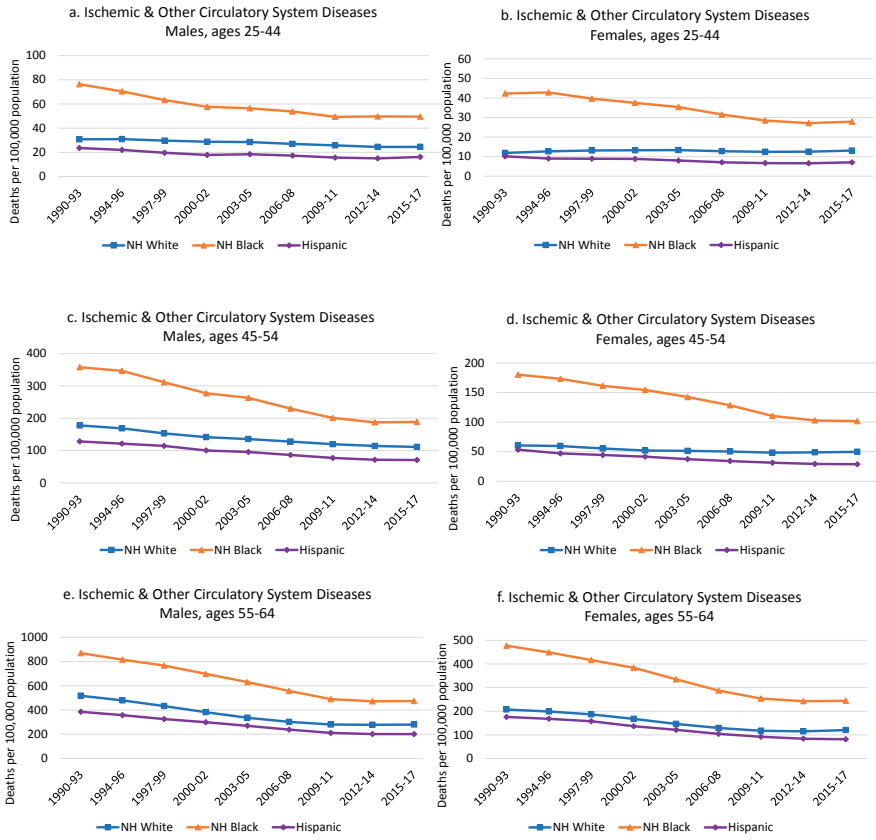

FIGURE 9-7 Mortality rates (deaths per 100,000 population) from ischemic heart disease and other circulatory system diseases among U.S. working-age males and females (ages 25–64), 1990–2017, by sex, age, and race and ethnicity.
NOTE: Each panel shows mortality rates for non-Hispanic (NH) Whites (blue line), NH Blacks (orange line), and Hispanics (purple line). Mortality rates for males are shown in the lefthand panels, while those for females are shown in the righthand panels. Mortality rates are shown for three age groups: 25–44 (top panels), 45–54 (middle panels), and 55–64 years (bottom panels). Rates are age-adjusted to reflect a standard population age distribution.
SOURCE: Data from National Vital Statistics System Detailed Mortality Files, https://www.cdc.gov/nchs/nvss/deaths.htm.

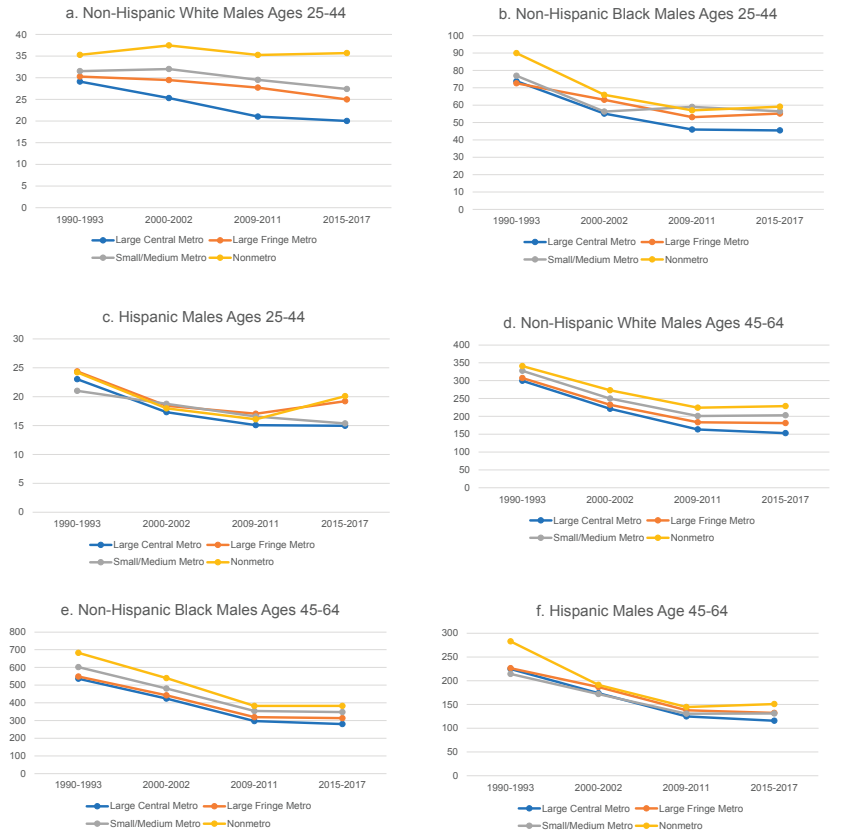

FIGURE 9-8 Mortality rate (deaths per 100,000 population) from ischemic heart disease and other diseases of the circulatory system among U.S. working-age males and females (ages 25–64), 1990–1993 through 2015–2017, by metropolitan status.
NOTE: Mortality rates are shown for those ages 25–44 (panels a-c and g-i) and 45–64 (panels d-f and j-l) across four levels of metropolitan status: (1) large central metropolitan areas (blue lines), (2) large fringe metropolitan areas (orange lines), (3) small or medium metropolitan areas (gray lines), and (4) nonmetropolitan areas (yellow lines). Trends in these four groups are presented separately by sex (males in panels a-f, females in panels g-l) and for non-Hispanic (NH) Whites (panels a, d, g, and j), NH Blacks (panels b, e, h, and k), and Hispanics (panels c f, i, and l). Rates are age-adjusted by 10-year age group.
SOURCE: Data from https://www.cdc.gov/nchs/nvss/nvss-restricted-data.htm.

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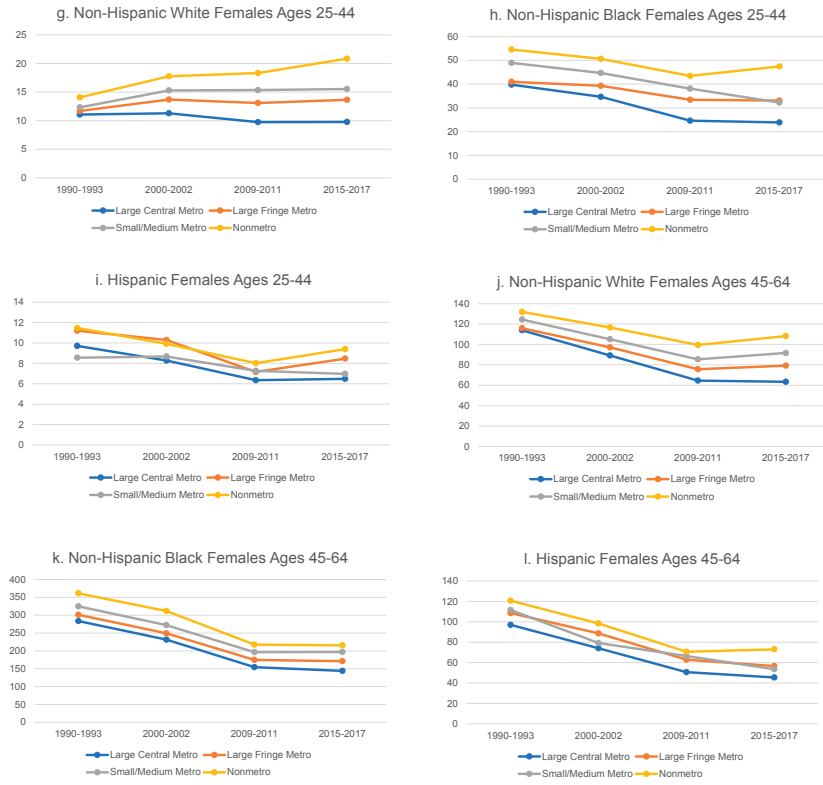

FIGURE 9-8 Continued

White adults, disparities between large central metros and nonmetros widened, just as they did for all-cause mortality. They widened among younger working-age White adults (ages 25–44) because mortality decreased in large central metros but increased elsewhere, particularly in nonmetros. Mortality also declined among older working-age Whites (ages 45–64), but these declines were largest within large central metros and smallest within nonmetros. Among younger working-age Blacks and Hispanics, the mortality gap by metropolitan status was steady throughout the period until 2010, when it widened among Hispanics.

Between 1990 and 2017, working-age mortality from ischemic heart disease and other circulatory diseases declined in every state (Figure 9-9). The District of Columbia experienced the largest absolute and relative declines. In general, the largest declines occurred in Northeastern states and the smallest in several Western and Southern states. As of 2015–2017, the South accounted for 8 of the 10 states with the highest rates of male

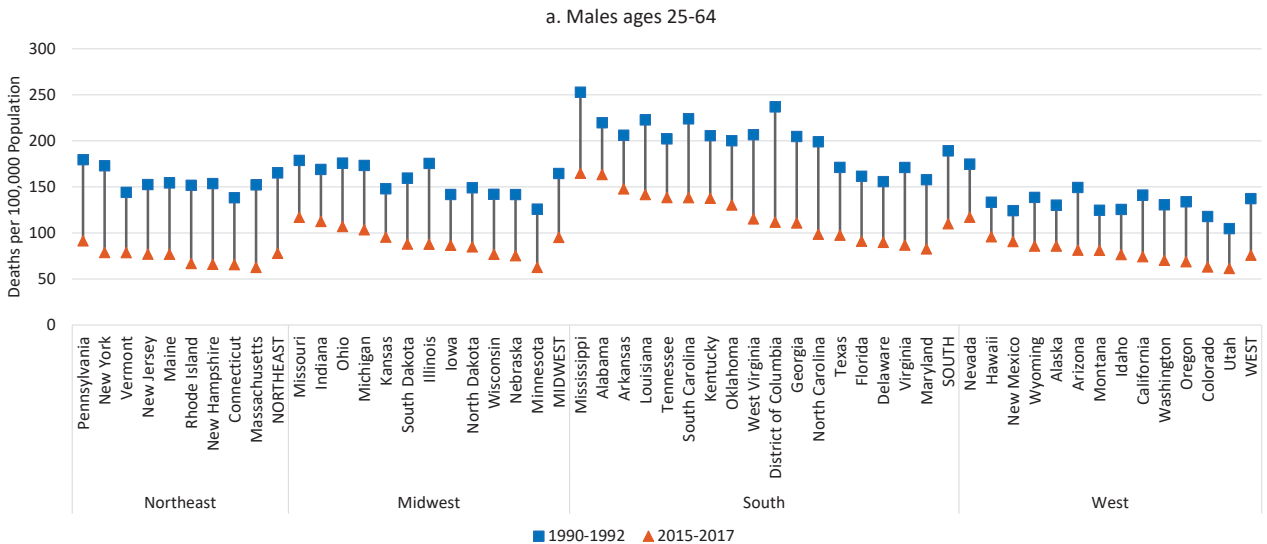

FIGURE 9-9 Mortality rates (deaths per 100,000 population) from ischemic heart disease and other diseases of the circulatory system among U.S. working-age males and females (ages 25–44), 1990–1992 and 2015–2017, by region and state.
NOTE: Mortality rates are shown for 1990–1992 (blue squares) and 2015–2017 (orange triangles), along with the changes over time (black connecting lines). Mortality rates for males are shown in panel a, while those for females are shown in panel b. Rates are age-adjusted by 10-year age band. States are ordered from highest to lowest mortality rate in 2015–2017 within region.
SOURCE: Data from CDC WONDER Online Database, https://wonder.cdc.gov.

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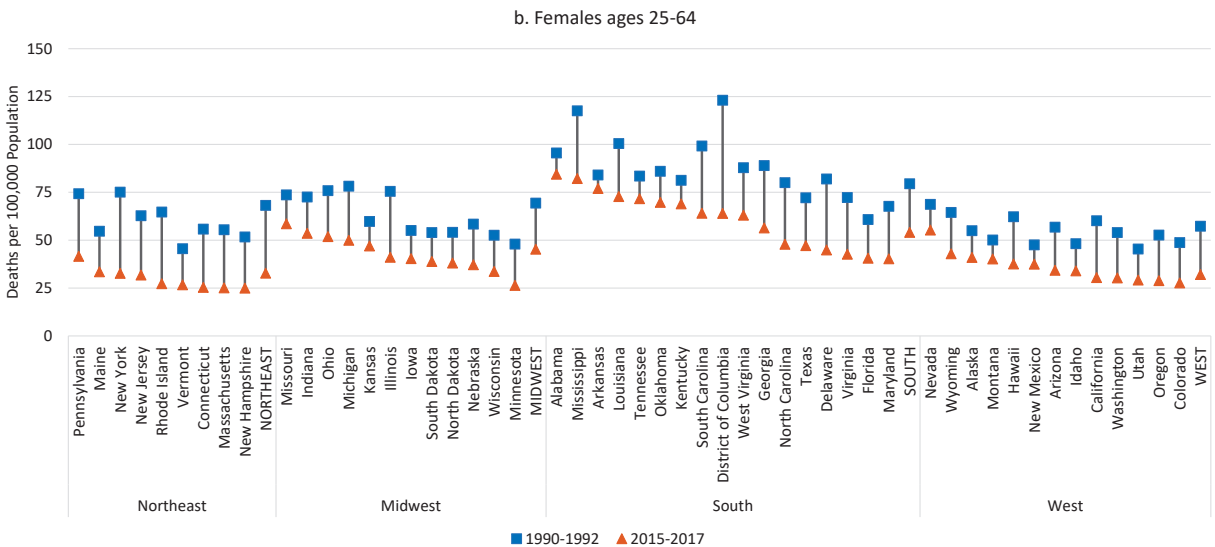

FIGURE 9-9 Continued

mortality from these diseases (the two other states were Nevada and Missouri); all 10 states with the highest female rates were in the South. This was the case despite the comparatively large declines in mortality that occurred in the South over the study period.

Summary of Trends

Cardiovascular disease (CVD) was the leading cause of death among working-age males and Black females throughout the study period, and the second leading cause of death (after cancer) among White and Hispanic females. Mortality from CVDs declined substantially throughout the 1990s and 2000s among working-age adults, particularly Black males and females. These improvements were due primarily to steady reductions in death rates from ischemic heart disease and other circulatory diseases during the 1990s and 2000s. However, these mortality reductions in ischemic heart disease and other circulatory diseases masked concerning trends in mortality due to hypertensive heart disease and ENM diseases that were rising among young adults, White adults, and Hispanic males during the 1990s and 2000s. These countervailing mortality forces ended when reductions in cardiovascular mortality among all population groups stalled in the 2010s. From 2009–2011 to 2017, mortality from ischemic heart disease and other circulatory system diseases remained largely unchanged among U.S. working-age adults (though see footnote 1 above). At the same time (after 2010), mortality from hypertensive heart disease increased among younger (ages 25–44) working-age adults (except Hispanic females), White working-age adults, and older (ages 55–64) working-age adults, and remained steady among other working-age adults, while mortality due to ENM diseases increased among all working-age adults, particularly Blacks.

These troubling changes in cardiometabolic mortality were particularly pronounced in the South and in areas outside of large central metros, which, along with the Northeast, generally experienced the most favorable trends in cardiometabolic mortality. As a result of these trends, the gap in mortality by metropolitan size grew over time, particularly among White working-age adults.

The contribution of cardiometabolic mortality to the recent rise in all-cause working-age mortality was therefore due to the net increases in mortality from cardiometabolic diseases after 2010, when mortality declines in ischemic heart disease and other circulatory system diseases stalled and no longer offset the rising rates of mortality from ENM diseases and hypertensive heart disease. These trends in cardiometabolic mortality are likely related; that is, the factors causing the rise in mortality from ENM diseases and hypertensive heart disease may also be related to the recent slowdown in reductions in mortality from ischemic heart disease and other

circulatory diseases. Moreover, the magnitude of the long-term decline in mortality from ischemic heart disease and other circulatory diseases has masked the significant implications of the recent stagnation in this trend since 2010. For example, recent research by Mehta, Abrams, and Myrskylä (2020) demonstrates that the slowing pace of decline in cardiovascular mortality was an important factor in the longer-term stagnation in U.S. life expectancy, playing a much larger role since 2010 relative to other causes of death, including drug-related causes. These authors show that drug-related mortality contributed to the shorter-term decline in life expectancy over 2014–2017, whereas stagnation in mortality due to CVD contributed to the longer-term flattening trend in life expectancy. In simulations of life expectancy at age 25, the authors found that the gains in life expectancy would have been greater by a factor of 4 for males and 8 for females if mortality from CVD had continued its rate of decline after 2010, compared with no increase in drug-related deaths since 2010. Their findings indicate that the slowdown in progress in CVD has been more consequential for U.S. life expectancy relative to increasing drug-related mortality. Similarly, Masters, Tilstra, and Simon (2018) found that without "cohort-based increases in metabolic disease mortality risk, the recent increases in middle-age U.S. White women's overall mortality rates likely would not have occurred" (p. 87). Indeed, the stalling reductions in mortality from CVD among U.S. working-age adults will likely continue to shape U.S. mortality trends in the future (more on this below).

Turning next to explanations for the related trends in mortality from ENM diseases, hypertensive heart disease, and ischemic heart disease and other circulatory diseases between 1990 and 2017, three important themes are relevant. First, the obesity epidemic, fueled by expanding obesogenic environments, has been a major cause of increasing disease, affecting multiple body systems and leading to deaths from hypertension, diabetes, stroke, and CHD (Mensah et al., 2017). Second, while declining smoking rates and medical advances over the past 50 years have greatly lowered mortality due to CVD, declines in smoking have slowed, and the contributions of further medical innovation have had less impact in recent years. Third, cardiometabolic diseases may be rising as a result of other social, economic, and cultural changes that have undermined economic security, intergenerational mobility, and social support networks, which in turn have led to increased chronic stress among the working-age population. The next section presents the committee's assessment of the evidence related to each of these explanations.

EXPLANATIONS FOR THE TRENDS IN CARDIOMETABOLIC MORTALITY

Explanations for the above trends in cardiometabolic mortality point to the three themes noted above: rising rates of obesity; diminishing returns of medical advances; and social, economic, and cultural change.

Rising Rates of Obesity

Prevalence and Subgroup Differences

The most important trend potentially explaining the rise in working-age mortality due to ENM diseases and hypertensive heart disease and the stagnation in improvements in mortality due to ischemic heart disease and other diseases of the circulatory system is the increased prevalence of obesity in the U.S. population (Lloyd-Jones et al., 2016; Mensah et al., 2017; Sidney et al., 2016). This report uses the Centers for Disease Control and Prevention's (CDC's) definition of obesity as weight that is higher than what is considered a healthy weight for a given height based on body mass index (BMI). BMI is calculated as weight in kilograms divided by height in meters squared, rounded to one decimal place. Obesity in adults is indicated when BMI is greater than or equal to 30 as a measure of abnormal or excessive fat accumulation that presents a risk to health (Hales et al., 2020). Rates of obesity began to rise in the early 1980s, a trend that has continued as a period-based phenomenon affecting children and adults of all ages (Reither, Hauser, and Yang, 2009). The prevalence of obesity among U.S. adults nearly tripled, increasing rapidly from the late 1970s through the early 2000s, with sustained but less rapid increases since then. The age-adjusted prevalence of adult obesity, defined as the percentage of adults ages 20–74 with a BMI of 30 kg/m^2 or above, was 42.4 percent in 2017–2018 compared with 15.0 percent in 1976–1980 (Fryar, Carroll, and Ogden, 2018; Hales et al., 2020).

Obesity rates vary by age, sex, race, ethnicity, and socioeconomic status. Older adults tend to have higher rates of obesity relative to younger adults (ages 20–39), although age differences in obesity have diminished as its prevalence has increased over time (Fryar, Carroll, and Ogden, 2018; Hales et al., 2020). Black women have higher obesity rates than Black men, but there are no significant differences in prevalence between men and women among White, Asian, or Hispanic adults (Hales et al., 2020). In 2017–2018, 56.9 percent of Black women were obese, compared with 41.1 percent of Black men. Overall, obesity rates are highest among Blacks (49.6%), followed by Hispanics (44.8%), Whites (42.2%), and Asians (17.4%).

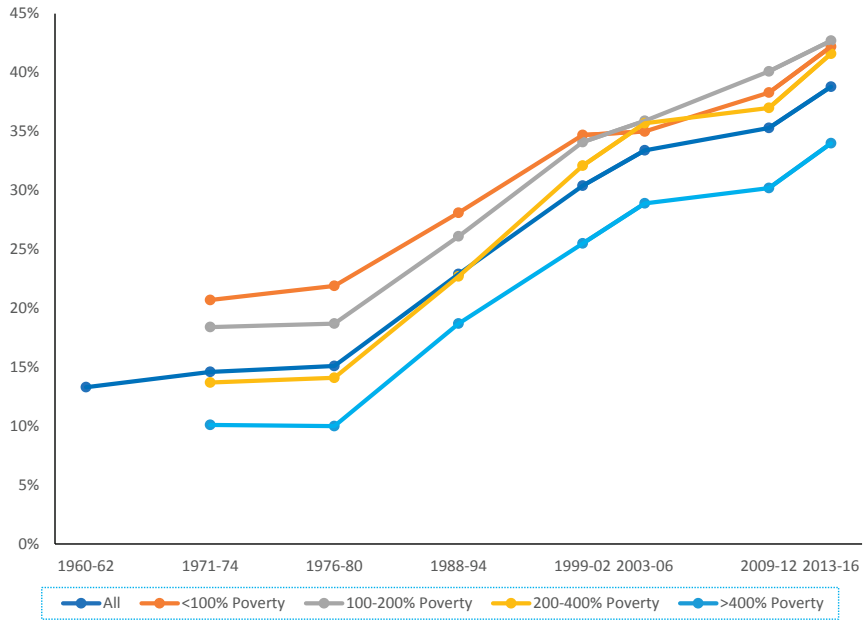

FIGURE 9-10 Obesity rates by income measured as percentage of poverty in each respective period, among U.S. adults, 1960–2016 (obesity defined as body mass index [BMI] >30).
NOTE: Data are from the National Health and Nutrition Examination Survey (NHANES). The sample is ages 20–74 for 1960–1962, 1971–1974, and 1976–1980 and ages 20+ thereafter.
SOURCE: NCHS (2019a).

Rates of obesity have increased in a similar fashion among adults in all income categories, as shown in Figure 9-10, and higher-income populations consistently have lower rates of obesity relative to those with lower income. Working-age adults with a college degree have a lower prevalence of obesity compared with their less-educated peers (Figure 9-11), although this pattern is not consistent by race and ethnicity and sex. For example, Black men show no difference in rates of obesity by educational attainment (Ogden et al., 2017).

Given differentials in obesity by socioeconomic status, some research has examined whether obesity explains differentials by socioeconomic status in the recent rise in working-age mortality. The evidence to date suggests that changes in obesity are not exerting differential impacts on working-age mortality by socioeconomic subgroups of the population. Cutler, Meara, and Richards-Shubik (2011) show that changing rates of obesity were not

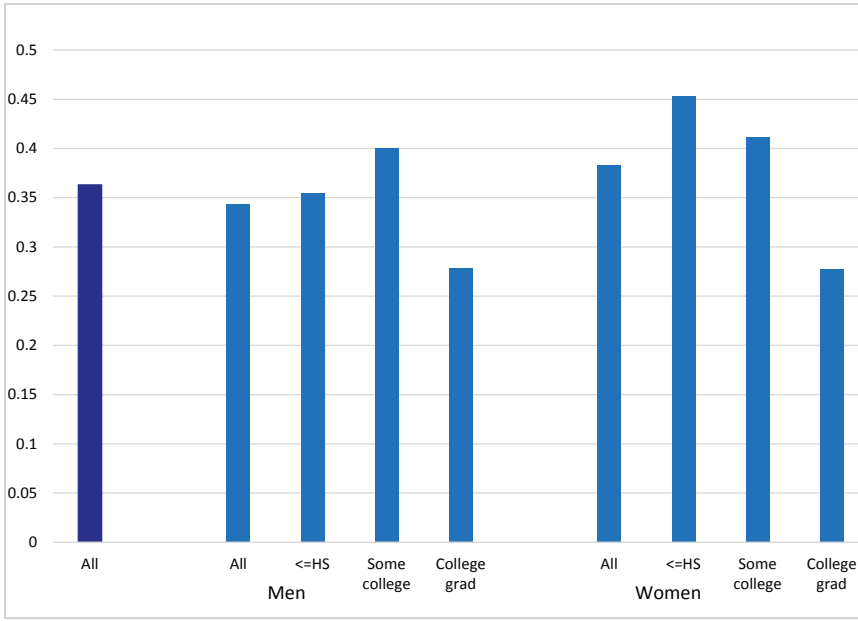

FIGURE 9-11 Obesity prevalence by educational attainment for men and women, 2011–2014 (obesity defined as body mass index [BMI] >30).
SOURCE: Prevalence of Obesity Among Adults, by Household Income and Education—United States, 2011–2014, *Morbidity and Mortality Weekly Report*, https://www.cdc.gov/mmwr/volumes/66/wr/mm6650a1.htm.

responsible for widening educational differences in mortality among U.S. adults (ages 25–74) between the 1970s and early 2000s. And Geronimus and colleagues (2019) demonstrate that changes in mortality at working ages (25–64) due to CVD and diabetes between 1990 and 2015 were highly similar for low- and high-educated Black and White men and women. Together, these studies suggest that changes in obesity, while clearly contributing to working-age mortality for key causes of death over the past 25 years, has had little impact on socioeconomic disparities in those trends.

Obesity-Related Mortality

In the face of such dramatic increases in obesity in recent decades, a body of work has examined obesity's role in shaping mortality patterns and trends. The evidence that obese adults are at higher risk of mortality from cardiometabolic diseases relative to nonobese adults is extensive. Obesity increases the risks of hypertension, stroke, CHD, type 2 diabetes,

and several site-specific cancers and related mortality (Adair et al., 2014; Basu et al., 2014; Di Angelantonio et al., 2016; Eisenmann, 2003; Manson et al., 1990; Prospective Studies Collaboration, 2009; Yu et al., 2017; Zhang et al., 2008). The Prospective Studies Collaboration (2009) carried out a meta-analysis of the association between BMI and mortality among 900,000 individuals in 57 prospective studies designed primarily to evaluate risk factors for CVD. That meta-analysis showed that both overweight (BMI = 25.0–29.9 kg/m^2) and obesity (BMI >30 kg/m^2) status were associated with increased all-cause mortality; on average, each additional 5 BMI units over normal (e.g., BMI = 20–25 kg/m^2) was associated with a 30 percent increase in all-cause mortality. The proportionate increase in mortality risk was greatest for overweight and obese younger adults ages 35–59 and decreased slightly in linear fashion in each subsequent 10-year age group. Increasing BMI greatly increased cardiovascular mortality, including deaths from ischemic heart disease, stroke, heart failure, and hypertensive heart disease, as well as from diabetes and kidney disease. The meta-analysis shows that the effects of BMI on heart disease and stroke are mediated in part by increases in blood pressure and diabetes (Prospective Studies Collaboration, 2009). However, the analysis includes smokers and those with preexisting cancer, who tend to have lower BMIs as a result of illness, likely leading to an underestimate of the association between BMI and all-cause mortality.

A follow-up study by Berrington de Gonzales and colleagues (2010) examined the relationship between BMI and all-cause mortality in a pooled analysis of 19 prospective studies that included 1.46 million White adults. The analysis was limited to healthy participants ages 19–84 who had never smoked, had had their BMI measured, and had been followed for a median of 10 years to ascertain mortality events. The authors found that hazard ratios of death from any cause were higher among White men and women with overweight and obesity relative to those with normal weight and that the risks were greater for those with overweight or obesity at younger ages. Their findings suggest that overweight or obesity in young adulthood versus later in life may have a greater effect on heightening the risks of disease and death (although older survey participants introduce a health bias; see below). Importantly, the hazard ratios associated with overweight and obesity were higher for cardiovascular mortality than for mortality from cancer and other causes.

Subsequent studies improved upon prior study designs to better understand how obesity is related to mortality. For example, studies with multiple measures of BMI across the life course or with retrospective reports of BMI at an earlier life stage have attempted to estimate when in the life course weight gain or high BMI is most related to subsequent morbidity and mortality risks, and whether the life-course patterns of obesity exposure explain

differential mortality risks (Abdullah et al., 2011; Cao, 2015; Engeland et al., 2004; Ferraro, Thorpe, and Wilkinson, 2003; Gray et al., 2011; Owen et al., 2009; Preston, Mehta, and Stokes, 2013; Stokes and Preston, 2016; Tirosh et al., 2011; Zheng et al., 2017). The evidence indicates that obesity experienced in early life, especially young adulthood, is associated with higher subsequent mortality because obesity tracks across the life course and operates through increased risks of cardiometabolic diseases (e.g., diabetes, hypertension, and CVD) (Engeland et al., 2004; Gray et al., 2011; Tirosh et al., 2011; Zheng et al., 2017). Moreover, any exposure to obesity, even among individuals who subsequently lose weight and achieve a normal BMI, is associated with higher subsequent mortality (Stokes and Preston, 2016). It is clear that longer durations of obesity across the life course are associated with greater cardiometabolic disease risks and mortality (Abdullah et al., 2011; Everhart et al., 1992; Harris et al., 2020; Reis et al., 2013).

Finally, several studies show the need to consider the cohort-based prevalence of obesity when estimating the percentage of mortality attributable to obesity (Masters, Powers, and Link, 2013; Masters et al., 2013; Olshansky et al., 2005; Reither, Olshansky, and Yang, 2011; Reither, Hauser, and Yang, 2009). Masters, Powers, and Link (2013) and Masters and colleagues (2013) demonstrate how cohort variation confounds age-specific hazard estimates of the obesity–mortality relationship. When cohort membership is not considered, the age-specific mortality hazards of BMI/obesity appear to weaken with age (Berrington de Gonzales et al., 2010; Kuk and Ardern, 2009; Prospective Studies Collaboration, 2009; Stevens et al., 1998). Masters and colleagues (2013) found that age-related sampling biases affect the obesity–mortality relationship because of attrition: older obese individuals are disproportionately missing in survey samples because of illness and higher death rates. After accounting for survey selection bias and cohort differences in the prevalence of obesity, these authors found that the obesity–mortality relationship remained strong at all ages. Because obesity prevalence has increased with successive cohorts (Lee et al., 2010, 2011; Robinson et al., 2013), mortality risks are expected to increase as younger cohorts age. Indeed, Masters and colleagues (2013) show that obesity has accounted for an increasing share of U.S. deaths in recent decades—about 18 percent of all deaths between the ages of 40 and 85 during the period 1986–2006. These figures are consistent with estimates by Stokes and Preston (2016) that attribute about 16 percent of U.S. deaths among adults ages 50–84 to obesity.

While the obesity epidemic swept through the entire age distribution of the U.S. population in a specific time period, its health consequences have followed a cohort-specific pattern (Masters et al., 2013). Thus as noted earlier, those who were younger when the epidemic occurred (i.e., those born in the 1980s) and are now in their 30s have been exposed to obesogenic

environments for a longer period of their lives relative to previous cohorts of young adults. Indeed, younger adults in more recent birth cohorts have been exposed to obesogenic environments for most of their lives and are therefore most at risk of its cardiometabolic health consequences compared with when older birth cohorts were the same age (Robinson et al., 2013). Data from the National Longitudinal Study of Adolescent to Adult Health (Add Health) study on a cohort born in the late 1970s and early 1980s who have been followed to midlife confirm this sobering fact. When members of the Add Health cohort were in their late 20s in 2008, 37 percent were obese; 10 years later, in 2016–2018, when they were in their late 30s, 47 percent were obese. The cardiometabolic consequences were already emerging for this cohort in 2008 when 26 percent had hypertension, 6 percent had diabetes, and 27 percent had prediabetes (Harris et al., 2019). The cardiometabolic profile of the Add Health cohort represents the new generation of working-age adults in the United States, and is reflected in the trend data showing that among Black and Hispanic younger adults (ages 25–44), increases in mortality from ENM diseases and hypertensive heart disease in 1990–2017 make up a larger share of the rise in all-cause mortality at these ages relative to older Black and Hispanic adults (see Tables 4-2 and 4-3, respectively, in Chapter 4).

Explaining the Increase in Obesity

Identifying the determinants of the rise in obesity can point to modifiable factors for potential interventions to reduce cardiometabolic mortality. This section reviews the scientific evidence on the causes of the U.S. obesity epidemic following the conceptual framework in Chapter 6, which differentiates downstream (i.e., proximate) from upstream (meso-level and macrostructural) drivers. Note that the focus is on how upstream and downstream factors *changed* to bring about the increase in obesity and by extension, a change in cardiometabolic mortality. For obesity and mortality risks to change, the determinants of obesity and mortality must have changed as well.

Proximate determinants The most proximate causes of obesity involve genetics and behavior. Cardiometabolic conditions and diseases are at least partly heritable, so genes always play a role (Ehret et al., 2011; Nikpay et al., 2015; Ravussin and Ryan, 2018). Given that inherited genes are fixed throughout one's lifespan, however, genetic factors can matter in the rise in obesity and related changes in cardiometabolic mortality only if they combine with changes in behavior. Health behaviors such as diet, smoking, alcohol use, and physical exercise affect cardiometabolic health risks. Sedentary lifestyles and workplace environments, including long commutes

and car-based work and prolonged screen time, reduce physical activity and increase the risks of obesity (Church and Martin, 2018; Ekelund et al., 2011). Diets high in fat, sugar, and carbohydrates and low in fiber, associated with regular consumption of processed foods, increase the risks of overweight and obesity (Hall, 2018; Malik et al., 2010; Walker et al., 2020). How have these health behaviors changed since 1990 to affect the risks of obesity and cardiometabolic mortality?

Obesity results from an imbalance between calories consumed and calories expended. The evidence on the relative contribution of each of these factors is not extensive, but the literature offers some insights. Since the 1980s, caloric intake has increased substantially. Cutler, Glaeser, and Shapiro (2003) report that calories consumed per adult increased by about 200 per day between the late-1970s and mid-1990s. In steady state, 200 calories per day amounts to about 20 pounds of weight annually. At the same time, caloric expenditure appears to have changed little. Between 1975 and 1995, time spent in housework fell, but time spent at work rose (Cutler, Glaeser, and Shapiro, 2003, Table 3). TV hours increased, but so did active recreation time. By this analysis, the major issue to explain is why caloric intake has increased so greatly.

However, that is not the only interpretation of the evidence. Over the first half of the 20th century, caloric expenditure fell as people moved off farms and into cities. At the same time that the need for calories fell, caloric intake fell apace. Thus, weights did not change because people made adjustments in what they consumed to match their falling needs. One might ask why the same cannot be said about the more recent period. Indeed, by 2010, the most recent year for which caloric consumption data are available, average daily calories continued to climb by another 100 additional calories over the mid-1990s level (Desilver, 2016). As caloric intake has increased, why have people not increased their caloric expenditure to match their greater intake? Trends in physical activity provide part of the answer. Research suggests that regular recreational exercise has not changed over the past few decades, but occupational activity has (Church and Martin, 2018). By 2006, only 20 percent of American jobs required high levels of physical activity, compared with 1960 when more than 50 percent of jobs required levels of physical activity that met the current daily physical activity goals. The increased low levels of occupational activity mean that many individuals have such low energy expenditure that caloric intake has become uncoupled from expenditure (Church and Martin, 2018).

Environmental determinants Much attention has been devoted to the role of the obesogenic environment, defined as an environment that promotes weight gain and is not conducive to weight loss within the home or workplace (Swinburn, Egger, and Raza, 1999; Townshend and Lake, 2017).

Such features of the built environment as automobile-dependent design, decreased walkability, and limited access to green spaces and recreational opportunities combine with food advertising and availability to help shape patterns of physical activity and diet that influence the risks of obesity and cardiometabolic disease (Hill et al., 2003). To the extent that these environmental factors have changed over time, they may be influencing mortality trends, although there would be long lag times in their effects given the lengthy causal chains linking these more distal factors to cardiometabolic mortality. Thus, changes to the built environment in the mid-20th century, from the construction of interstate highways in the 1950s and 1960s to the emergence of fast food restaurants, could explain increases in obesity in subsequent decades and an increase in current rates of obesity-related mortality. Moreover, as the obesity epidemic spread, exposure to these period-based changes in the obesogenic environment has been greater for more recent cohorts, increasing their susceptibility to becoming obese.

A large body of research has examined how aspects of the obesogenic environment have contributed to obesity levels by influencing physical activity and dietary behaviors at the individual and community levels (Cobb et al., 2015; Townshend and Lake, 2017). Yet the wide range of study designs, methods, metrics, and environmental variables employed in various studies makes cross-comparison and strong evidence elusive (Church and Martin, 2018; Davis, Plaisance, and Allison, 2018; Hall, 2018; Mackenbach et al., 2014; Sturm and An, 2014). While the basic drivers of obesity are straightforward (more energy consumed than expended), its etiology is multifactorial and complex. The combination of multiple factors (including built environment and social changes) may have created the "perfect storm" for obesity and its consequences for cardiometabolic mortality risk (Ravussin and Ryan, 2018). Rigorous studies have identified the singular role of these factors: (1) inactivity in daily life resulting from automobile dependence (Frank et al., 2007; Lopez-Zetina, Lee, and Friis, 2006), (2) longer commute times and reduced time to prepare foods (Christian, 2012; Zhang et al., 2014), (3) greater reliance on ultraprocessed and high-calorie foods (Juul et al., 2018; Steele et al., 2016), (4) extensive availability and marketing of relatively low-cost processed and high-calorie products (Drewnowski, 2004; Hruby and Hu, 2015), and (5) more sedentary lifestyles linked to work and leisure activities that rely increasingly on passive behaviors (changes in the nature of work and use of electronic devices for entertainment) (Lakdawalla and Philipson, 2009; Sturm and An, 2014). The sum of this research provides substantial evidence for how physical environments have changed to become increasingly obesogenic and how obesogenic environments are associated with obesity rates. Remaining to be understood, however, are the causal pathways by which the changing environment fueled the level and spread of obesity, and, by extension, the

rise in mortality from ENM diseases and hypertensive heart disease or the slowing declines in mortality from ischemic heart disease and other circulatory diseases.

Research on the impact of the food environment on diet focused initially on proximity to healthy and unhealthy food sources (often using types of food stores as a crude proxy for the availability of healthy food). A number of earlier studies suggested that proximity to sources of healthy food (e.g., stores selling fruits and vegetables) was related to healthier diets (Larson, Story, and Nelson, 2009; Morland, Wing, and Roux, 2002) and, conversely, that proximity to fast food outlets was related to greater consumption of calorie-rich diets (Davis and Carpenter, 2009; Lopez, 2007). However, longitudinal evidence documenting the relationship between changes in food environments and healthy versus unhealthy diets is less common and not conclusive (Caspi et al., 2012; Sturm and An, 2014).

It may be that the food environment operates at a much larger spatial scale than has been studied in existing empirical work. For example, food environments may operate primarily through the ubiquitous availability (and promotion) of low-cost, high-calorie foods (and drinks) in multiple contexts encountered by individuals in the course of their daily lives. Consumption of processed and ultraprocessed foods is also ubiquitous in home, work, school, and leisure environments (Poti et al., 2015; Steele et al., 2016), representing 25–60 percent of total daily energy intake (Steele et al., 2016). Ultraprocessed foods undergo multiple physical, biological, and/or chemical processes and generally contain food additives, almost none of which are nutritious or healthy (Monteiro et al., 2019). Not surprisingly, the evidence indicates that consumption of these foods contributes to the risks of obesity (Mendonça et al., 2016), hypertension (Mendonça et al., 2017), diabetes (Srour et al., 2020), and CVDs (Srour et al., 2019).

Macrostructural determinants Macro-level technological changes have greatly altered the way people produce, obtain, and consume food, especially highly processed, palatable, and cheap food with high amounts of sugar, fat, salt, and flavor additives (Cutler, Glaeser, and Shapiro, 2003; Hall, 2018). Such innovations as vacuum packing, improved preservatives, deep freezing, artificial flavors, and microwaves have enabled food manufacturers to cook food centrally and ship it to consumers for rapid consumption. Agricultural innovations and the mechanization of work once performed by humans have increased the efficiency of food production as well as the quantity of food produced, and thereby provided Americans with the cheapest food in the world (Lakdawalla and Philipson, 2009). Access to processed food has greatly increased through vending machines, takeaways, cafes, convenience stores, and fast food restaurants (Morland and Evenson, 2009). As a result, traditional normative eating behaviors have shifted over time from

time-consuming home cooking to more ubiquitous snacking and consumption of large-portion-size meals, often at restaurants.

Cutler, Glaeser, and Shapiro (2003) examined how technological change affected rates of obesity in the United States. They argue that obesity resulted from technological changes that reduced the price—in terms of both money and time—of obtaining food, especially less-healthy food. Whereas the technology for producing and distributing fresh food (e.g., fruits and vegetables) has not changed much over time, the technology for producing, preserving, and distributing less-healthy food has increased markedly. For example, soda can be mass produced and distributed more readily than in the past, advances in preservatives have made such foods as potato chips and cookies available on virtually every street corner, and fresh pizza can now be made easily and sold in more locations. Not surprisingly, people consume more food as its monetary and time costs decline. Internationally, countries, especially English-speaking countries, that allow more mass production of food have experienced greater increases in obesity (Bleich et al., 2008).

Given the evidence of direct links between technological changes in food production and distribution and the rise in obesity, one role for government is to provide incentives for the production and distribution of healthy foods and disincentives for the mass production and promotion of unhealthy foods. Recent innovations have led to the use of many traditional technologies, such as fermentation, extraction, encapsulation, fat replacement, and enzyme technology, to produce new healthy food ingredients, reduce or remove undesirable food components, add specific nutrient or functional ingredients, modify the composition of foods, mask undesirable flavors, and stabilize ingredients (Hsieh and Ofori, 2007). New techniques currently in development include the use of high-pressure processing to substantially extend the shelf life of healthy food products. This technique preserves the sensorial and nutritional properties of food by not involving heat treatment and maintains the food's original freshness throughout its shelf life without the addition of chemical preservatives (McFadden, 2018).

Unfortunately, some innovations in food science, such as the introduction of synthetic sweeteners, have also contributed to the obesity epidemic. Beginning in the 1980s, farm subsidies authorized by Congress encouraged an oversupply of corn, thereby lowering its cost and encouraging the production of high-fructose corn syrup, a sweetener that has made a major contribution to obesity. U.S. per capita consumption of high-fructose corn syrup increased from 0.8 g per day in 1970 to 91.6 g in 2000 (Bray, Nielsen, and Popkin, 2004), and added sugar intake from corn sweetener rose by 359 percent from 1970 to 2007 (Sturm and An, 2014).

Finally, the food industry—aided by legislation and budget decisions promoted by lobbyists and politicians from agricultural states—has

successfully leveraged advertising and marketing techniques to boost consumption of calorie-dense foods (Sadeghirad et al., 2016). Likewise, restaurants promoting inexpensive, unhealthy, and "all you can eat" menus have proliferated, especially in the socioeconomically disadvantaged neighborhoods that are at greatest risk for obesity.

The need for solutions is widely recognized in the public and private sectors, driven not only by public health concerns but also by the threat obesity poses for employers, the business community, and the armed services. Further work is needed to build on recent efforts—some led by the food industry itself and others by public health authorities—to discourage the production and purchase of unhealthy foods or at least give consumers better information with which to make healthier food choices. Examples include self-regulation of or restrictions on misleading advertising (Graff, Kunkel, and Mermin, 2012), pricing and tax strategies (Blecher, 2015; Novak and Brownell, 2011; Powell et al., 2013), "Nutrition Facts" product labels mandated by the Food and Drug Administration (FDA) (Food and Drug Administration [FDA], 2020), menu labeling by restaurants (VanEpps et al., 2016), and zoning restrictions limiting the proliferation of fast food restaurants. The fact that U.S. caloric intake per capita outpaces that of other high-income countries (Institute of Medicine and National Research Council [IOM and NRC], 2013) suggests the need to identify structural causes, from lax restrictions on the food industry and advertisers to cultural differences in lifestyle, such as levels of physical activity.

Governments, for example, could seek to address the issues that prevent people from offsetting the consequences of ever-cheaper food. The increase in food availability has occurred in parallel with people's having a good deal of additional time; this is the case particularly for women, who no longer spend as many hours cooking and cleaning as they once did (Sturm and An, 2014). At the societal level, people could use this additional time to work off the added calories from prepared food and still have time left over (Cutler, Glaeser, and Shapiro, 2003). Many people talk about wanting to make this trade, but society at large has not chosen to do so, perhaps because it is easy to eat today and delay exercise until tomorrow. Helping people engage in the behavior that is necessary to reduce obesity is a key public priority.

The promotion of healthy foods and diets among children and youth is especially critical given that healthy habits and lifestyles are formed early in life, when health trajectories tend to become set into young adulthood (Harris et al., 2006; Kane et al., 2018). School breakfast and lunch offerings, access to sugary drinks, and the contents and locations of vending machines are increasingly being monitored to promote access to healthy snacks and daytime meals for children. Some evidence indicates that increasing the costs of nutritionally less-desirable foods is most likely to

have an effect on body weight among youth, people of low socioeconomic status, and those at risk of obesity (Powell et al., 2013). Taxes on sugary drinks and educational programs on the health consequences of poor food choices might help steer the public toward more healthy diets, and more research is needed to evaluate the effects of such policies and programs on the consumption of healthier food.

Prevention and Treatment of Obesity

Extensive reviews of treatment programs to reduce obesity and prevention programs to avoid obesity and maintain healthy weights have provided limited evidence of the long-term success of these programs (Jeffery et al., 2000; Johnston et al., 2014; Stice, Shaw, and Marti, 2006), although many local interventions and programs have shown short-term success with weight loss and increased physical activity (Community Guide, 2020a, 2020b; Johnston et al., 2014; Wang et al., 2013). Research clearly points to the need to alter the food environment to reduce obesity (Mattes and Foster, 2014); the challenge appears to be how to maintain healthy lifestyle changes for more permanent reductions in weight and body mass (Sobol-Goldberg, Rabinowitz, and Gross, 2013). In terms of prevention, the evidence points overwhelmingly to avoiding obesity at all costs. As noted earlier, a growing body of research shows that any experience of obesity over one's life course, longer life-course durations of obesity, and earlier life-course exposure to obesity result in heightened morbidity and mortality risks (Abdullah et al., 2011; Harris, Duncan, and Boisjoly, 2002; Owen et al., 2009; Reis et al., 2013; Stokes, Ni, and Preston, 2017; Zheng et al., 2017). Thus, prevention programs need to target children and adolescents most at risk of obesity (e.g., racial/ethnic minorities, females, people living in poverty and with low socioeconomic status) and those who are overweight or experiencing weight gain to intervene before obesity trajectories become set throughout adulthood. Overall, the lack of long-term success for weight loss and exercise programs in maintaining healthy weights speaks to the need for program development; experimental designs; and quantitative and qualitative research to uncover the social, biological, and behavioral impediments to permanent weight loss.

Diminishing Returns of Medical Advances

Starting in the late 1960s, there was a remarkable turnaround in the century-long trend of increasing cardiovascular mortality in the United States. By the 1970s, death rates due to CVD, and in particular CHD and stroke, were in sharp decline, registering a 70 percent decline by 2010 (Mensah et al., 2017). Medical innovations in drug development

and prevention, treatment, and control of chronic diseases, together with increasing knowledge about the health effects of cigarette smoking, fueled this decline. Studies suggest that medical advances and surgical treatments explain about 47 percent of the decline, while reductions in major risk factors (e.g., smoking, poor diet, lack of physical activity) explain approximately 44 percent (Ford et al., 2007; Mensah et al., 2017).

The 1964 Surgeon General's Report on Smoking and Health publicized the dangers of cigarette smoking, triggering a series of tobacco control efforts that successfully reduced tobacco use (see Chapter 11). The benefits of treating moderate hypertension were demonstrated in the early 1970s, and the benefits of lowering cholesterol emerged in the early 1980s, leading to the establishment of the first standards and targets for blood pressure and cholesterol management in 1987 (Mensah et al., 2017). By the late 1980s, powerful new drugs for managing blood pressure (e.g., angiotensin-converting enzyme inhibitors and beta-adrenergic blocking agents) and cholesterol (e.g., statins) also contributed to lowering CVD mortality. Furthermore, the late 1980s introduced thrombolysis[2] and angioplasty as standard treatments for acute myocardial infarction, stroke, and pulmonary embolisms. Coronary stents were approved by the FDA in the 1990s and widely used thereafter.

As discussed previously, the 21st century brought a slowdown in the impact of medical advances in treating CVD (Mensah et al., 2017). The new century saw some additional clinical trials of statin drugs, a focus on treating isolated systolic hypertension[3] in the elderly, and a lowering of blood pressure targets to increase blood pressure control, but innovations after 2000 had a smaller impact relative to those in prior decades. Stagnation in improvements in CVD mortality became evident after 2010, garnering public attention (e.g., Bever, 2019; McKay and Winslow, 2016; Painter, 2019) and stimulating research to find an explanation (Ma et al., 2015; Mensah et al., 2017; Shah et al., 2019; Sidney et al., 2016). Several explanations for the diminishing returns of medical advances in lowering CVD mortality after 2010, detailed in the next section, have been proposed: more incremental development of medical innovations, rising obesity that blunted the impact of medical advances, and differential delivery of or access to the medical advances that enabled the reductions in CVD mortality.

International data do not support the explanation that a lack of medical innovations after 2010 is responsible for the stalling improvements in CVD. The flattening of the CVD mortality trend in the United States

[2]Thrombolytic agents dissolve dangerous blood clots in blood vessels to improve blood flow and prevent organ damage.

[3]Isolated systolic hypertension is the most common form of high blood pressure in adults over age 65. It occurs when diastolic blood pressure is less than 80 mm Hg and systolic blood pressure is 130 mm Hg or higher.

after 2010 has occurred in other industrialized countries as well (Case and Deaton, 2017; Lopez and Adair, 2019; Ma et al., 2015; Preston, Vierboom, and Stokes, 2018; Shah et al., 2019; Sidney et al., 2016). International comparisons are shown in Figure 9-12 (adapted from Mehta, Abrams, and Myrskylä, 2020). Although all of the 10 countries with high life expectancy in this figure experienced flattening trends in CVD mortality after 2010, the United States has not kept pace with the reductions in CVD mortality seen in these other countries, suggesting that other factors specific to the United States have been stalling continued improvements. Prime among those factors is obesity and its cardiometabolic consequences, which, as noted above, typically lag obesity exposure (Mensah et al., 2017; Olshansky et

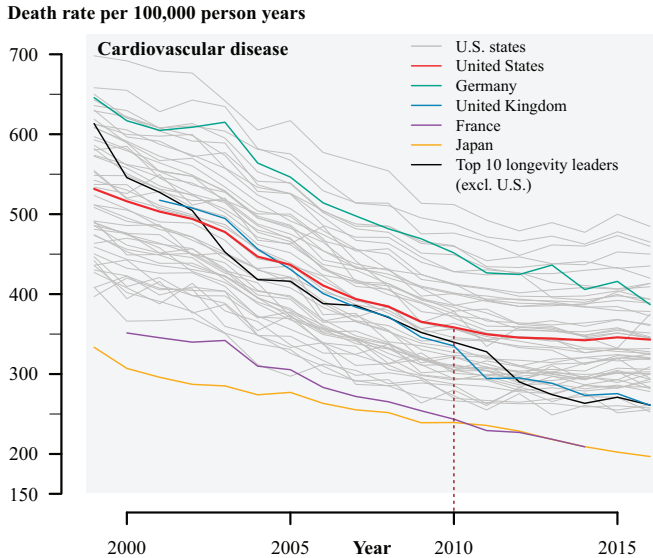

FIGURE 9-12 Death rates for cardiovascular disease (CVD) in the United States and selected peer countries, 1999–2017.
NOTES: Age-standardized death rates (per 100,000 person-years) for CVD from 1999 to 2017. Gray shaded lines are trends for individual U.S. states and DC. "Longevity leaders" denotes the average of countries that had the highest life expectancy at birth in 2010 (Japan, Switzerland, Singapore, Australia, Spain, Iceland, Italy, Israel, Sweden, and France). Sexes are combined. CVD deaths include deaths from heart attacks and strokes (International Classification of Diseases [ICD]-10 codes I00 to I78).
SOURCE: Adapted from Mehta, Abrams, and Myrskylä (2020). This figure is an excerpt of the original, licensed under Creative Commons Attribution-NonCommercial-NoDerivatives License 4.0 (CC BY-NC-ND).

al., 2005). Preston, Vierboom, and Stokes (2018) estimate that rising levels of BMI during 1988–2011 reduced life expectancy at age 40 by .9 years in 2011 and have reduced the impact of many beneficial factors working to improve mortality, such as declining smoking rates and medical advances that include pharmacological treatments for several obesity-related chronic conditions, such as hypertension, hyperglycemia, and dyslipidemia. Indeed, the cardiometabolic consequences of the obesity epidemic were beginning to emerge in data in 2009 showing an increase in the prevalence of high blood pressure and cholesterol, a decrease in physical activity, and an increase in BMI and high blood glucose (Mannsverk et al., 2016; McEwen et al., 2011; Pilkerton et al., 2015). Thus, the slowdown in improvements in mortality, particularly CVD mortality, in the past decade may be due to the lagged cardiometabolic consequences of the obesity epidemic, now under way for more than 30 years.

Another factor unique to the U.S. context that may explain the diminishing returns of medical advances in reducing CVD mortality is the persistent racial, ethnic, and socioeconomic disparities in chronic health conditions and control of chronic conditions related to cardiometabolic mortality that slow continued gains once those most at risk or those with greater health care access are treated. When new pharmaceuticals and other therapeutic treatments to control hypertension and heart disease were developed, delivery of these treatments was naturally targeted to those with the highest levels of disease (e.g., the highest levels of cholesterol or blood pressure), sharply reducing CVD mortality in the 1970s and 1980s (Weisfeldt and Zieman, 2007). Treating those with more moderate or mild cardiometabolic conditions, by contrast, results in marginal gains in improvement of CVD mortality, so that progress slowed after 2000.

In addition, many individuals may not be receiving or correctly using medications that are effective in controlling hypertension, diabetes, and heart disease, and many may be struggling to discontinue tobacco use. Research has shown that poor adherence to treatment results from a variety of barriers, including those experienced by patients (Ho, Bryson, and Rumsfeld, 2009; Rashidi et al., 2020) and those introduced by the health care system (Banerjee et al., 2016). Baroletti and Dell'Orfano (2010) divide the barriers faced by patients into three categories: socioeconomic (e.g., lack of adequate health care coverage, unemployment, poverty, concerns about cost), communication related (e.g., inadequate instructions, language barriers, mental illness), and motivational (e,g., not appreciating the gravity of these conditions or benefits of treatment, fears and concerns, and cultural beliefs) (Baroletti and Dell'Orfano, 2010). Understanding the barriers faced by patients and designing solutions to overcome those barriers is thus a priority for the control of cardiometabolic mortality and chronic diseases more generally.

When cardiovascular treatments were first introduced and when the dangers of smoking were first recognized decades ago, individuals with greater access to care, higher education, and other socioeconomic advantages were more likely to receive care and to quit smoking (Hiscock et al., 2012; Kanjilal et al., 2006; Ma et al., 2005; Sterling et al., 2019; U.S. Department of Health and Human Services [HHS], 2011). Much of the progress in lowering CVD mortality in prior decades may reflect the widespread uptake of these interventions among these more advantaged population groups, but adults who faced greater barriers to care may have been left behind. People of color and those of lower socioeconomic status face greater barriers to adherence to treatment targets and have higher rates of tobacco use (Cokkinides et al., 2008; Kiefe et al., 2001). The field of implementation science (also known as translational and health services research) studies interventions to close this gap between recommended care and the care actually received by individuals, especially members of vulnerable groups (Bauer et al., 2015; University of Washington, n.d.). The magnitude of this gap first became apparent in 2003, when a classic study documented that U.S. adults received only 54.9 percent of recommended care (McGlynn et al., 2003).

That gap has persisted, especially among marginalized populations that have limited access to care and live in settings that make adherence difficult. For example, fewer than half of U.S. adults (44%) with hypertension have achieved optimal blood pressure control (Dorans et al., 2018), and slightly more than half (55%) who could benefit from lipid-lowering agents are currently taking that medicine (Mercado et al., 2015). Disparities in the prevalence of chronic diseases mirror disparities in control of those diseases. In 2015–2016, for example, Blacks had higher rates of hypertension (57.3%) but lower rates of blood pressure control (37.2%) relative to Whites (43.8% and 45.7%, respectively) (Dorans et al., 2018). Moreover, individuals with hypertension, hyperlipidemia (high cholesterol levels), and insulin resistance may be asymptomatic and escape detection. This is the case especially among younger adults, who get fewer regular medical checkups and have less access to screenings for disease at work or in other community settings (Zhang and Moran, 2017). National estimates show that three-quarters of young adults ages 24–32 with measured hypertension were unaware of their condition (e.g., had not received a medical diagnosis), and two-thirds with measured diabetes were similarly unaware (Nguyen et al., 2011, 2014).

It is well documented that both research to identify strategies for reducing these disparities in care and the evidence-based programs and policies that emerge from translational research have been less heavily funded relative to investments in new drugs and technologies (Dzau and Balatbat, 2019; Woolf, 2008). This imbalance is counterproductive with respect to

reducing mortality. For example, improving adherence to older-generation treatments can save more lives than developing newer agents with incrementally greater efficacy (Woolf and Johnson, 2005). Thus, after years of declining CVD mortality due to the broad uptake of pharmacotherapy and smoking cessation among advantaged populations, the recent decline in the pace of progress may reflect the failure to invest adequately in closing the prevention and treatment gap among high-risk populations. If so, a resurgence in progress in lowering CVD mortality in the United States could be stimulated by more robust efforts to help these populations overcome socioeconomic and environmental barriers to receiving effective cardiovascular treatments and successfully discontinue tobacco use.

A closer look at tobacco use is illuminating. The reduction in U.S. smoking first occurred among men in the early 1950s and later among women beginning in the late 1970s, producing a substantial decrease in mortality for U.S. adults (Cutler, 2008; Wang and Preston, 2009). Following the U.S. Surgeon General's Report on Smoking and Health (1964), cigarette smoking rates declined 67 percent, from 42.6 percent in 1965 to 13.7 percent in 2018 (American Lung Association, n.d.; Creamer et al., 2019). However, an array of new tobacco products, including e-cigarettes, has entered the market in the past decade, with 19.7 percent of U.S. adults reporting use of any tobacco product in 2018 (Creamer et al., 2019). That prevalence is even higher among males, adults less than 65 years old, non-Hispanic American Indians/Alaska Natives, those with a high school education or less, those with an annual household income of less than $35,000, and those with a disability or serious psychological distress (Creamer et al., 2019).

The educational disparities in the reduction of smoking are especially noteworthy. While smoking declined for all groups, greater progress occurred among adults with more education (Figure 9-13). Studies by Cutler, Meara, and Richards-Shubik (2011) and Montez and Zajacova (2013) show that reductions in smoking behavior have been greater among the highly educated than among the less educated. Over the period 1974–2016, the smoking rate declined by 76 percent among people with a college degree but by just 35 percent among those with only a high school degree. Yet despite this widening educational disparity in smoking, only a modest portion of the widening educational difference in U.S. adult mortality in recent decades is attributable to smoking because widening educational disparities in other trends (e.g., use of drugs) were more important contributors. Further research is needed to address the barriers to smoking cessation faced by populations that continue to smoke at higher rates, but whether interventions to overcome these barriers can refuel the decline in smoking and reductions in CVD mortality remains to be seen.

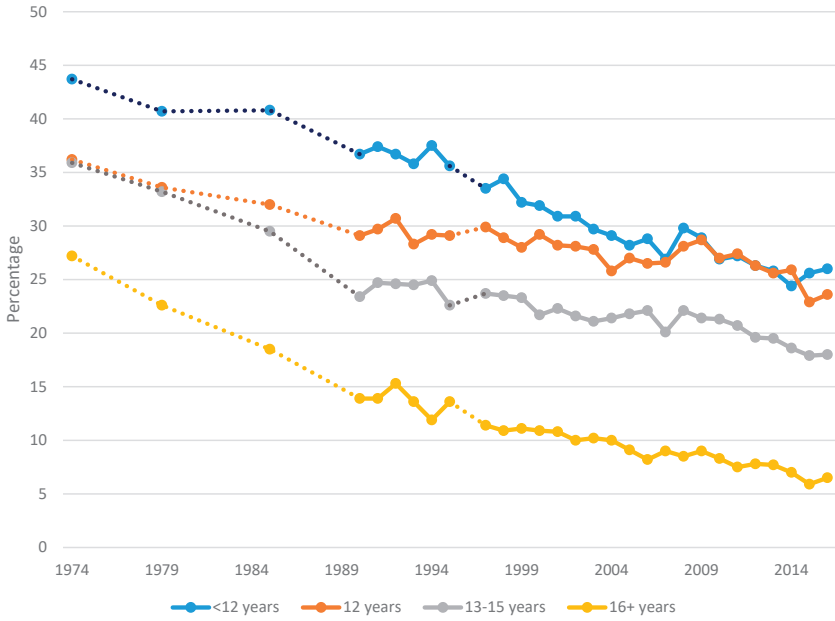

FIGURE 9-13 Trends in smoking by education among adults ages 20–74, 1974–2016.
NOTES: The years shown in this figure are those for which data have been published. Data for 1992 and later are not strictly comparable with data for earlier years because of a change in the National Health Interview Survey (NHIS). Because of the redesign of the NHIS in 1997, comparisons with data from prior years must be conducted with caution.
SOURCES: Data for 1974–2007 are from Centers for Disease Control and Prevention, National Center for Health Statistics, *Health, United States, 2009*, Table 61. Data for subsequent years are as follows: 2008–2009: *Health, United States, 2010*, Table 59; 2010–2012: *Health, United States, 2013*, Table 57; 2013–2014: *Health, United States, 2015*, Table 48; 2015–2016: *Health, United States, 2017*, Table 48.

Social, Economic, and Cultural Change

Social, economic, and cultural shifts over the past 50 years have profoundly changed the family, work, and community environments of daily life in America, and the possibility that these shifts may have contributed to cardiometabolic morbidity and mortality cannot be dismissed. Changes in family structure and rising family instability over the past 50 years have been well documented (Cavanagh and Fomby, 2019; Cherlin, 2009). Since the 1970s, increases in divorce, cohabitation, and nonmarital childbearing have greatly altered the family contexts in which children are raised and the financial and social capital to which they have access (Bianchi, 2011; Casper and Bianchi, 2001; Cherlin, 2009; McLanahan and Sandefur, 1994;

Sayer, Bianchi, and Robinson, 2004). During this period, families were affected by macroeconomic changes that included the loss of manufacturing jobs, which provided employment opportunities for many low-educated and unskilled males, and the growth of the service sector, which employed largely females and more highly educated individuals. Single mothers were now able to support a family on their own—albeit at much-reduced wages and earnings relative to a two-worker family—and the economic status of men, especially those with less education and fewer job skills, suffered (Oppenheimer, 2000, 2003). The rise in family instability, combined with additional economic distress caused by the Great Recession, permeated entire family systems and disrupted the family-based safety net on which many working-age adults once relied. Communities hardest hit by the decline in manufacturing—especially in the textile, mining, and steel industries—experienced widespread unemployment, economic insecurity, diminished access to health care, and increasing individual- and familial-level stress. Recent cohorts of middle- and working-class young adults now face the realization that they will not live a better life than their parents, as the American Dream of intergenerational mobility has drastically declined (Cherlin, 2014; Chetty et al., 2017).

These shifts coincided with other social and cultural changes in American life. Jobs now required increasing educational credentials, resulting in rapidly increasing college enrollments but also burdening many young people, especially those lacking a 4-year degree, with college debt that caused both their finances and their careers to suffer (Velez and Woo, 2017; Wei and Horn, 2013). The digital era ushered in new lifestyles featuring ubiquitous, multiplatform, and uninterrupted access to online, electronic, and digitally streamed information and introduced new forms of social interaction and network formation (IOM and NRC, 2015). Individuals without the skills to access these forms of communication remained isolated from job opportunities and social interaction. Almost all forms of daily activity at work, at home, and in social groups were being accomplished at a faster pace than in prior generations (Tapscott, 2008; Vogels, 2019). The speed and volume of information in daily life created new stresses for those managing work, home, and family responsibilities and new forms of trauma related to body image, bullying, and other adverse experiences (McEwen and McEwen, 2017).

These daily, gradual, and long-term influences from the social and physical environments have contributed to cardiometabolic morbidity and mortality risks through multiple pathways, including the influence of financial hardships on health care access and affordability and on adverse living and work conditions that affect endocrine and cardiovascular diseases (Lian, 2018; Steptoe and Kivimäki, 2013). For example, job loss means loss of health insurance, and financial strains put out-of-pocket costs for health

care (and other household needs, such as housing, food, and transportation) out of reach. Individuals with chronic conditions and comorbidities cannot afford prescription medications, blood glucose testing, or doctor's appointments, and economic pressures force many families to resort to consuming inexpensive, calorie-dense foods; to live in unhealthy housing and neighborhoods; and to work in jobs that discourage physical activity. Job loss, family breakdowns, and economically depressed neighborhoods increase social isolation and erode social supports that are vital to health and longevity (House, Landis, and Umberson, 1988; Yang et al., 2016).

These cascading and multiplicative social, economic, and cultural changes have led to increasing levels of chronic stress with known adverse effects on cardiometabolic health. For example, adults who experience social isolation and workplace stress have a 1.5-fold and 1.3-fold increased risk, respectively, of CHD (Steptoe and Kivimäki, 2013). Numerous studies with rigorous repeated cross-sectional or longitudinal designs have found that chronic exposure to low socioeconomic status is associated with increased risk of the metabolic syndrome (Elovainio et al., 2011; Loucks et al., 2007; Manuck et al., 2010; Ramsay et al., 2008). Analyses of other stressors, including loneliness (Whisman, 2010), marital stress (in women but not men) (Whisman, Uebelacker, and Settles, 2010), and workplace stress (Chandola, Brunner, and Marmot, 2006), have found a longitudinal association with the onset of the metabolic syndrome, even after controlling for socioeconomic status. Chronic stress and psychological distress are also associated with the components of the metabolic syndrome, such as higher waist circumference (central obesity); high BMI (general obesity); and dyslipidemia, indicated by higher triglycerides and lower HDL cholesterol (Chandola et al., 2008; Kivimäki et al., 2009). Several meta-analyses likewise have found an association between stress and obesity (Nyberg et al., 2012; Wardle et al., 2011).

The increasing daily stresses that characterize contemporary society are felt more intensely among certain population groups. Racial/ethnic discrimination, lack of educational opportunities and economic advancement, and incarceration are the norm rather than the exception for some of the nation's most vulnerable groups. Additional stresses associated with social and economic inequality and exposure to structural racism and microaggression have been widely documented and are associated with higher rates of cardiometabolic disease and other adverse health outcomes (Chetty et al., 2020; Gee and Ford, 2011; Link and Phelan, 1995; Phelan and Link, 2015; Williams, 1999; Williams, Lawrence, and Davis, 2019). The stresses associated with the increased pace of daily life may be especially acute for younger adults who came of age in the digital era but lack financial or career stability (IOM and NRC, 2015). As discussed earlier, two decades of research has documented that exposure to adverse childhood experiences,

including poverty, leaves a lifelong imprint on the brain and body and undermines long-term health, increasing the incidence of substance abuse, heart disease, diabetes, and poor health behaviors (Flaherty et al., 2013; McEwen and McEwen, 2017; Su et al., 2015).

Chronic stress can cause both biological and behavioral responses that affect health. Repeated activation of the stress response system keeps levels of stress hormones elevated, which in turn increases levels of blood glucose, inhibits insulin production to prevent glucose from being stored, increases glucose intolerance, contributes to the buildup of fat tissue and weight gain, increases pulse rate and blood pressure, and damages blood vessels and arteries (Lupien et al., 2006; McEwen and Lasley, 2002; Seeman et al., 1997). Stress may also induce unhealthy coping behaviors, such as smoking, alcohol and drug abuse, and unhealthy eating habits that may affect cardiometabolic risks and help explain observed mortality trends (Geronimus et al., 2010; Jackson, Knight, and Rafferty, 2010). For example, the stress hormone cortisol triggers cravings for high-fat, sugary, and salty foods (Chao et al., 2017; Tryon et al., 2013), and the extra calories consumed as a result are converted to fat deposits that increase the risk of heart disease, diabetes, high blood pressure, and stroke.

Chronic stress can bring on feelings of despair and hopelessness. When individuals lack hope for their future and feel that they have nothing to lose, they may engage in unhealthy and risky behaviors because they are apathetic about their health or the risk of premature death (Gaydosh et al., 2019; Goldman, Glei, and Weinstein, 2018; Harris, Duncan, and Boisjoly, 2002). To the extent that despair and hopelessness are associated with chronic stress, an increase in those feelings may also play a role in increasing rates of obesity, hypertension, stroke, and diabetes, although as detailed in Chapter 7, the evidence that rates of despair have increased or are responsible for the increase in working-age mortality is inconclusive.

Substantial research has documented these profound shifts in U.S. social and economic conditions since the 1970s (Cherlin, 2014; McLanahan and Sandefur, 1994; Wilson, 1987) and accompanying cultural changes in American work and home lives in the digital era, but the ways in which such macro-level long-term changes affect cardiometabolic mortality are difficult to ascertain. Research is only beginning to illuminate how daily adverse environmental exposures that chronically activate the brain's stress management system can lead to dysregulation of individual and multiple body systems, including the immune, metabolic, and cardiovascular systems, involved in predisease pathways (Hertzman and Boyce, 2010; McEwen and Lasley, 2002; NRC, 2001). Whether such long-term shifts that have occurred over decades and are codependent and synergistic can lead to population-level changes in cardiometabolic mortality is unknown, and the research tools in the form of data, study design, and methodology with

which to address this broad and important question are not yet available. There is more evidence, however, for how certain population groups are more vulnerable to adverse social and economic changes and the associated biological consequences of chronic stressors, and this avenue of research may be more fruitful in policy and programmatic efforts to reduce such disparities and thereby achieve health equity within American society.

SUMMARY

The contribution of cardiometabolic mortality to the recent rise in working-age mortality is complex and involves several countervailing trends. Death rates due to ENM diseases and hypertensive heart disease generally increased during 1990–2017, and especially since 2010. While there have been significant long-term reductions in mortality from ischemic heart disease and other diseases of the circulatory system since 1990, much of that progress stalled after 2010. The combination of these trends acted to increase all-cause mortality after 2010 because the slowdown in mortality declines from ischemic heart disease and other circulatory diseases no longer offset the rise in mortality from ENM diseases and hypertensive heart disease.

Certain populations were especially vulnerable to rising cardiometabolic mortality. Mortality due to hypertensive heart disease increased among young adults, White adults, and Hispanic males during the 1990s and 2000s. Mortality due to ENM diseases increased during the 1990s among all working-age adults except younger (ages 25–44) Hispanics. Most younger working-age adults saw additional increases in mortality from ENM diseases through the 2000s.

The stalling of improvements in CVD mortality in the 2010s has exposed these troubling trends in cardiometabolic mortality. Since the 2009–2011 period, mortality from ischemic heart disease and other circulatory system diseases has remained largely unchanged among working-age adults, while mortality from hypertensive heart disease and ENM diseases has increased among all younger (ages 25–44) working-age adults except Hispanic females; all White working-age adults; and all working-age males, particularly Blacks. These worrisome changes in cardiometabolic mortality have been particularly pronounced in the South and in nonmetros and other areas outside of large central metros. Large central metros and the Northeast region of the country have generally experienced the most favorable trends in cardiometabolic disease mortality over time. Because of these trends, the gap in mortality by metropolitan size has grown over time, particularly among White working-age adults.

There are three potential explanations for these trends: the obesity epidemic, which has been identified as a major cause of increasing mortality

risks affecting multiple cardiovascular and metabolic systems; the diminishing returns of medical advances in lowering CVD mortality as the lagged cardiometabolic consequences of the obesity epidemic blunted the impact of medical innovations, and disparities in awareness of, access to, and use of drug therapies and smoking cessation persisted; and other social, economic, and cultural changes that increased multiple stressors among the working-age population, especially vulnerable subgroups. Although all three explanations have some support, the increased prevalence of obesity in the U.S. population is the most significant.

Obese adults are at higher risk of mortality from cardiometabolic diseases relative to nonobese adults; obesity increases the risks of hypertension, stroke, CHD, and type 2 diabetes. Rates of obesity have increased in a similar fashion among adults in all income and educational categories, such that over time, income and educational differentials have remained more or less the same: higher-income versus lower-income populations have a lower prevalence of obesity, and working-age adults with a college degree have a lower prevalence of obesity compared with their less-educated peers. As a result, changes in obesity are having little impact on socioeconomic disparities in working-age mortality trends.

The most proximate causes of obesity involve health behaviors that produce an imbalance between calories consumed and calories expended. The question remains as to what factors have led people to consume more and not expend as many calories as they are consuming. Much attention has been devoted to the role of the obesogenic environment, defined as an environment that promotes weight gain and is not conducive to weight loss within the home and environment. While substantial evidence indicates how Americans' physical environments have changed to become increasingly obesogenic, the multifactorial and complex pathways by which these changing environments have increased the level and spread of obesity and, by extension, the rise in mortality from ENM diseases and hypertensive heart disease or the slowing declines in mortality from ischemic heart and other circulatory diseases represent major research challenges.

Medical advances in drug development and prevention, treatment, and control of chronic diseases, together with increasing knowledge about the health effects of cigarette smoking, played a large role in the long-term decline in mortality from ischemic heart disease and other circulatory diseases that began in 1970, but their impact lessened after 2010, when progress in reducing CVD mortality stalled. The diminishing returns of medical advances after 2010 appear to be due to the lagged cardiometabolic consequences of the rise in obesity to which adults of working age had been exposed for 30 years. In particular, young adults approaching midlife have been exposed to obesogenic environments their entire lives and are experiencing severe cardiometabolic consequences (e.g., rising rates

of diabetes, hypertension, and CVD) compared with their peers of prior generations. Indeed, the rising trends in mortality from ENM diseases and hypertensive heart disease among young adults reflect this cohort effect of the obesity epidemic. Therapeutic approaches can address the cardiometabolic consequences of obesity, but disparities in access to and uptake of these treatments may also have slowed progress in reducing CVD mortality. Increasing investments in closing the prevention and treatment gap in high-risk populations could help reduce these disparities to achieve greater equity in therapeutic care and spur continued reductions in CVD mortality.

Finally, social, economic, and cultural changes over the past 50 years represent a natural progression among all advanced societies around the world. These changes have increased the pace and efficiency of work and social interactions but have also necessitated more education, training, and technological skills to keep up with the faster pace of life and dwindling opportunities for social mobility. These shifts have left those without the necessary education and training behind, as more vulnerable populations have lost the ability to find decent-paying jobs to support a family or live in a good neighborhood. Falling behind exacts a biological and emotional toll on those with less education; racial/ethnic minorities; those living in rural areas; and young adults in particular, who lack the resources to avoid the multiple daily stressors that can slowly and permanently damage their endocrine, metabolic, and cardiovascular systems and thereby increase their mortality risks. While research has established these links between chronic stress and CVD risks, it has not provided evidence that long-term social, economic, and cultural changes have brought about the recent changes in cardiometabolic mortality.

IMPLICATIONS FOR RESEARCH AND POLICY

The evidence suggests that increasing obesity is the most important explanation for rising rates of cardiometabolic diseases, including diabetes, hypertension, and CVD, in the United States, driving up rates of mortality from ENM diseases and hypertensive heart disease and stalling progress in mortality due to ischemic heart disease and other circulatory diseases. Moreover, research indicates that the earlier in life one becomes obese, the earlier and more severe are the adverse health consequences because most people who become obese do not become nonobese in their lifetime (Harris et al., 2020). Thus the promotion of healthy weights and the prevention of obesity are a critical research and policy priority. This priority is not new; what is new is mounting evidence that obesity is a key driver of cardiometabolic morbidity and mortality trends, especially among young working-age

adults, and will continue to be so for some time in the future unless progress is made in reducing obesity in the population and disparities therein.

The 2012 National Academies' report *Accelerating Progress in Obesity Prevention: Solving the Weight of the Nation* (IOM, 2012a) advances a number of policy recommendations regarding obesity prevention that are highlighted here as starting points for the committee's research recommendations. That report uses a systems perspective to advocate for change in the environments related to five areas for obesity prevention: environments for physical activity, food and beverage environments, messaging environments, health care and work environments, and school environments. Similar to the committee's conceptual framework in Chapter 6 (Figure 6-1), the 2012 report outlines the ways in which upstream environmental change in societal messaging about (un)healthy food and diets filters down to and influences the meso-level environments of the physical neighborhood, schools, health care, work, and food and beverage consumption in independent and overlapping ways to affect obesity among individuals nested within families and communities. The present report views obesity prevention through the same lens and calls for the use of a life-course, multilevel, multifactorial approach to identify the multiple interactive causal pathways and drivers most responsible for the rise in obesity. In 2021, the research community is in a better position to undertake this work than it was in 2012, with new and additional data sources needed to apply this life-course, multilevel approach.

Research applying such an approach is needed to enable the development of a multipronged public policy strategy for mitigating this major public health problem. Almost all obesity scholars point to the important role of obesogenic factors in the physical and food environments, including individual health behaviors involving diet and physical exercise and societal-level changes in food production, transportation systems, access to green spaces, and sedentary work environments. For example, there is evidence that technological changes in the way food is produced, distributed, and consumed have contributed to the increase in obesity, and that public health policy can play a role in improving the production of healthy foods and reducing the distribution and consumption of unhealthy foods, especially among children and adolescents. The research reviewed in this chapter also documents the success of healthy diets in achieving *short-term* weight loss, and regular exercise is almost always beneficial in reducing overweight and obesity. However, efforts to maintain lifestyle changes that support healthy diets and programs to promote regular exercise (e.g., worksite health promotion programs, free gym memberships) may not always work or be sustainable. Thus, research is needed to explore the factors that erode short-term successes in diet and exercise changes and, conversely, the factors that promote long-term lifestyle changes that can reduce obesity, with a focus

on environmental drivers (e.g., occupational activity, access to recreational space and activities; exposure to chemicals, food deserts, economic inequality, residential segregation, duration of use of electronic screens, increased consumption of medications). In sum, the committee recommends research to evaluate the effectiveness of programs and policies designed to improve cardiometabolic health using a multilevel framework.

Many programs and policies implemented at the societal or community level are upstream and vary in their impact on individuals' behavior and health because the mechanisms through which they operate vary by local context and individual characteristics. Since the 2012 National Academies' report was issued (IOM, 2012a), more data sources enabling examination of how macro-level policies and programs are linked to individuals and the environments in which they live and work have become available with which to conduct the multilevel research recommended in the present report. For example, several national longitudinal studies, such as the Health and Retirement Study, the National Longitudinal Study of Adolescent to Adult Health (Add Health), the Fragile Families and Child Well-Being Study, the Panel Study of Income Dynamics, and the National Longitudinal Study of Youth, provide data at multiple levels (state, county, census tract, family, individual) and measure behavioral and health outcomes of individuals throughout their life course. With such multilevel longitudinal data, researchers will be able to identify the intervening multilevel mechanisms (e.g., family poverty, consumption of sugary drinks, physical activity) through which macro-level policies and programs (e.g., soda taxes, urban development) operate to affect cardiometabolic morbidity and mortality.

RECOMMENDATION 9-1: Federal agencies, in partnership with private foundations and other funding entities, should support research that evaluates the effectiveness of programs and policies designed to improve U.S. cardiometabolic health and that considers the impact of changes at multiple levels of analysis:

- **At the individual level, research should continue to evaluate the effectiveness of programs and policies that promote consumption of healthy foods (e.g., mandatory labeling of food ingredients or components, fruit and vegetable subsidies) and the adoption of healthy lifestyles (e.g., subsidies for sports activities; urban development that prioritizes walking, biking, and transit). Likewise, research should continue to evaluate the effectiveness of programs and policies that discourage the consumption of poor-quality foods (e.g., sugar and soda taxes, nutritional standards and dietary guidelines from the U.S. Department of Agriculture and the U.S. Department of Health and Human Services) and unhealthy lifestyles (e.g.,**

insurance rating based on poor health habits such as smoking, zoning laws for fast food restaurants and alcohol outlets).

- **At the societal level, research should consider systemic changes in food production, workplace systems, and transportation and other societal-level changes in the United States that foster and sustain obesogenic environments and sedentary lifestyles to determine the pathways through which such environments have deleterious consequences for population health.**

Identifying the upstream to downstream factors that can explain how the physical environment leads to rising obesity and thus to rising cardiometabolic morbidity and mortality is a research priority, but it is a challenging one posing substantial data demands and requiring longitudinal studies or well-designed experiments and interventions. Although experimental designs tend to lack generalizability, they provide some of the strongest evidence of causal impacts—a major gap in the current literature on the effects of obesogenic factors in the environment. The committee therefore endorses experimental research designs that examine the impact of changes in such obesogenic factors as access to green space, walkability, dependence on automobiles, density of fast food restaurants and alcohol outlets, and availability and promotion of high-calorie and processed foods on changes in obesity prevalence and BMI levels. There may be opportunities to take advantage of existing neighborhood experimental and evaluation projects, such as Moving to Opportunity or the New York City Housing and Neighborhood Study, by linking built environment data elements to the pre- and post-neighborhood geographic locations of families in these studies.

RECOMMENDATION 9-2: Federal agencies, in partnership with private foundations and other funding entities, should support research that uses experimental designs and takes advantage of existing neighborhood experimental projects to examine the causal role of factors in the obesogenic environment and determine which have the greatest role in the rise in obesity prevalence and body mass index levels.

The food industry and policy makers alike have contributed to the obesity epidemic in both intentional and unintentional ways. The food industry has successfully leveraged advertising and marketing techniques to boost consumption of calorie-dense foods. The U.S. Department of Agriculture, which for decades has promulgated dietary guidelines to promote healthy eating and combat obesity, issued farm subsidies in the early 1980s that generated an oversupply of corn, thereby lowering its cost and encouraging the production of high-fructose corn syrup, the major sweetener added to the food supply. The use of high-fructose corn syrup and the wide distribution

of low-cost, calorie-dense foods have combined to raise U.S. caloric intake per capita far above that of other high-income countries. Some efforts are under way to regulate food advertising, tax unhealthy foods and beverages, mandate product labels, list calorie content on restaurant menus, and implement zoning of fast food restaurants and alcohol outlets. But further action on the part of both the food industry and public health authorities is needed to discourage the production and purchase of unhealthy foods, or at least provide consumers with better information with which to make healthier food choices. Tighter regulations to improve the nutritional content of foods and beverages, prohibit misleading advertising, alter pricing to encourage the consumption of healthy foods, or use zoning and land use policies to influence the location of food stores and restaurants could make it easier for the public to make healthier dietary choices but also encroach on the free market rights of the food and restaurant industries.

POLICY CONCLUSION 9-1: To reduce the per capita calorie consumption and body mass index levels of the U.S. population, policy makers will need to implement laws and regulations that preserve a healthy balance between the rights of the food industry, advertisers, grocers, and restaurants to enjoy free market competition and the public health imperative to limit the promotion and consumption of foods and beverages that contribute to obesity.

Mounting evidence shows that the current generation of young adults nearing midlife (i.e., those born in the late 1970s and early 1980s), who have spent their entire lives amid an obesity epidemic, is at high risk of morbidity and mortality from obesogenic conditions. Those born in the 1990s, 2000s, and 2010s have also experienced an obesogenic environment their entire lives, with foreboding health consequences for future cohorts of young and middle-age adults. Obesity needs to be avoided in early life because any exposure, and especially prolonged exposure, can greatly increase risks for cardiometabolic diseases and mortality. In addition, the early stages of chronic disease associated with obesity often present as asymptomatic risk factors (e.g., hypertension, hyperlipidemia, glucose intolerance), especially among young adults for whom permanent biological damage has not yet manifested in disease. Young adults often fall through the cracks of regular medical care if they do not have stable employment or health insurance that provides for regular health care screening. U.S. cohort studies make clear that the obesity epidemic has made the cardiometabolic health of young adults today much worse than that of their peers in previous cohorts. The alarming health status of today's young adults portends a shorter work life, with societal consequences for productivity and growth, and escalating medical care costs for individuals, families, and society

as a whole. The committee therefore believes that prevention of obesity should focus on children and young adults, especially those in vulnerable populations.

RECOMMENDATION 9-3: Designers of obesity prevention programs should focus on developing programs that start early in life and target children and adolescents most at risk of obesity (e.g., racial/ethnic minorities, females, people living in poverty and in neighborhoods of low socioeconomic status) and those who are overweight or gaining weight, thus intervening before obesity trajectories become set throughout adulthood.

Persistent disparities in access to evidence-based preventive and therapeutic care for the control of chronic diseases hinder continued progress in lowering CVD mortality. Appropriate use of medications that are effective in controlling hypertension, diabetes, and heart disease and thus in lowering CVD mortality is not as widespread as would be ideal, and persistent disparities in successful management of these chronic diseases continue to slow progress in mortality reduction. Because much of this shortfall may result from individuals not fully understanding how to care for their conditions (because of inadequate instructions, language barriers, or poor communication) or being unable to implement or maintain care (because of challenges with accessing and affording medications, providers, and other resources), improved systems for solving these challenges is an important priority for continued improvement in cardiometabolic mortality. Efforts to improve systems for delivering existing treatments might yield more benefit in lowering mortality relative to incremental gains from innovative new treatments. For cardiometabolic conditions in particular, more implementation research is needed to better understand barriers to accessing and maintaining effective control and use of highly effective medications and other treatments. A priority for such research is to design solutions that address the needs of high-risk populations, including communities of color and less-educated and lower-income populations who face higher rates of hypertension and other cardiometabolic risk factors, whose conditions often escape early detection, and who face added challenges in accessing information and clinical services to treat these conditions. Further research likewise is needed to address the barriers to smoking cessation faced by populations that continue to smoke at high rates, especially those of lower socioeconomic status.

RECOMMENDATION 9-4: To improve systems for delivering preventive care (e.g., smoking cessation) and existing treatments for hypertension, diabetes, and heart disease, federal agencies, in partnership

with private foundations and other funding entities, should support research focused on better understanding the barriers to prevention and control of cardiometabolic disease faced by individuals—especially less-educated and lower-income populations—and evaluating potential solutions for removing those barriers.

Finally, social, economic, and cultural shifts over the past 50 years have profoundly changed the family, work, and community environments of daily life in the United States, and many of these changes have increased stress in the lives of Americans. This increasing and chronic stress may be contributing to the rising trends in mortality documented in this report, including deaths due to substance use, suicide, and cardiometabolic diseases. The daily sources of stress are not likely to abate, and they are felt more intensely among certain subgroups of the population. Exposure and responses to stress are strongly graded by socioeconomic status such that those with less education, lower income, greater job instability, and long-term unemployment experience much more stress in their lives and are more likely than those of higher socioeconomic status to engage in unhealthy behaviors (e.g., smoking, alcohol and drug use, overeating of ultraprocessed foods, self-harm) as a way of coping with that stress. Policy approaches in this realm include upstream macro-level approaches to reduce downstream socioeconomic disparities and promote healthy behaviors that lessen stress (e.g., exercise, social interaction, sleep, meditation). The committee therefore developed a cross-cutting recommendation for more research to explore the sources of increasing stress in the lives of Americans and to identify which population subgroups are most affected by that stress (see Chapter 11, Recommendation 11-5).

10

The Relationship Between Economic Factors and Mortality

The data presented in previous chapters suggest that social and economic factors have contributed to mortality trends in the United States. During the study period (1990–2016), for example, the increase in working-age (ages 25–64) mortality was greatest among adults with less education. As shown in Chapter 3, large increases in working-age mortality occurred in the industrial Midwest and central Appalachia, areas deeply affected by the collapse of manufacturing plants and coal mines, on which many communities depended for economic vitality and stable employment. Working-age mortality rates increased during a period in the United States in which middle-class and low-income people faced reduced access to well-paying jobs, rising housing and health care costs, and difficulties ensuring that their children could obtain a good education and climb the economic ladder. Research has shown that exposure to prolonged economic adversity may affect health outcomes via multiple mechanisms, including gaps in health care; chronic stress;[1] anxiety; depression; and unhealthy coping behaviors, from smoking and overeating to drug and alcohol abuse, suicide, and violent crime.

The complex interrelationships among economic conditions, place, and time, together with the inability to conduct controlled experiments, make it difficult to prove causal associations or confidently isolate the health impacts of economic trends (Gonsalves, 2019). Nevertheless, a growing body of literature provides some insight. For example, ethnographic

[1]Chronic stress is itself biologically harmful to health, with known effects on neuroendocrine function, the immune system, and epigenetic transmission.

studies reviewed by the committee document the withering influence of economic pressures on the health of communities and their vulnerability to chronic stress, anxiety, substance abuse, depression, and suicide (Chen, 2015; McLean, 2016; Silva, 2019; Thompson et al., 2020). Much of the other empirical research examines changes in overall mortality, but some focuses on specific causes of mortality, with growing interest in deaths due to suicide and substance abuse. This chapter examines evidence of the relationship between selected economic factors and mortality. The focus is on general economic conditions, economic fluctuations in employment, plant closures, trade pressure, and economic inequality. Related discussion of policies intended to improve economic well-being (e.g., minimum wage laws) is included in Chapter 11.

CONCEPTUAL CHALLENGES

Assessing the relationship between economic conditions and mortality is difficult for a number of reasons. First, the impacts of intermediate- and short-term economic changes may differ from those of more sustained changes, and empirical analyses of long-term impacts are challenging because of many intervening factors. Second, confounding and complex interactions may mask effects. For example, economic deprivation may be correlated with education, making identification of the pure effects of economic well-being difficult to isolate.

Moreover, different demographic groups may experience economic conditions differently, and economic effects may interact with other time-varying contextual and environmental factors, such as the availability of opioids. For example, economic factors may give rise to physical and psychological pain, but the connection between that pain and mortality may reflect the introduction of opioids. Thus the combination of pain and opioids may have created conditions that led to significant mortality, while the absence of either might have greatly dampened the dramatic rise in mortality that occurred.

Finally, the effects of changes in economic conditions may vary based on the reasons behind the changes. The effects of plant closings, for example, may differ from those of cyclical recessions. Likewise, broad-based economic decline in communities may have a broader effect relative to individual economic setbacks.

ASSOCIATION BETWEEN ECONOMIC DEPRIVATION AND WORSE, AND RISING, MORTALITY

A considerable body of research demonstrates that lower-income Americans have worse outcomes relative to their wealthier counterparts. Thus,

adults ages 50 and above living in poorer areas have higher mortality rates than those living in wealthier areas (Currie and Schwandt, 2016). The difference in life expectancy between the wealthiest 1 percent and the poorest 1 percent of individuals in the United States is approximately 15 years (Chetty et al., 2016). The increase in life expectancy from 2001 to 2014 was not uniform: life expectancy increased by 2.34 years and 2.91 years for men and women, respectively, in the top 5 percent of the income distribution but by only 0.32 year and 0.04 year for their counterparts in the bottom 5 percent. Within the bottom income quartile, life expectancy was approximately 5 years longer in geographic locations with the highest versus those with the lowest longevity. The lower life expectancy among the latter individuals was significantly correlated with health behaviors such as smoking, but not with physical environment factors, labor market conditions, income inequality, or access to medical care. Life expectancy for these individuals also was correlated with government expenditures, fraction of college graduates, and fraction of immigrants.

Seminal research by Case and Deaton (2015) documents trends in mortality associated with economic status. Specifically, using death records and national survey data, these authors found that among those ages 45–54, mortality among non-Hispanic Whites (Whites) rose by 34 per 100,000 population between 1999 and 2013—approximately 0.5 percent per year. This increase in working-age mortality was driven largely by increases in drug and alcohol poisoning and suicide, known as "deaths of despair" (see Chapters 7 and 8, respectively), among those with a high school education or less, along with a slowdown in progress against mortality from heart disease and cancer, the two leading killers in middle age (see Chapter 9). These trends were accompanied by increases in morbidity, including deterioration in self-reported physical and mental health, and rising reports of chronic pain. Case and Deaton (2017, 2020) later extended their analysis and proposed a hypothesis whereby poor labor market conditions and cumulative disadvantage experienced by successive cohorts (delineated by birth year) can help explain this increased mortality (Case and Deaton, 2017, 2020). These basic associations are supported by the committee's analysis.

Although the evidence relating economic disadvantage to mortality is strong, the relationship is complex. Some studies have found the effects of economic despair on mortality for Whites but not for non-Hispanic Blacks (Blacks) or Hispanics (Hollingsworth, Ruhm, and Simon, 2017; Pierce and Schott, 2020). Case and Deaton (2017, 2020) found that although mortality is higher in an absolute sense among Blacks than Whites, mortality rates among Blacks still improved until about 2010. Thereafter, absolute year-to-year increases in working-age mortality among non-White populations during the study period (1990–2017) matched or exceeded those among Whites, suggesting that past differences by race may have reflected timing more than

fundamental protections from the forces that contributed to increased mortality among working-age adults. The committee's analysis supports the notion that recent trends in increased mortality extend beyond Whites.

Moreover, while drug (opioid) overdoses accounted overwhelmingly for rising working-age mortality during the study period, alcohol-related diseases, suicides, unintentional injuries, and organ diseases contributed as well. The breadth of factors contributing to increases in working-age mortality in the past decade—from external causes such as substance abuse to diseases involving multiple body systems and pathophysiological processes—eludes attribution to a single cause, such as opioids or obesity. Rather, it suggests the possibility of one or more upstream systemic causes, such as economic disadvantage or increasing stress that can affect health across multiple pathways and disease processes (Woolf et al., 2018). Yet as noted above, the relationship between mortality and economic disadvantage is complex, and it may be exacerbated or ameliorated by mediating factors. Different groups may experience economic deprivation differently, or economic well-being relative to expectations may matter more than economic well-being per se. But in any case, differential effects by geography and race suggest a more complex story than the conclusion that economic deprivation leads to increases in mortality.

One way to isolate the impact of economic conditions on mortality is to examine the effects of specific economic shocks. Research typically focuses on two types of shocks: job loss, particularly when caused by plant closings, and trade/import competition.

Job Loss and Deaths of Despair

One body of quasi-experimental analysis looks at specific economic shocks at the individual level and tends to find that such shocks lead to poor health and mortality outcomes. One such shock examined in the literature is job loss. The literature suggests that the immediate impact of losing a job does not explain suicide risk, but being part of a mass layoff event or being unemployed for an extended period of time appears to be associated with increased suicide risk (Classen and Dunn, 2012). Sullivan and von Wachter (2009) suggest that for men, late-career job loss is associated with mortality rates in the year after displacement that are 50–100 percent higher than would otherwise be expected. The effect on mortality declines sharply over time, but even 20 years after displacement, the authors estimate a 10–15 percent increase in annual death hazards. In their study, they compare mortality rates among displaced workers with those among their nondisplaced peers and also find that workers with larger losses in earnings see more substantial increases in mortality rates relative to those with smaller earnings losses. A related study from the Netherlands found that after controlling for

worker characteristics, workers who lost their jobs because of firm closure had a 34 percent or 0.60 percentage point increase in probability of death within 5 years (Bloemen, Hochguertel, and Zweerink, 2018).

In other research, data from Denmark suggest that job loss due to a plant closure, particularly in the few years following the loss, increases the risk of overall mortality and of death from circulatory disease; suicide and suicide attempts; and death and hospitalization due to traffic accidents, alcohol-related disease, and mental illness (Browning and Heinesen, 2012). According to these data, the risk of overall mortality is 79 percent higher in the year of displacement, 35 percent higher 1–4 years after displacement, 17 percent higher 1–10 years after displacement, and 11 percent higher 1–20 years after displacement. Importantly, Venkataramani and colleagues (2020) document that the increase in opioid mortality between 1999 and 2016 was significantly greater in manufacturing counties that had experienced the closure of an automobile plant (Venkataramani et al., 2020). They estimate that 5 years after a county experiences a plant closure, mortality due to opioid overdoses will increase in that county on average by 8.6 deaths per 100,000 population compared with unexposed counties, an 85 percent increase relative to the preclosure mortality rate.

Overall, job loss appears to have negative impacts on certain health-related behaviors. However, employability (and the ease with which those laid off can find new employment) does appear to mitigate the effects of job loss, so these effects may be concentrated among an at-risk subset of workers (Deb et al., 2011).

Trade/Import Competition and Mortality

Another line of quasi-experimental research examines the impact on health of adverse economic conditions attributable to international trade. Specifically, while international trade may be beneficial for overall economic growth, increased trade can prove disruptive to the domestic manufacturing industry, resulting in layoffs, plant closings, and reduced earnings for exposed workers (Autor et al., 2013). The particular role of import competition is important. Studies of the relationship between imports and mortality have found that areas more versus those less affected by imports experience worse health outcomes. For example, a $1,000 increase in competing Chinese imports per worker corresponds to a 7.8 percent increase in the morbidity of poor mental health, adding approximately 3 days of poor mental health per year for the average adult (Lang, McManus, and Schaur, 2019). Additionally, firms particularly susceptible to import competition may make decisions that put workers' health at greater risk. Injury risk, for example, increases by 13 percent at the smallest plants (McManus and Schaur, 2016). Thus it is important to look at the effects of economic

shocks, which may be sudden and geographically concentrated, on those most acutely affected.

Taking advantage of differential exposures to trade liberalization due to Congress's granting of Permanent Normal Trade Relations (PNTR) to China in 2000, Pierce and Schott (2016) found an increase in mortality due to "deaths of despair." Counties in the 75th percentile of exposure to PNTR had a 0.42–0.63 higher suicide mortality rate per 100,000 population compared with those in the 25th percentile, accounting for 4–6 percent of the average age-adjusted suicide mortality rate across counties. Likewise, shifting a county from the 25th to the 75th percentile of exposure to PNTR was associated with an almost 30 percent higher mortality rate from accidental poisonings, which include drug overdoses. This increase in drug-related mortality was observed across a large portion of the working-age population (most age bins in the 20–54 age group). Importantly, this association was observed only among Whites, consistent with the notion that non-Whites are less affected by job loss (see Chapter 8). Similarly, focusing on young adults ages 18–39, Autor, Dorn, and Hanson (2019) found an increase in mortality due to increased import exposure. According to these authors, the average decade-level rise in import exposure induced an additional 64.4 male relative to female deaths per 100,000 population (of each gender) per decade (Autor, Dorn, and Hanson, 2019). Furthermore, manufacturing trade shocks were found to cause significant increases in male mortality due to drug and alcohol poisoning, HIV/AIDS, and homicide (Autor, Dorn, and Hanson, 2019).

LACK OF A STRONG ASSOCIATION BETWEEN ECONOMIC FLUCTUATIONS AND INCREASED MORTALITY

Despite clear evidence relating economic disadvantage to mortality and demonstrating the deleterious economic consequences of job loss and trade competition, market-level economic fluctuations due to the business cycle are not strongly associated with increased mortality, including that related to opioids, at the population level. Although one might expect worse economic conditions to lead to worse health outcomes, a large body of literature suggests a potentially counterintuitive effect—that increases in the unemployment rate actually lead to a decrease in mortality. Ruhm (2000), for example, estimates that a 1 percentage point increase in the state unemployment rate decreased total mortality by 0.5 percent. Similar patterns appear to hold in other industrialized countries, so this effect apparently is not unique to the United States (Gerdtham and Ruhm, 2006). These results appear to be explained in part by a reduction in the motor vehicle deaths and other accidents that occur among those younger than 45

during economic upswings. In addition, nursing homes experience severe shortages of aides when the economy is strong, explaining why higher mortality among the elderly is expected (Stevens et al., 2015). Despite finding decreases in mortality during economic downturns, however, Ruhm (2000) did find an exception for suicides, estimated to rise by 1.3 percent with a 1 percentage point increase in the state unemployment rate. Effects on mental and behavioral health and physical health thus appear to differ.

The evidence relating fluctuations in economic conditions and drug-related mortality is mixed. Research from the United Kingdom has shown that a 1 percentage point increase in unemployment leads to an increase of 18–25 percent in opioid doses prescribed per capita (Vandoros, Gong, and Kawachi, 2020). Relatedly, another study found that as the annual county unemployment rate increased by 1 percentage point, the opioid death rate increased by 3.6 percent, and the rate of emergency department visits due to opioid overdose increased by 7.0 percent (Hollingsworth, Ruhm, and Simon, 2017). Quasi-experimental studies using somewhat longer periods of analysis suggest a weaker effect. Indeed, Ruhm (2018b) found that after controlling for various demographic and geographic variables, changes in economic conditions (including unemployment rates and import exposure) explained less than one-tenth of the observed increase in drug deaths that occurred from 1999 to 2015 and even less of the growth in mortality involving opioid analgesics or illicit opioids. Ruhm's analysis differs from those of other authors cited earlier, such as Hollingsworth, Ruhm, and Simon (2017), because of the nature and length of the economic changes studied. The latter studies link changes in mortality to short-term economic changes (often in the local unemployment rate). By contrast, Ruhm (2018b) looks at medium-term economic changes that incorporate changes in a 3-year average poverty rate, in a 3-year average of median household income in 2015 dollars, in a 4-year average of median home prices, and in a 3-year average of the unemployment rate, as well as a measure of import competition.

The evidence that excess mortality is concentrated in disadvantaged populations but that fluctuations in economic conditions are related only weakly to mortality may appear contradictory. Yet these findings are consistent with the view that economic disadvantage makes populations susceptible to developments that threaten health. Over the past decades, the most significant of such developments was the introduction and diffusion of new opioids. Because disadvantaged populations suffer more from pain relative to the more advantaged, they would have been prescribed more pain medications during this period and may have been more susceptible to the opioid crisis as a result (Poleshuck and Green, 2008).

ASSOCIATION BETWEEN INCOME INEQUALITY AND MORTALITY

The relation between income inequality and health is complex, as it can involve several different causal and noncausal processes. A number of studies have shown that areas (e.g., countries, regions, cities, neighborhoods) with higher income inequality tend to have worse population health (e.g., higher mortality) (Backlund, Sorlie, and Johnson, 2007; Beckfield, 2004; Ben-Shlomo, White, and Marmot, 1996; Kaplan et al., 1996; Kennedy, Kawachi, and Prothrow-Stith, 1996; Kondo et al., 2009; Wilkinson, 1992; Wilkinson and Pickett, 2010). At least three different processes could contribute to these associations.

First, it is well established that income (at the individual or family level) is strongly related to health in a graded fashion (although a threshold effect and possibly declining benefit at high incomes have been documented in some studies) (Braveman, Egerter, and Williams, 2011). Because areas with higher income inequality often have larger proportions of the population at lower income levels, areas with more inequality will have worse health simply because of differences in the distribution of income.

Second, it has been hypothesized that income inequality may itself be related to worse health (even after accounting for absolute income) because of the psychosocial consequences of living in an unequal society. Living in a society that is more unequal could worsen health because the experience of inequality generates stress and reduced social cohesion and trust (Daniels, Kennedy, and Kawachi, 2000; Hastings, 2018; Subramanian and Kawachi, 2004; Wilkinson, 1996). Variants of this hypothesis posit that these adverse psychosocial impacts of income inequality are more pronounced (or only present) in persons of lower income, who therefore experience more adverse effects from living in a more unequal society.

Third, it has been hypothesized that income inequality may be related to worse health (even after accounting for absolute income) because societies or areas with less income inequality tend to have more egalitarian social policies or investments that benefit population health (Kawachi and Kennedy, 1999; Kawachi et al., 1997). According to this hypothesis, it is not the inequality that is the problem but the lack of public response to it. As with the hypothesis focused on inequality as a stressor, it has sometimes been posited that these effects are more pronounced in persons of lower income, for whom the social safety net is more important. Of course, an interesting question is whether lower income inequality is the cause or the consequence of these more egalitarian policies.

Research on whether there is or is not an income inequality effect on mortality (or other health outcomes) that is independent of absolute income has generated substantial debate (Lynch et al., 2004; Mellor and Milyo,

2001). Much of the debate hinges on whether analyses properly account for other population-level factors. For example, Deaton and Lubotsky (2003) argue that studies demonstrating a state income inequality effect did not properly control for race or other population-level characteristics that are differentially distributed across states and correlated with inequality. Deaton (2003) suggests that inequality may not be the driving factor so much as the correlate of more fundamental driving factors (Deaton, 2003). Others, however, have presented evidence that disputes these arguments (Ram, 2005; Wolfson and Beall, 2017).

Regardless of whether there is or is not an identifiable independent effect of income inequality on health (after individual-level income is accounted for), what is clear is that societies with larger income inequality tend to have worse health.

SUMMARY

The relationship between economic conditions and mortality is complex. Descriptive analysis of trends suggests that disadvantaged White populations relative to other racial/ethnic groups have experienced a disproportionate increase in mortality, but recent data suggest that other disadvantaged populations have experienced worse mortality trends as well.

The evidence related to the impact of a change in economic conditions on mortality is less conclusive. Quasi-experimental analyses of job loss, plant closings, and disruption from foreign trade support the notion that economic disruption increases mortality. But these studies focus on assessing changes in economic well-being due to a narrow set of causes, such as plant closings and trade disruptions, as opposed to such broader causes as technical change and general economic trends related to productivity, including automation and a host of related processes. Furthermore, not all findings from these analyses are robust across specifications. Other analyses examining changes in county-level economic conditions and mortality have found little effect of economic factors on drug mortality (a major component of increased working-age mortality), but a major impact of opioid availability.

The best interpretation of the relationship between economic well-being and mortality suggests that economic hardship is associated with higher mortality, especially in the context of widespread availability of potent and life-threatening medications. However, the overall impact of the direct economic shocks that have been examined (i.e., short-term changes in economic circumstances) may be modest, and more work is needed to quantify the magnitude of this impact. A more important connection between economic deprivation and mortality may arise as people of lower socioeconomic status (SES) are exposed to a series of such shocks, whose cumulative

effect is large even if each individual shock is small. These individuals also may be more susceptible to adverse events (e.g., the introduction of opioids) when they occur. For example, deterioration in economic conditions may have led to some excess mortality, but the greater effect is that as opioids were introduced, lower-SES individuals were more affected. This pattern may be causal and specific to opioids because lower-SES individuals were more likely to have pain and thus more likely to be prescribed opioids, or it may be a general phenomenon whereby lower-SES populations are more affected when events increase overall mortality.

IMPLICATIONS FOR RESEARCH AND POLICY

Much more work remains to be done to investigate the relationship between economic factors and mortality (see Recommendation 11-6 in Chapter 11). Economic research highlights several economic factors that influence income and may thus influence health: technological change, including automation; expansion of global trade to countries with lower wages; and policy changes, including making unionization more difficult and increasing the pay of chief executive officers (Autor, 2010). There is significant economic debate about the importance of each of these factors. The general sense is that technological change is likely the most important issue in differential wage trends by demographic group, but there is debate around this point.

For understanding health trends, the key issue is how these changes influence economic outcomes and health among different population groups, directly or indirectly. As technology changes, it—along with other factors—affects the income distribution and returns to education. More broadly, technological changes affect the nature of work, which in turn affects the stability of employment relationships and the overall financial risk faced by individuals over the course of their lifetime. Understanding the impact of this risk, as well as the effects of frequent job transitions on health, is important.

A second area of needed research is understanding how economic conditions interact with other factors in affecting mortality. For example, changes in medium-term economic conditions in themselves do not appear to have led to a very large increase in opioid deaths, but the increase in opioid availability may have contributed to a large increase in mortality because of a reservoir of susceptible individuals resulting from economic issues.

A third important set of questions involves understanding the relationship between the duration of economic hardship and mortality. Short-term or transient economic shocks may be less salient than longer-term declines in economic well-being. Similarly, the variability of economic status itself may have important consequences for health that are not well understood.

A fourth issue that deserves more analysis is how public programs to alleviate economic deprivation affect mortality (see Chapter 11 for a discussion of specific policies designed to improve economic well-being, such as the Earned Income Tax Credit and an increase in the minimum wage). The benefits of employment may extend beyond the associated economic rewards, and replacing income earned from wages with public support may not lead to the same outcomes.

Another important set of research questions is related to how economic well-being is measured, questions that are important both substantively and methodologically. Taking the example of how to measure economic hardship, many studies in this area focus on unemployment rates. Yet unemployment is measured relative to the labor force, and adverse economic conditions may cause people to leave the labor force. Conceptually, therefore, it may be better to measure the employment rate among working-age adults. Similarly, many measures of economic well-being focus on income levels, but relative income may also matter. Individuals with a given level of income may be worse off if they previously were wealthier than if they always had that income. Similarly, although research has examined the role of economic inequality in health, more work could be done to examine whether measuring absolute income or income relative to others in the community is a better predictor of health outcomes. Research is needed to illuminate how income should be measured—absolute or relative—how to count access to social programs, or whether to use point-in-time versus lifetime income. Finally, it is important to assess the extent to which individual-level versus community-level income (or wealth) may be important because the latter may influence a range of factors that affect people beyond their individual incomes, such as area traits (e.g., availability of fresh food) and tax revenue that can be used to fund social supports.

PART III

11

Implications for Policy and Research

This chapter provides background for and develops the policy and research implications of the committee's analysis and findings. Policy implications encompass how the evidence in this report suggests the need for changes in policy with the potential to curb the recent increase in working-age mortality and/or narrow disparities in working-age mortality in the coming years. The committee stresses the immense challenge of predicting policy impacts in this area of science. As reviewed throughout the report, studies of mortality trends and patterns, especially at the national level, rely almost exclusively on observational data and federal statistics. As a result, causal evidence in this area is limited, and controlled experiments are difficult, if not infeasible. Moreover, as also discussed throughout the report, the key hypothesized influences on working-age mortality patterns and trends are numerous and operate concomitantly at multiple levels. Many of the proposed drivers operate across the life course and/or across decades—in either period or cohort fashion—to influence mortality patterns and trends. This report therefore focuses on an exceptionally complex set of patterns, trends, and explanations for which clear or simple solutions are lacking.

Despite such complexity and the necessary reliance on observational and administrative data, the committee emphasizes the urgency of policy action in the face of a population health crisis that is claiming the lives of working-age adults in the prime of their lives, a crisis that has been further exacerbated by the COVID-19 pandemic. The trends and patterns documented in Chapters 2–4 of the report clearly show that life expectancy in the United States is lower than that in all other similar high-income

democratic countries; that working-age mortality rates increased during the period examined for this study (1990–2017) in the United States but not in those other countries, with pronounced increases for particular causes of death; and that disparities in working-age mortality are either stubbornly wide (e.g., non-Hispanic [NH] Blacks [Blacks] compared with non-Hispanic Whites [Whites]) or widening (e.g., individuals with low versus high levels of education). This report demonstrates further that working-age mortality rates are significantly higher, and rising more rapidly, in some geographic areas of the country than in others.

This report also identifies significant knowledge gaps and data limitations that have critical implications for further research designed to isolate the key drivers and modifiable factors that explain the rise of and disparities in working-age mortality. The need for additional research is pressing. From a historical perspective, the rise in U.S. working-age mortality and recent resulting declines in life expectancy are relatively new phenomena. As this report documents, because the rise in working-age mortality was specific to certain causes of death but with varying patterns by age, sex, race and ethnicity, socioeconomic status, and geography, existing research into these complex and multilayered patterns is sparse, and emerging research attempting to better understand the explanations for these changing patterns is nascent. Therefore, much remains to be learned, and the committee offers a number of research recommendations focused on generating better evidence that can serve as the basis for policy evaluation and refinement.

This chapter first presents a framework that establishes three general content areas in which the committee's policy and research implications are most relevant: (1) medical science and health care access and delivery, (2) public health, and (3) social and economic policy. Within each category, the chapter provides a brief overview of policies that have resulted in success, failure, or some mix of success and failure in the past. With this experience in mind, the chapter then brings together the policy and research implications of earlier chapters to emphasize how those implications fit within these three content areas. Importantly, this chapter also presents new policy and research implications within the three content areas that cut across all of the previous chapters. As part of this discussion, the chapter also steps back to reflect on the key thematic findings regarding economic change, socioeconomic inequality, and vulnerability that are presented across the various chapters of the report. The chapter concludes with a brief discussion of lessons learned from the COVID-19 pandemic.

For ease of reference, Table 11-1 at the end of the chapter lists all of the recommendations and policy conclusions presented in the report, grouped thematically in the categories of opioids, other drugs, and alcohol; suicide; cardiometabolic diseases; cross-cutting themes; and data needs.

A FRAMEWORK FOR THE CATEGORIZATION OF POLICY AND RESEARCH IMPLICATIONS

This chapter draws on historical insights and this report's findings to suggest ways in which changes in policy and further research are likely to impact future trends and disparities in U.S. working-age mortality. As noted above, this discussion is organized into three broad categories of potential policy intervention and areas for more research:

- **Medical science and health care access and delivery.** This category includes advances in medical science, such as new pharmaceuticals and surgeries to treat patients with disease. This category also includes policies that provide working-age individuals with greater access to health care (e.g., Medicaid) and that lead to advances in medical practice, such as those that have resulted in more and more effective cancer screening. This category tends to have its greatest influences on individual-level health outcomes that are highly sensitive to interventions by the health care system. In language consistent with the conceptual framework described in Chapter 6, this set of policies focuses most centrally on *downstream* targets.
- **Public health (broadly defined).** This broad category includes a range of programs and policies aimed at improving population health by promoting and supporting healthy behaviors, by eliminating environmental hazards, and by promoting access to preventive interventions. Public health strategies to promote healthy behaviors include communication and education campaigns, strategies that create healthier environments (e.g., building walkable neighborhoods, subsidizing access to healthy foods, limiting portion sizes, restricting advertising of unhealthy products), and laws and regulations (e.g., limits on the density of tobacco outlets, taxation of unhealthy products, restrictions on sales to minors, required use of seatbelts, limits on access to guns). Strategies to eliminate environmental hazards include creating and enforcing air pollution and water quality standards, eliminating lead from housing and the air, and cleaning up hazardous sites. Preventive interventions include facilitating access to screening programs and tobacco cessation programs and implementing harm reduction strategies, such as needle exchange programs. A key feature of public health strategies is that they impact the population as a whole and are focused more on preventing than on treating adverse health outcomes.
- **Social and economic policy.** This broad category includes policies not specifically directed at health, such as minimum wage laws,

family leave policies, civil rights legislation, zoning regulations, and tax law. The health-in-all-policies approach recognizes that a broad range of policies outside of the health care and public health sectors—including those focused on civil rights, education, labor markets, housing, income, and transportation—have important health implications. Social and economic policies tend to operate on broad scales (e.g., national, state) and to have their greatest influence on *upstream* influences on health, including the distal drivers of working-age mortality and disparities therein (House, 2015; Schoeni et al., 2008). Thus, this category of policies best fits with the committee's conceptualization of macro-level influences on mortality trends and disparities as developed in Chapter 6. Policies targeting upstream social and economic factors may also be especially important for mortality disparities because they tend to focus on vulnerabilities of population subgroups that are due to social and economic inequalities. Research on the macro-level effects on health and mortality of economic and social structure and change is relatively new. Therefore, recommendations for further research in this area are provocative and are intended to spur research that can provide stronger empirical support for social and economic policies to promote the public good of health and well-being.

Finally, it should be noted that these categories of policy influence are often synergistic and mutually reinforcing. For example, public health campaigns focused on efforts to increase rates of adherence to treatment recommendations for chronic disease may work hand-in-hand with health care policy (e.g., broader access to Medicaid) and social and economic policy (e.g., policies that increase employment) to influence reductions in working-age mortality. Similarly, efforts by health care providers to address obesity require changes in the built environment to provide people with access to sidewalks and stores that sell healthy foods, but the ability of people to live in such neighborhoods or to afford healthy foods depends on economic, zoning, and housing policies. Thus the committee uses the above three-category policy and research framework to organize the discussion that follows while recognizing that the assignment of the committee's recommendations to any single category is imprecise and that some of the recommendations are salient to more than one category.

MEDICAL SCIENCE AND HEALTH CARE ACCESS AND DELIVERY

Background

Advances in medical science have contributed to rising U.S. life expectancy through reductions in mortality rates from key causes of death, especially since 1960 (Cutler, 2004; Cutler, Deaton, and Lleras-Muney, 2006). As discussed in Chapter 9, innovations in drug development and prevention, treatment, and control of chronic diseases explain about one-half of the remarkable decline in cardiovascular mortality seen from 1970 to 2010 (Mensah et al., 2017). At the same time, it should be noted that pharmaceutical innovations and medical treatments can have serious deleterious mortality consequences, best illustrated by the role of the pharmaceutical industry in the opioid epidemic, discussed in Chapter 7. And errors in the delivery of care compromise patient safety and can result in unintended adverse consequences, including deaths (Institute of Medicine [IOM], 2000, 2007a; Sunshine et al., 2019).

Among the greatest successes in recent decades was the introduction of protease inhibitors and highly active anti-retroviral treatment for treatment of HIV infection, which led to pronounced reductions in working-age mortality due to HIV/AIDS starting in the mid-1990s, as documented in Chapter 3. Other medical innovations have greatly improved health and reduced U.S. working-age mortality in recent decades. Examples include advances in cancer screening driven by technological breakthroughs; advances in chemotherapy, radiation therapy, and other oncologic treatment modalities (Armstrong et al., 2016; Smith et al., 2010); improved emergency response systems to stabilize motor vehicle crash victims and quickly transport them to trauma centers, as well as advances in trauma care itself (Nathens et al., 2000); technological advances in the treatment of coronary artery disease and stroke; and new care delivery models to speed transport and emergency treatment of patients with acute coronary events or signs of stroke. Looking to the future, naloxone, an easy-to-administer opioid antagonist, can be used to counter the effects of an opioid overdose and thereby save lives.

Advances in preventive medicine have also occurred. For example, nicotine gum and smoking cessation medications were introduced to help treat nicotine addiction, while the development of vaccines has prevented deaths due to infectious diseases. In many cases, the impact of medical breakthroughs has been amplified by public health initiatives and changes in medical protocols, such as clinical guidelines, checklists, and electronic prompts adopted by health care systems and providers to systematize screening (e.g., for cancer, hypertension, hyperlipidemia, HIV infection);

immunizations; and monitoring of factors implicated in chronic diseases, such as blood pressure, cholesterol levels, and blood sugar.

Medical and pharmaceutical innovations are heavily influenced by public policies. For example, new drug development often derives in part from basic research sponsored by the National Institutes of Health. Drug marketing exclusivity, although temporary, allows pharmaceutical firms to control prices and possibly reap large profits. Yet while many of the drugs developed in this market environment have been beneficial, the high costs of drugs and devices have in some instances led insurers to restrict coverage and patients to forego medications, reducing access to care and dampening the beneficial effects of innovation. Adverse health impacts of drugs have also been observed. Vioxx, for example, a nonsteroidal anti-inflammatory drug developed to treat arthritis, was forced off the market after it led to the death of an estimated 55,000 patients from cardiovascular failure (Biddle, 2007). The opioid epidemic is perhaps the most serious example of the adverse consequences of improper promotion, testing, and approval of prescription drugs. It is essential to understand whether events such as these occurred because of inadequate policies or failure to adhere to existing policies or regulations (e.g., appropriate postmarketing surveillance or communications with health professionals) if such adverse drug impacts are to be avoided.

Health care financing policy has also influenced health outcomes across multiple age groups, including the working-age population, in both positive and negative ways. Health insurers—governmental and private—have been instrumental in promoting the uptake of evidence-based preventive services. Among its provisions, the Affordable Care Act of 2010 requires all health plans to provide first-dollar coverage of all services recommended by the U.S. Preventive Services Task Force. Private health plans have also encouraged mammography screening and other preventive services, and research has clearly shown that waiving out-of-pocket expenses increases the uptake of preventive services (Briss et al., 2000; Newhouse, 1993; Solanki and Schauffler, 1999).

A growing body of evidence suggests that the expansion of Medicaid under the Affordable Care Act has improved health and mortality outcomes for children and adults, including those of working age. Medicaid expansion has been associated with a roughly 9 percent reduction in all-cause mortality among working-age adults exposed to the policy change; this effect appears to be growing with time and is estimated to be saving the lives of thousands of working-age Americans each year (Miller et al., 2019). Another study found that expansion states have experienced a 6 percent reduction in opioid overdose deaths and an 11 percent reduction in heroin-related deaths (Kravitz-Wirtz et al., 2020). Individuals in states that

expanded Medicaid coverage also have experienced better health outcomes relative to those in states that deferred expansion (Antonisse et al., 2018).

In a study comparing Medicaid expansion states (Kentucky and Arkansas) with a nonexpansion state (Texas), expansion was associated with a $337 per capita reduction in annual out-of-pocket spending, significant increases in preventive health visits, and a 23 percent increase in the proportion of respondents who described their health as "excellent" (Sommers et al., 2017). Medicaid expansion has also been associated with improved blood pressure control, but not lower rates of in-hospital mortality among heart failure patients (Cole et al., 2017; Wadhera et al., 2018). Among patients with end-stage renal disease, Medicaid expansion has significantly increased the quality of predialysis care and lowered mortality by 8.5 percent (Swaminathan et al., 2018). Medicaid expansion also appears to be beneficial for surgical patients and has been shown to improve outcomes among patients with diverticulitis, aortic aneurysm, peripheral artery disease, cholecystitis, and appendicitis through earlier hospital admission and more optimal care (Loehrer et al., 2018).

Implications for Policy and Research

The above background overview highlights the important role that medical science and health care, as well as health care access and delivery policies, have played in working-age mortality trends and disparities in recent decades, with largely positive but also some seriously negative consequences. Based on its findings regarding the leading causes of death responsible for increasing working-age mortality, the committee developed policy and research recommendations relevant to providing access to care and treatment for persons at risk of dying from drug poisoning, alcohol-related causes, and suicide; to providing care and treatment to reduce obesity; to addressing other metabolic and cardiovascular conditions; and to instituting regulatory policy to avoid future catastrophes like the opioid epidemic.

As discussed above, there is growing evidence that Medicaid expansion under the Affordable Care Act has led to lower mortality among working-age adults living in expansion states. Accordingly, the committee recommends that those states that have not yet expanded access to Medicaid do so as soon as possible, and that research to analyze the long-term effects of Medicaid expansion on the health and mortality of the working-age population be supported. The committee realizes that Medicaid expansion, like many interventions, requires added public expenditures. Yet existing evidence based on common thresholds for cost-effectiveness shows that program to be cost-effective (see, e.g., Sommers, 2017), suggesting that the added spending is warranted.

RECOMMENDATION 11-1: Given recent findings regarding largely better health and lower mortality among working-age adults who live in states that have expanded Medicaid under the Affordable Care Act, the 12 states that have not yet expanded access to Medicaid should do so as soon as possible. The National Institutes of Health and private foundations should also support research to analyze the long-term effects of Medicaid expansion on the health and mortality of the working-age population.

The committee's findings also indicate substantial unmet needs with regard to mental health care and substance use treatment. These include provider shortages due to inadequate funding, fragmented delivery systems, the lack of parity in behavioral health plans, and scarce options for the uninsured (Carlo, Barnett, and Frank, 2020). The committee recommends that, as part of addressing the demand side of the U.S. substance use problem, policy makers increase access to and the affordability of quality substance use and mental health treatment (see Chapter 7, Recommendation 7-1), and notes that the expansion of Medicaid (see Recommendation 11-1) would help increase access to such treatment.

Both the National Institute on Drug Abuse (NIDA) and substance use treatment professionals advise that addiction be considered a chronic disease and treated accordingly (Dennis and Scott, 2007; Hser et al., 2015; McLellan et al., 2000; National Institute on Drug Abuse [NIDA], 2005). However, more research is needed on the effectiveness of behavioral health interventions in reducing mental illness and its consequences, on improved methods for delivering mental health and substance use treatment, on harm reduction, and on the extent to which inadequate access to these services has contributed to rising working-age mortality from substance use and suicide (see Chapter 7, Recommendation 7-2). Examples of specific research gaps that require attention include improving behavioral approaches to prevention of drug use relapse, addressing the role of nonsubstance-related conditions in addictive behaviors, and developing better interventions to counter the adverse effects of various social groups in promoting substance use.

Despite increasing medical knowledge on how to improve cardiovascular health (e.g., reduce smoking, improve diet, engage in more exercise) and the development of pharmaceuticals (e.g., statins, antihypertensives) to control chronic cardiovascular conditions, persistent socioeconomic and racial/ethnic disparities in care outcomes persist because many individuals face barriers to care that prevent them from fully benefiting from advances in medical knowledge or receiving recommended preventive services, diagnostic tests, and treatments. The committee therefore contends that efforts to improve implementation systems to overcome these barriers and improve the quality and timeliness of treatments for hypertension, diabetes, and

heart disease may help reduce such disparities and help reverse the stalling of improvements in cardiovascular mortality that began in the 2010s. Translational research and implementation science play an important role in addressing barriers to optimal care faced by individuals and modifying the procedures used by providers, health systems, and insurers to close the gap between recommended care and the care many patients receive. The committee therefore recommends more research to better understand the barriers to effective prevention, diagnosis, and treatment of chronic conditions, with special emphasis on the challenges faced by less-educated and lower-income populations (see Chapter 9, Recommendation 9-4).

PUBLIC HEALTH

Background

History has demonstrated that reliance on access to medical innovations and health care access and delivery aimed at individuals, while important, has a limited impact on reducing or narrowing disparities in mortality. Individual health behaviors, such as tobacco use, account for a large proportion of preventable deaths (Mokdad et al., 2004), but the factors that influence lifestyle behaviors are complex. Decades ago, when the Framingham Heart Study (Levy and Brink, 2005) and other observational studies established the major cardiovascular risk factors—e.g., smoking, poor diet, sedentary behavior, hypertension—the policy response was to urge the public (and counsel patients) to change their behaviors and adopt healthy habits. To identify smokers and counsel them to quit, physicians were advised to systematically complete the "5A's" (Ask, Advise, Assess, Assist, and Arrange) for every patient (Glynn and Manley, 1995). For most Americans, however, behavior change (and maintenance) proved difficult, especially in combating such powerful habits as smoking and overeating, which were heavily promoted by corporations and advertisers and reinforced by cultural norms. Far too often, those with the determination to change their lifestyle encountered barriers, such as limited access to affordable nutritious foods; outdoor places to exercise, walk, or cycle; counseling programs for smoking cessation or weight loss; and medical care for hypertension. Given the limitations of reliance on individual-level change, public health policy initiatives at the national, state, and local levels have been crucial to support behavior change.

The case of tobacco control policy, perhaps the greatest public health success of the 20th century, is instructive. Specifically, much of the reduction in working-age mortality from tobacco-related diseases (e.g., lung cancer, ischemic heart disease) that is documented in Chapter 3 reflects the nation's remarkable success in reducing smoking. Fully 42 percent of

American adults smoked cigarettes in 1965, 1 year after U.S. Surgeon General Luther Terry released the landmark report on the dangers of tobacco (Advisory Committee to the Surgeon General of the Public Health Service, 1964; U.S. Department of Health and Human Services [HHS], 2014). The reduction in smoking from 42 percent in 1965 to 13.7 percent in 2018 was achieved in part by a succession of public health policy interventions at the national, state, and local levels, along with other influences, such as social networks that changed the culture around smoking (Pampel, 2002) and early efforts by employers and businesses to discourage smoking. These interventions included not only educational campaigns (e.g., public service announcements, warning labels on tobacco products) but also widespread implementation of laws and regulations aimed at discouraging tobacco use. For example, the suite of policies that reduced smoking rates included cigarette taxes (Hoffman and Tan, 2015), bans on indoor smoking (Frazer et al., 2016), and restrictions on the sale and marketing of tobacco products (particularly to minors) (Harder, 1996).

Exposing the role of the tobacco industry in promoting its products despite knowledge of the addictive properties of nicotine was crucial to continued declines in smoking. Documents disclosed in the 1990s revealed that manufacturers were aware of the health risks posed by tobacco as early as the 1950s (Glantz and Balbach, 2000). The release of these materials fueled legal action by more than 40 states to sue tobacco companies for violating consumer-protection and antitrust laws. The Master Settlement Agreement was reached in 1998, requiring manufacturers to curtail marketing practices and provide perpetual payments to the states for the costs of tobacco-related illnesses (more than $200 billion over the first 25 years) (Myers, 2018). In 2006, in *United States vs. Phillip Morris*, a federal court held several tobacco companies liable for racketeering; in 2009, a law gave authority to the Food and Drug Administration (FDA) to regulate the tobacco industry.[1] This history would find parallels in recent discoveries about prior knowledge of the addictive properties of prescription opioids in the pharmaceutical industry and new lawsuits by states seeking remedies for losses due to the opioid epidemic.

Although not as successful as tobacco control, policies designed to reduce alcohol use, such as prohibiting alcohol sales before age 21, restricting the density of alcohol outlets, raising the minimum legal drinking age (in the 1970s and 1980s), and adopting "zero tolerance" policies, have been effective (Carpenter, 2004; Carpenter and Dobkin, 2009; Carpenter et al., 2007; O'Malley and Wagenaar, 1991; Sherk et al., 2018; Wagenaar and Toomey, 2002). Actions taken by government and the automobile

[1]Family Smoking Prevention and Tobacco Control Act of 2009, Pub. L. No. 111-31, 123 Stat. 1776. Available: https://www.congress.gov/111/plaws/publ31/PLAW-111publ31.pdf.

industry—including federal standards for automobile safety, the enactment of laws to enforce speed limits, mandated use of occupant restraints, the prosecution of individuals driving under the influence of alcohol or drugs, and the development of safer vehicles—were central to the reduction in deaths from transport accidents (Byrnes and Gerberich, 2012; Dinh-Zarr et al., 2001; Macpherson and Spinks, 2008; National Academies of Sciences, Engineering, and Medicine [NASEM], 2020b). Even more broadly, public health policies have been important in reducing mortality through a wide range of environmental measures. For example, water fluoridation (Iheozor-Ejiofor et al., 2015) and lead abatement have led to significant health improvements (Wilson et al., 2006). Likewise, the Clean Air Act and associated evidence-based regulation of criteria pollutants resulted in major reductions in contaminants, reducing morbidity and mortality from many diseases linked to air pollution (Samet, Burke, and Goldstein, 2017).

In other areas, however, public health policies and programs have been less effective. For example, 1920s-era alcohol prohibition and the Reagan-era "Just Say No" campaign were ineffective solutions for preventing substance use and addiction (Hornik et al., 2008). "Just Say No" demonized illicit substances and aligned drugs in general with a vaguely defined deviant group, and presented substance use as a collective moral failure of specific communities instead of treating them as a public health issue. The failure of this approach is also illustrated by the ineffectiveness of the Drug Abuse Resistance Education (D.A.R.E) program (Pan and Bai, 2009; Robert Wood Johnson Foundation, 2010), which brings police officers to schools to warn students about the dangers of drugs.

A number of public health policies and interventions have been proposed to create environments that are less obesogenic and are conducive to healthy eating and physical activity. These include conducting educational campaigns (in mass media and at work), implementing menu labeling and providing dietary guidelines, increasing access to healthy foods and limiting access to unhealthy foods (through subsidies and taxes), promoting physical activity through changes in built environments and workplace interventions (focused on walkability, public transport, and standing desks), and passing zoning laws to limit the density of alcohol outlets and fast food restaurants (Community Guide, 2020a; IOM, 2007b, 2012a; Johnston et al., 2014; Lee et al., 2019). These efforts have had success in specific settings but have not always been generalizable, sustainable, or easy to adopt on a national scale (Lee et al., 2019). Yet while many of these efforts have been local and not widespread, policy makers and the food industry share some of the blame for this lack of progress. The U.S. Department of Agriculture, which for decades has promulgated dietary guidelines to promote healthy eating and combat obesity, has also protected the meat and dairy industries (Nestle, 2013) and, since the 1980s, has issued farm subsidies that have generated

an oversupply of corn, thereby lowering its cost and encouraging the production of high-fructose corn syrup, the major sweetener added to the food supply. The widespread use of high-fructose corn syrup and the production of low-cost calorie-dense foods may help explain the marked increase in obesity rates that occurred after these products were introduced (Franck, Grandi, and Eisenberg, 2013).

Policies to address food insecurity and/or the nutrition of vulnerable populations have been effective, helping to reduce health and mortality risks. For example, access in childhood to the Supplemental Nutrition Assistance Program, also known as Food Stamps, reduced the incidence of metabolic syndrome and working-age mortality among persons ages 40–64 (Heflin, Ingram, and Ziliak, 2019; Hoynes, Miller, and Simon, 2015), and the Special Supplemental Nutrition Program for Women, Infants, and Children program achieved success in improving maternal and child health outcomes (Bitler and Currie, 2005; Chorniy, Currie, and Sonchak, 2020; Currie, 2001, 2009).

A general lack of adequate investment in public health and related infrastructure at the national, state, and local levels has been a major challenge in implementing successful public health interventions. A case in point is the nation's underinvestment in governmental public health agencies, a problem that has been recognized for decades (Bhutta, 2012; DeSalvo et al., 2017; IOM, 2002, 2012b; Mays and Hogg, 2015). This underinvestment has limited the ability of state and local health departments to fully sustain traditional public health services, as well as to create broad and innovative programs to better prevent and control acute and chronic illnesses. This underinvestment has also made the nation more vulnerable to public health crises, hampering the ability to respond adequately to natural disasters, disease outbreaks, or pandemics.

In sum, evidence indicates that the impact of public health initiatives on working-age mortality has been mostly positive, but that these initiatives have not been uniformly successful, and many potential policies (e.g., soda taxes, built environment interventions), although promising, have not been implemented on a broad or national scale. The example of tobacco control is a clear success: the combination of tax policy, smoking restrictions, and public health campaigns clearly altered behavior, improved health, and reduced mortality due to cigarette smoking among working-age adults. Other clear successes include laws targeting air and water pollution and vehicle safety, which in both cases compelled upstream action to improve downstream exposures to health risks. Purely informational campaigns have been less successful but may have laid the foundation of support for stronger policy action in specific cases, such as cancer screening, the prevention and management of HIV/AIDS, and detection and control of hypertension and high cholesterol.

Implications for Policy and Research

The above background overview suggests that there is an important role for public health policies in improving U.S. health and reducing working-age mortality in the future. Based on the findings presented in this report, the committee developed several policy recommendations focused on curbing the availability of addictive drugs and reducing rates of obesity and smoking. The committee also identified several gaps for which additional public health research could help pave the way for future policy recommendations that could help reduce working-age mortality.

Considering the vast loss of life that resulted from the approval, production, distribution, and promotion of opioids and other highly addictive drugs (see Chapter 7), the committee strongly recommends strengthening regulatory control and monitoring of the development and marketing of prescription narcotics. In addition, the committee recommends developing, funding, and enforcing tough internal standards within the pharmaceutical industry, with strong sanctions for violation of these standards. Moreover, the committee recommends that government policy makers at all levels invest in programs focused on substance use as a public health issue and pursue alternatives to arrest and incarceration. These programs should be aimed at reducing barriers to and encouraging entry into substance use disorder treatment (see Chapter 7, Recommendation 7-1).

The United States has already experienced some early successes with such drug-related policies, as discussed in Chapter 7. Guidelines limiting physician prescribing of opioids, monitoring to identify excessive levels of prescribing, and the implementation of "pill mill" laws requiring providers to provide clinical documentation from medical records to support the prescribing of these drugs have been effective in controlling the misuse of prescription opioids (Kiang et al., 2019). Alpert and colleagues (2019) found that states that monitored physicians' prescribing of opioids and other Schedule II drugs had fewer deaths due to prescription opioids (Alpert et al., 2019). However, there have also been some unintended consequences of the tightening of opioid prescribing, including the substitution of illicit opioids (heroin, fentanyl) for prescription opioids among individuals who had developed addiction or dependence (Hadland and Beletsky, 2018; Mallatt, 2019); rapid and unsafe titration of opioids among chronic pain patients (Kertesz, Gordon, and Satel, 2018); and substantial difficulty among chronic pain patients, including older adults with multiple chronic health conditions, in accessing the opioids needed to manage their pain (Ritchie et al., 2020). Given the preventable loss of life due to the opioid epidemic that unfolded over the past 25 years, the committee strongly endorses more caution with respect to bringing highly addictive and potentially lethal drugs to market. However, policies that reduce access

to prescription opioids without addressing demand are likely to have limited success in reducing drug overdose rates (Ciccarone, 2019; Cicero, Ellis, and Kasper, 2017; Dasgupta, Beletsky, and Ciccarone, 2018; Hadland and Beletsky, 2018).

This report also identifies key knowledge gaps for which more research on supply-side issues is needed. To further support the committee's policy recommendation for better regulatory control of narcotic prescription drugs, further public health research is needed on the mechanisms that underlie physicians' and patients' unintended responses to tighter regulation of drugs posing a high risk of misuse and addiction. Evidence shows, for example, that some individuals who were dependent on prescription opioids were pushed by their inability to obtain those drugs to markets for heroin and fentanyl. Research on strategies for preventing such unintended negative consequences should therefore be conducted in parallel with the development of policies for better regulatory control of narcotic prescription drugs (see Chapter 7, Recommendation 7-3).

The substance use crisis extends beyond drugs. The committee also found considerable evidence of increasing working-age mortality from alcohol-related diseases (see Chapter 4), the causes of which are not entirely clear. The committee therefore urges research to arrive at a better understanding of how changes over time in alcohol consumption, changes in the advertising and promotion of alcohol, and changes in the cultural acceptance of alcohol use have contributed to increases in alcohol-related mortality (see Chapter 7, Recommendation 7-3).

One demand-side argument attributes the trend in mortality due to drugs, alcohol, and suicide to increased vulnerability to substance use brought on by both long-term underlying and recently increasing stress, distress, "despair" (for which there are currently no clinically validated measures), pain, and mental illness (particularly depression). However, fundamental data are lacking on the epidemiology of mental health to confirm whether stress, distress, despair, pain, and depression have definitively increased, let alone to link those increases to trends in drug or alcohol use. A substantial literature documents that persons with mental illness are at increased risk of substance use disorders (NIDA, 2020; Unger et al., 1997; Volkow, 2001). Although mental illnesses and substance use disorders are therefore closely intertwined, it has been difficult for both researchers and policy makers to understand trends and disparities in stress, distress, despair, pain, and depression and how such trends may be related to substance use and substance use disorders. The committee therefore recommends further research on physical pain and the various psychosocial indicators that increase and/or decrease the risk of unhealthy behaviors related to substance use.

RECOMMENDATION 11-2: Federal agencies, in partnership with private foundations and other funding entities, should support research that tracks physical pain and the various psychosocial indicators, including stress, distress, despair, hopelessness, coping, resilience, and grit, that increase and/or decrease the risk of unhealthy behaviors related to substance use at the population level; explores relationships between these indicators and various causes of mortality and morbidity; and examines how trends in these indicators and their associations with mortality and morbidity vary by demographic group, socioeconomic status, and geography.

The rise in suicide mortality among Whites, especially White men, documented in this report occurred in the context of changing means of suicide. Despite evidence that more firearm-related suicides occur in states with looser gun regulations and greater gun ownership, the proportion of all suicide deaths related to firearms declined from 1990 to 2017, while the proportion due to hanging, suffocation, and strangulation increased. Thus, more needs to be known about means of suicide to better understand the increase in different modalities, how they differ by sex, and what factors might precipitate the choices made in this regard. Research on the role of gun control laws and gun availability in suicide mortality is particularly warranted, with attention paid to the causal effect of changes in gun control laws and gun availability on trends in suicide mortality (see Chapter 8, Recommendation 8-1). Given the different levels, trends, and disparities in mortality due to drug poisoning, alcohol, and suicide, the committee examined each of these causes of death separately rather than grouping them into a single category of "deaths of despair." Nonetheless, research is needed to test more effectively whether there are important drivers of mortality that may be common across these three causes. Specifically, the committee recommends public health research to explore how the various mechanisms that explain sociodemographic and geographic differences and temporal changes in mortality due to drug poisoning compare with those that explain sociodemographic and geographic differences and temporal changes in mortality due to alcohol and suicide (see Chapter 7, Recommendation 7-3).

Chapter 9 provides important explanations for the troubling stall in declines in mortality (and recent mortality increases among some demographic subgroups) due to cardiometabolic diseases among working-age adults. The key explanation for these trends is the increasing prevalence of adult obesity, coupled with long-term exposure to obesity that all too often begins in childhood, especially among recent cohorts (Abdullah et al., 2011; Owen et al., 2009; Stokes, Ni, and Preston, 2017). Chapter 9 also highlights disparities in obesity across population subgroups—including by race and ethnicity, gender, and socioeconomic status—that influence

cardiometabolic disparities in mortality (Hales et al., 2020; Lee, Harris, and Gordon-Larsen, 2009). Public health policy can play an important role in reducing obesity, with particular attention to curbing trajectories of potentially problematic weight gain in childhood and adolescence. Accordingly, the committee recommends that obesity prevention programs start early in life and be targeted to children and adolescents most at risk (e.g., racial/ethnic minority groups, females, and people living in poverty and neighborhoods of low socioeconomic status) and those who are overweight or gaining weight to intervene before obesity trajectories become set throughout the life course (see Chapter 9, Recommendation 9-3).

As discussed in Chapter 9, however, the causes of obesity are multifaceted and therefore difficult to address with single public policy initiatives, a point underscored by the limited headway made in reducing obesity over the past three decades (IOM, 2012a; Ravussin and Ryan, 2018). More research is needed to identify the multilevel and interactive causes of obesity to support the development of a multipronged public policy approach for addressing this major public health problem with as much success as was achieved through tobacco control policies and public health programs. Almost all obesity scholars point to the important role of obesogenic factors in the physical and food environments, including the interplay between individual health behaviors involving diet and physical exercise and societal-level changes in food production, transportation systems, green space, and sedentary work environments.

For example, there is evidence that technological changes in the way food is produced, distributed, and consumed have contributed to the increase in obesity, and that there is a role for public health policy in improving the production of healthy foods and reducing the distribution and consumption of unhealthy foods, especially among children and adolescents. The committee's findings also document the success of healthy diets in achieving weight loss, at least for the short term, and the fact that regular exercise is almost always beneficial in reducing overweight and obesity. However, efforts to promote lifestyle changes involving healthy diets and regular exercise (e.g., through worksite health promotion programs and free gym memberships) may not always be effective or sustainable. Thus, research is needed to explore the factors that erode short-term success in diet and exercise changes and, conversely, the factors that promote long-term lifestyle change to reduce obesity. A focus of this research should be on environmental drivers (e.g., occupational activity, exposure to chemicals, food deserts, green space and walkability, economic inequality, residential segregation, duration of exposure to electronic screens, advertising, the increase in "hyperpalatable" processed foods). Specifically, the committee recommends research evaluating the effectiveness of programs and policies that promote the consumption of healthy foods and adoption of healthy

lifestyles, as well as those that discourage the consumption, manufacturing, and advertising of highly processed and poor-quality foods and unhealthy lifestyles. The committee also recommends considering how systemic changes in food production, workplace systems, urban design, and transportation and other societal-level changes have fostered and sustained obesogenic environments and sedentary lifestyles to determine how those environments have deleterious consequences for population health (see Chapter 9, Recommendation 9-1).

As noted above, research on environmental impacts is complex, and the wide range of study designs, methods, and environmental variables involved makes it difficult to identify causal pathways. As a first step toward identifying some of the key drivers, the committee recommends research using experimental designs and taking advantage of existing neighborhood experimental projects (e.g., Moving to Opportunity) to examine the causal role of obesogenic factors in the environment and determine which are most responsible for the rise in obesity prevalence and body mass index levels (see Chapter 9, Recommendation 9-2).

The food industry—aided by legislation and budget decisions promoted by lobbyists and politicians from agricultural states—has also contributed to the obesity epidemic by successfully leveraging advertising and marketing techniques to boost consumption of calorie-dense foods (Charlebois, Tamilia, and Labrecque, 2007; Freudenberg, 2014; Nestle, 2013). As noted earlier, the farm subsidies authorized by Congress in the 1980s encouraged the oversupply of corn, thereby lowering its cost and encouraging the production of high-fructose corn syrup, a major sweetener that has become ubiquitous in American processed food. U.S. per capita consumption of high-fructose corn syrup increased from 0.8 g per day in 1970 to 91.6 g in 2000 (Bray, Nielsen, and Popkin, 2004). In addition, restaurants promoting inexpensive, unhealthy, and "all you can eat" menus have proliferated, especially in socioeconomically disadvantaged neighborhoods most at risk for obesity.

The need for solutions is widely recognized in the public and private sectors, driven not only by public health concerns but also by the threat obesity poses for employers, the business community, and the armed services. Further work is needed to build on recent efforts—some led by the food industry itself and others by public health authorities—to discourage the production and purchase of unhealthy foods or at least give consumers better information with which to make healthier food choices. Examples include self-regulation of or restrictions on misleading advertising (Graff, Kunkel, and Mermin, 2012), pricing and tax strategies (Blecher, 2015), "Nutrition Facts" product labels mandated by the FDA (U.S. Food and Drug Administration [FDA], 2020), menu labeling by restaurants (VanEpps et al., 2016), zoning decisions to reduce the proliferation of fast food restaurants, and other measures. Evaluating policies and initiatives that affect the diets

of young people is crucial, including replacing corn syrup with natural sweeteners, taxing sugary drinks and soda, restricting advertising targeting children, and removing vending machines from schools. That U.S. caloric intake per capita outpaces that of other high-income countries (Institute of Medicine and National Research Council [IOM and NRC], 2013) suggests the need to identify and address structural causes for that U.S. distinction, from more permissive regulation of the food industry and advertisers to cultural differences in lifestyle (see Chapter 9, Policy Conclusion 9-1).

Although tobacco control policy has been a great U.S. public health success, reductions in smoking behavior have been much greater among the highly educated than among the less educated, leaving large disparities in tobacco use that in turn contribute to disparate rates of mortality from cardiometabolic diseases and cancer. The committee recommends further public health research to address the barriers to smoking cessation and prevention of initiation faced by populations that continue to smoke at high rates, especially those with less education or income, and to evaluate programs that have been successful in promoting smoking cessation or preventing initiation (see Chapter 9, Recommendation 9-4).

In understanding and addressing public health problems and diseases, it is also important to take account of important comorbidities and co-occurring conditions in the absence of which a person may not have died (e.g., alcohol or drug involvement in motor vehicle or pedestrian accidents, chronic substance use and heart disease, injection drug use and bloodborne infectious disease). The COVID-19 pandemic has vividly illustrated the critical role of comorbidities: those who have obesity, autoimmune diseases, hypertension, or heart disease have died at much higher rates from COVID-19 infection relative to those without such conditions. Most approaches to coding and describing trends in mortality (including the approach used in this report) assign a single cause of death—the underlying cause, defined as "the disease or injury which initiated the train of morbid events leading directly to death, or the circumstances of the accident or violence which produced the fatal injury" (World Health Organization [WHO], 2011, p. 31). However, most deaths involve numerous contributing, sequential, and overlapping conditions. Assigning a single cause of death is therefore a simplistic and somewhat artificial way to describe and understand mortality trends and their explanations.

RECOMMENDATION 11-3: Researchers should more frequently use the International Classification of Diseases (ICD)-10 codes for multiple causes of death in their examination, analysis, and explanations of mortality trends and disparities in order to better identify the various factors that act together in causing death and how those factors and their combinations change over time.

SOCIAL AND ECONOMIC POLICIES

Background

Many medical and public health policies discussed above focus on improving health one patient or one disease at a time. In contrast, social and economic policies typically are not designed to reduce mortality for any or all causes of death but may affect survival nonetheless. Policies outside the sectors of medicine and public health that influence education, jobs, income, wealth, housing, racial/ethnic inequalities, immigration, and other determinants of health may also influence trends and disparities in working-age mortality. Macro-level social and economic policies—enacted by cities, states, and the federal government—have the potential to alter the distribution of key societal resources (e.g., income, education, housing, wealth). They also can reduce or exacerbate social inequities (e.g., by gender, race and ethnicity, immigrant status, sexual minority status, socioeconomic status, and ability status) and disparities in health and mortality (House, 2015; Hummer and Hamilton, 2019; Phelan, Link, and Tehranifar, 2010; Schoeni et al., 2008).

Researchers and policy makers face key challenges when considering social and economic policies (see Chapter 10 for discussion of empirical work on the relationship between economic factors and mortality). First, the effects of such policies on mortality are difficult to measure because the policy initiatives tend to be quite distant from the more proximate causes of mortality (e.g., stress, obesity, smoking) and operate mainly through indirect pathways (e.g., changes in social and economic conditions). Second, changes in social and economic conditions may not be due exclusively to policy changes. For example, the downsizing of the U.S. manufacturing sector that claimed so many jobs was fueled in part by factors largely outside the control of policy makers, such as the growth in automation and information technologies and other global market forces. The significant potential for confounding further makes it difficult to infer causality from associational studies of policy contexts and mortality trends and disparities. For example, trends in death rates due to drug poisoning differed between the United States and European countries over the past 25 years even though recessions and economic disruptions due to globalization that were experienced by the United States also affected European countries. This divergence may well be due to U.S.–European policy differences in such areas as social welfare programs and universal health care that prolonged the economic pain for families in the United States. However, the evidence is not rich enough to disentangle the extent to which the more favorable experience in Europe reflects differences in policies related to economic

security and unemployment, differences in medical culture related to use of pain medications, or some other factors unrelated to policy.

With the challenges of social and economic policy in mind, the discussion below first describes a few examples of policies related to broader social and economic well-being that have been demonstrated to influence mortality in the United States, and then summarizes policy and research implications of social and economic factors.

As a first example, studies have shown that some income-related policies influence population health and working-age mortality. Research has demonstrated that the Earned Income Tax Credit (EITC) increases employment and income and improves a variety of health outcomes (Hamad and Rehkopf, 2015; Hotz, 2003; Hoynes and Rothstein, 2016; Hoynes, Miller, and Simon, 2015; Komro et al., 2016; Strully, Rehkopf, and Xuan, 2010).[2] Evidence is also suggestive that minimum wage laws improve economic security and health outcomes[3] and may have impacts on working-age mortality (Andreyeva and Ukert, 2018; Dow et al., 2019; Kaufman et al., 2020; McCarrier et al., 2011; Narain and Zimmerman, 2019; Rosenquist et al., 2020; Tsao et al., 2016; Van Dyke et al., 2018). More broadly, policy efforts aimed at increasing educational attainment among the U.S. population have been shown to have effects on reducing mortality rates decades later (Halpern-Manners et al., 2020; Lleras-Muney, 2005). Other social and economic policies focused on unemployment benefits, the provision of food, preschool education and childcare, parental leave, housing, community development, economic growth and inflation, and taxation hold great potential for influencing health and reducing working-age mortality. The many policy options that fit into these domains, however, are rarely envisioned as health policies, and little empirical work has directly assessed their effects on trends and disparities in working-age mortality (House, 2015).

Many social and economic policies, especially those targeting populations of lower socioeconomic status, have been helpful in addressing racial/ethnic inequities. As noted earlier, as a result of the U.S. history of structural racism and related discriminatory practices that persist today, non-Whites—and particularly Blacks and American Indians—have faced greater barriers to education, good jobs, high incomes, and stable housing relative to Whites and have had fewer opportunities than Whites to transfer wealth to subsequent generations. Policies aimed at reducing such inequities, which played a historic role in advancing civil rights, have been

[2]These outcomes include children's cognitive abilities, birthweight, Apgar score, child development, fertility, cognitive development, mortality, natality, and postneonatal mortality.

[3]These outcomes include postneonatal mortality, heart disease death rate, suicide death rate, self-reported hypertension, self-reported fair or poor health, and self-reported mental and physical health.

associated with improved health and mortality outcomes among Blacks. For example, research has demonstrated an association between the Head Start program and other civil rights legislation of the 1960s and subsequent reductions in working-age mortality among Blacks (Almond and Chay, 2006; Johnson, 2019; Kaplan, Ranjit, and Burgard, 2008). Chetty and colleagues (2016) found that policies to improve housing opportunities in segregated communities, such as the Moving to Opportunity program, were associated with improved social and health outcomes for low-income and minority individuals. Unfortunately, however, Blacks have continued to experience disproportionately high mortality rates, and, as discussed in Chapter 3, progress in lowering working-age mortality and narrowing the Black–White mortality gap has now stalled. Indeed, during the 2010s, working-age mortality increased among all non-White populations, reversing decades of progress (Woolf et al., 2018).

Findings presented in this report provide ample evidence that the recent rise in U.S. working-age mortality was concentrated among socioeconomically disadvantaged populations, particularly those with a high school degree or less, and that U.S. working-age mortality remains substantially higher among Blacks and American Indians, in particular, compared with Whites. There is also reasonable evidence that some targeted social and economic policies, such as higher minimum wages and the EITC, can reduce mortality rates in these vulnerable populations (Dow et al., 2019; Kaufman et al., 2020). Evidence on investments in education-, housing-, and employment-related policies have also been shown to reduce racial/ethnic discrimination and racial/ethnic disparities in health and mortality (Williams, Lawrence, and Davis, 2019). Thus, while the associations between social and economic policy and working-age health/mortality remain ripe for research, the committee's findings also carry important policy implications.

Implications for Policy and Research

The committee's findings demonstrate that the largest increases in working-age mortality since 1990 occurred among Whites. At the same time, however, these findings reveal that 2010 was an inflection point when all-cause mortality rates also began to increase among other racial/ethnic groups of working-age adults. Thus, recent trends in working-age mortality are problematic for all racial/ethnic groups in the United States. The committee furthermore identified large and in some cases widening socioeconomic and geographic disparities in working-age mortality. The increases in mortality rates that occurred over the past 25 years among persons with a high school degree or less and in many nonmetropolitan portions of the country are urgent concerns that require greater focus by the research community. Many of these racial/ethnic, socioeconomic, and

geographic disparities in mortality are likely to be strongly influenced by macro-level (e.g., social, economic, cultural, policy) factors, both historical and contemporary.

While the explanations for high and rising working-age mortality discussed in this report are often specific to certain causes of death, they reveal some common underlying themes that affected particular population groups at different time periods or in different contexts.

The first of these themes relates to adverse economic trends (e.g., stagnant wages, industrial shifts, job losses) that affected certain geographic areas and population groups more than others. The loss of manufacturing, mining, and other jobs in the industrial Midwest and Appalachia in the 1970s–2000s led to long-term economic decline and associated social problems in these areas, particularly among White individuals without a 4-year college degree. Scholars have noted that Blacks in urban centers experienced similar economic and social transformations in earlier decades when manufacturing jobs left the cities; unemployment rates increased, particularly among males; and marriage rates plummeted (Cherlin, 2009, 2018; Torr, 2011; Wilson, 1987). The decline in economic conditions among Blacks that preceded the increase in drug-related mortality during the cocaine and heroin epidemics of the 1970s and 1980s is not thematically unlike the loss of jobs in suburban and rural areas among Whites that preceded the opioid epidemic in the 1990s and 2000s. The different governmental and policy responses to these epidemics are telling, however, revealing a deeper societal explanation not only for these discrepant policies but also for the persistent Black–White mortality gap: the drug crisis among Blacks of low socioeconomic status was treated primarily as a criminal justice problem, while the crisis among their White counterparts was largely recognized as a public health problem, although the criminalization of addiction remains strong today.

A second theme—which helps explain the pace and timing of rising 21st-century working-age mortality and long-standing racial/ethnic disparities in mortality that have persisted throughout U.S. history—is socioeconomic inequality. Inequality is defined as a state of unequal access to opportunity, resources, or means in a process leading to health and longevity (Braveman and Tarimo, 2002; Krieger, Williams, and Moss, 1997; McCartney, Collins, and Mackenzie, 2013). As outlined in the conceptual framework presented in Chapter 6, the magnitude and forms of socioeconomic inequalities experienced by social groups vary based on certain characteristics (e.g., race and ethnicity, age, sex, gender identity, sexual orientation) and operate at multiple levels (e.g., institutional, community, family, individual). Moreover, socioeconomic inequality is structured across groups according to entrenched social hierarchies (Massey, 2007). As a result, groups that have systematically been treated unfairly in society

because of racism or other forms of discrimination possess far fewer socioeconomic resources relative to advantaged groups (e.g., the White majority), creating large socioeconomic inequalities.

Socially patterned disparities in health and mortality reflect the downstream outcomes of these unequal upstream processes (Link and Phelan, 1995; Phelan, Link, and Tehranifar, 2010; Williams and Sternthal, 2010). Understanding the causes of inequality can enable policy makers to derive solutions by intervening on those causes to facilitate the goal of health equity, both within and among groups. As a consequence of the long history of structural racism in the United States (more on this below), Blacks and American Indians, in particular, have experienced long-standing and persistent inequalities in opportunities for educational attainment in high-quality schools, stable jobs with good incomes, wealth accumulation, and the kind of intergenerational mobility that would place them on socioeconomic parity with Whites. In recent decades, socioeconomic inequality has also deepened among Whites and within U.S. society as a whole.

The growing importance of education within U.S. society and of academic credentials in obtaining well-paying technical and professional jobs has left those without a college degree with fewer opportunities for stable employment, employment-related health benefits, and social mobility. After World War II, White males without a college degree often had opportunities for jobs in blue collar industries, with middle-class salaries, health benefits, and pension plans; to a lesser but still important extent, this was also true for Black males. However, the decades that followed the 1970s and 1980s and the shift from a manufacturing to a service economy brought growing inequality between less- and more-educated American workers, a trend that in some ways redefined the position of low-educated Whites in the U.S. social stratification system, with profound consequences for their health and longevity. For example, evidence presented in this report indicates that mortality from substance use increased more rapidly among less-educated relative to more highly educated White adults throughout 1990–2017 (Case and Deaton, 2015; Denney et al., 2009; Geronimus et al., 2019). Accompanying these trends is evidence of the social toll of rising inequality among less-educated Whites, who experienced greater social isolation, family breakdowns, and declining institutional support systems (Case and Deaton, 2020; Cherlin, 2014, 2018; Ruggles, 2015; Torr, 2011; Wilcox et al., 2012).

A third theme that emerges from the committee's explanations for the trends and disparities in working-age mortality is vulnerability, which mediates the degree to which adverse economic conditions and socioeconomic inequality make some groups more susceptible than others to morbidity and mortality risks. For example, as a result of educational, job, and housing discrimination, Blacks tend to work and live in segregated disadvantaged

neighborhoods, which increases their exposure to obesogenic, unsafe, and low-resource environments that lack mental and physical health care and increase their mortality risks. Today's drug overdose crisis emerged from a "perfect storm" in which the flooding of the market with highly addictive yet deadly products occurred as the population was growing more vulnerable to physical and emotional pain, generating a heightened demand (and market) for substances that could temporarily bring relief. This demand was initially met by increased availability of prescription opioids, prescribed predominantly to Whites. Restrictions on opioid prescribing, however, increased the demand for and supply of illegal drugs, which became more accessible to other vulnerable groups—working-age Blacks, Hispanics, and low-educated Whites who lacked access to prescription opioids. Indeed, rates of overdose involving heroin, fentanyl, and cocaine started to rise for all racial/ethnic groups after 2010.

Declining economic conditions, socioeconomic inequality, and vulnerability are themes that help in understanding how the different and changing social, economic, and geographic contexts of population subgroups may explain recent trends and disparities in working-age mortality. Macro-level shifts in economic conditions and inequalities that operate at all levels of society have made various subgroups vulnerable in different places and times. For example, much attention with respect to the widening educational disparities in mortality among Whites has focused on the shifting fortunes of less-educated Whites that have upended their social and economic position. Blacks, American Indians, and people of Hispanic and Asian descent have endured such vulnerabilities as being victims of enslavement, genocide, and discrimination for centuries. The "American Dream" that children would live a better life than their parents has often been out of reach for Blacks and American Indians, but in recent decades has also eroded for working-class Whites. Understanding the pathways by which racial/ethnic, socioeconomic, and geographic disparities in mortality occur and perpetuate is an area in need of additional research focusing on the upstream macro-level historical and contemporary drivers and the downstream processes through which they operate to create health inequities.

RECOMMENDATION 11-4: The National Institutes of Health and other government and private research funders invested in understanding the structural and policy determinants of health should support a robust research program aimed at identifying the macro-level historical and contemporary drivers (e.g., social, economic, cultural, policy) of health and mortality inequities and the mediators (e.g., environmental, socioeconomic, health care, biological, psychological, behavioral) through which these drivers operate to create and sustain persistent racial/ethnic, socioeconomic (income and education), and geographic

(including rural–urban, regional, and across- and within-state) disparities in U.S. working-age mortality. Particular emphasis should be on understanding policy solutions that may be effective in reducing and eliminating inequities in health and well-being.

While, as noted above, Whites have experienced increasing socioeconomic inequality in recent decades, very wide socioeconomic inequalities remain among racial/ethnic groups that favor Whites and have always disadvantaged minority groups. Blacks and American Indians/Alaska Natives (AI/ANs), in particular, have experienced institutional- and individual-level injustices for centuries, brought about by the long history of structural racism in U.S. society that continues to the present day (Alexander, 2010; Du Bois, 1899; Massey and Denton, 1993). The legacy and persistence of structural racism are reflected in the findings presented in this report, which show that working-age mortality rates were much higher among Blacks than among Whites throughout the 1990–2017 period. Although the quality of mortality data for AI/ANs and Asian Americans is problematic, summary findings suggest that AI/ANs have the highest mortality rates of all working-age adults, while Asian Americans have the lowest (see Annex Figure 4-1 and the table in Box 4-2 in Chapter 4). While the Black–White disparity in all-cause mortality narrowed between 1990 and 2017 as mortality rates generally decreased among working-age Blacks and stagnated or increased among working-age Whites, Black all-cause mortality rates across the working ages remained 33–54 percent higher than the rates for Whites in 2017. The Black–White disparity in mortality is so great that 400,000 additional Whites would need to have died each year for the rates for the two groups to be equivalent (Wrigley-Field, 2020). Reflecting this disparity, life expectancy at birth for Blacks was 3.9 years shorter than that for Whites in 2018 (Murphy et al., 2021).

Racial/ethnic disparities in working-age mortality differ across causes of death. While drug mortality rates increased for White males and females of all working ages during 1990–2017, with especially pronounced increases after 2010, Black males ages 55–64 had higher drug mortality rates than Whites throughout the study period. The steepest increases in suicide mortality occurred among Whites of all working ages, especially White males (although Black and Hispanic suicide rates began to increase after 2010). Among cardiometabolic causes of death, Whites of all working ages also experienced increasing mortality due to endocrine, nutritional, and metabolic (ENM) diseases and hypertensive heart disease from 1990 to 2017, while Black mortality from these diseases fluctuated and remained relatively flat over this period, the exception being younger Black adults (ages 25–44), for whom the rates increased after 2010. Even with these fluctuations, however, the Black mortality rates for ENM diseases and hypertensive heart

disease were two to five times higher than the White rates across all working ages and time periods. Blacks relative to Whites experienced a faster pace of decline in mortality from ischemic heart disease and other circulatory diseases from 1990 to 2010, but when mortality improvement stalled for all racial/ethnic groups, Black mortality rates remained almost twice as high as those of Whites in 2017. Because cardiometabolic mortality includes the leading causes of U.S. deaths today (e.g., heart disease, diabetes, stroke), it contributes the largest number and largest proportion of all deaths to U.S. mortality trends. Thus, the persistent Black–White disparity in cardiometabolic mortality is an important driver of the long-standing racial/ethnic gap in all-cause mortality that has disadvantaged Blacks throughout U.S. history. While data limitations prevented an in-depth examination of cause-specific trends in AI/AN working-age mortality, previous research suggests similarly heightened mortality due to cardiometabolic diseases for this group as well.

Structural racism remains a central explanation for the persistent disparity in mortality rates between Blacks and Whites. Structural racism can be defined as the systematic restriction of societal resources (e.g., high-quality schooling, stable work and fair pay, safe housing and neighborhoods, wealth, prestige, respect, freedom) through processes of exploitation, exclusion, normalization, and legitimization that routinely advantage Whites while producing cumulative and chronic adverse outcomes for people of color (Gee and Ford, 2011; Hummer and Hamilton, 2019; Phelan and Link, 2015). As an upstream macro-level factor, the impact of structural racism on health and mortality is challenging to study because of its systemic nature, which manifests in multiple societal institutions, policies, and environments, making it difficult to measure and statistically quantify. As noted earlier, moreover, structural racism operates on health and mortality through a multitude of downstream community and individual-level mechanisms that interact in and across time and space (Gee and Ford, 2011; Hummer, 1996; Phelan and Link, 2015; Williams and Collins, 2001; Williams and Mohammed, 2013) (see Figure 6-1 in Chapter 6).

Structural racism produces racial/ethnic inequalities through two primary mechanisms. The first and largest is limiting access to resources for achieving socioeconomic status, measured by educational attainment, occupational prestige, income, home ownership, and wealth (Gee and Ford, 2011; Hummer and Chinn, 2011; Phelan and Link, 2015). An illustrative pathway through which structural racism affects cardiometabolic mortality is residential segregation (Williams and Collins, 2001; Williams and Mohammed, 2013). Substantial evidence documents that patterns of discrimination in education, employment, income, access to credit, and the real estate industry relegate Blacks to residential environments that provide less access to healthy foods, space for exercise and leisure activities, clean

air and water, and safe and efficient transportation systems—all of which are important risk factors for obesity. Thus as discussed earlier, the legacy and persistence of structural racism contribute to the higher prevalence of obesity among Blacks and their higher rates of mortality from ENM diseases and hypertensive heart disease compared with Whites, whose death rates from these causes have been rising slowly but are still much lower than those of Blacks.

The second mechanism by which racism affects health is biological processes that damage multiple body systems through exposure to overt discrimination, violence, and the daily microaggressions experienced by people of color. The chronic stress of and ongoing vigilance for such experiences harm the neuroendocrine and immune systems and promote unhealthy coping behaviors (e.g., smoking, substance use) (Geronimus et al., 2010; Hicken, Lee, and Hing, 2018; Jackson, Knight, and Rafferty, 2010; McEwen and Lasley, 2002; Williams, Lawrence, and Davis, 2019). These harms can be experienced by people of color at all levels of education or social standing, which helps explain why racial/ethnic disparities in working-age mortality persist even after adjustment for socioeconomic status (Geruso, 2012; Hummer and Chinn, 2011; Williams, 1999; Williams, Priest, and Anderson, 2016).

The structural racism that produces these adverse health effects has a long history in the United States and is considered systemic because that history has left a deep imprint on so many of the policies and practices of the nation's institutions. Policies enacted decades ago (e.g., redlining, the practice originating in the 1930s of restricting home loans in Black communities) continue to have persistent effects, such as limiting access to resources for social and economic mobility among people of color (Rothstein, 2017). Such policies are not just a vestige of the past; the disadvantages produced by historical policies are compounded by modern policies that continue to be adopted by institutions at the national, state, local, and neighborhood levels in ways that systematically disadvantage people of color.

The committee recognizes that structural racism is too complex to be amenable to easy fixes; isolated interventions are therefore unlikely to achieve meaningful impact. As in other countries, such as South Africa decades ago, success in dismantling structural racism in the United States will invariably require a suite of solutions that confront the problem at multiple levels of society and across sectors. Racial/ethnic disparities in health outcomes will likely persist until this occurs. Given the magnitude of the racial/ethnic mortality disparities documented in this and other reports and the multidimensional institutional- and individual-level factors that help explain them, the committee realizes that, as with structural racism itself, one or even a small number of specific policy levers will be insufficient to eliminate racial/ethnic disparities in working-age mortality. Thus,

the committee's policy conclusion in this area is intentionally broad and is directed at macro-level (local, state, federal) factors, reflecting the long-term, institutionalized, and multifaceted racism and associated inequalities that perpetuate racial/ethnic disparities in working-age mortality.

POLICY CONCLUSION 11-1: To reduce and ultimately eliminate racial/ethnic and other socioeconomic inequalities that continue to drive racial/ethnic disparities in U.S. working-age mortality, policy makers and decision makers at all levels of society will need to dismantle structural racism and discriminatory policies of exclusion (in such areas as education, employment and pay, housing, lending, civic participation, criminal justice, and health care) and be intentional in ensuring that new social and economic policies serve to eliminate, and not perpetuate, the social and economic inequalities to which racial/ethnic minority groups have long been exposed.

Efforts to revitalize the communities that have been hit hardest by the substance use and overdose crisis will similarly need to address the larger economic and social strains and dislocations that made these communities vulnerable to opioids and other drugs over the past four decades. Doing so may require a holistic approach to community development that involves federal, state, and local governments as well as a range of private-sector actors (see Chapter 7, Policy Conclusion 7-1).

The explanatory chapters of this report on drug- and alcohol-related, suicide, and cardiometabolic mortality all, in some ways, implicate stress as a possible important mechanism contributing to increased mortality from these causes of death. Decades of research have identified chronic stress as a key determinant of health and racial disparities therein for such cardiometabolic conditions as hypertension (Williams, 1999), elevated body mass index and obesity (Hicken, Lee, and Hing, 2018), diabetes (Jackson, Knight and Rafferty, 2010), and heart disease (Williams and Jackson, 2005; Williams, Lawrence, and Davis, 2019). Chronic stresses may also play an important role in helping to explain trends in working-age mortality due to suicide and drug and alcohol use. Social, economic, technological, and cultural shifts over the past 50 years have profoundly changed the family, work, and community environments underlying daily life in the United States, potentially leading to increased stress. Chronic daily stress may be felt more intensely among certain subgroups of the population, especially those with fewer available resources for dealing with such stress in healthy ways. For example, Chapter 3 presents evidence that the increase in working-age mortality during the study period (1990–2017) was more pronounced and geographically widespread among U.S. women relative to men. Women in the United States have shorter lifespans and poorer health

outcomes compared with women in other high-income countries (IOM and NRC, 2013), a disadvantage that some have attributed to the unique stresses that U.S. life imposes on women (Montez et al., 2015). A research program focused on the stressful lives of Americans—one that includes uncovering the sources and the consequences of that stress—would transcend individual causes of death and could provide key insights into both high overall working-age mortality and disparities therein.

RECOMMENDATION 11-5: Given the potential connection of daily stressors to substance use, suicide, and cardiometabolic disease and mortality, federal agencies, in partnership with private foundations and other funding entities, should support research that documents the sources of increasing stress in the lives of Americans (e.g., student debt, foreclosures, job instability, economic insecurity, family instability) and identifies those groups most affected by increasing stress (e.g., the poor, immigrants, young adults, racial/ethnic minorities, women, those living in rural areas, the long-term unemployed, those without a 4-year college degree).

More generally, much more research is needed on the population health and mortality crisis that has been affecting working-age Americans for at least the past 25 years. Widespread recognition of the increase in working-age mortality occurred as recently as late 2015 (Case and Deaton, 2015). While much has been learned since then, as documented in this report, the problem is of such magnitude that two additional streams of research—socioecological and cross-national—are urgently needed to focus squarely on this crisis.

RECOMMENDATION 11-6: Federal agencies, in partnership with private foundations and other funding entities, should support quantitative and qualitative interdisciplinary research on how factors defined at multiple levels (e.g., nation, state, community, family, individual) relate to working-age mortality, especially to deaths involving drug and alcohol use, suicide, and cardiometabolic disease.

- **This research should examine how mortality due to drugs and alcohol, suicide, and cardiometabolic disease varies by individual-level demographic characteristics (including sex, race and ethnicity, and socioeconomic status), economic and social factors (e.g., social integration, unemployment, income inequality, public policy), and various levels of geographic characteristics that may change over time (e.g., geographic characteristics of counties, state and local jurisdictions, labor markets, and neighborhood environments).**

- **The research should be designed to uncover protective and predisposing factors unique to specific population subgroups that can inform policies designed to reduce disparities in working-age mortality.**
- **The research should explore how mortality is affected by long-term changes in the economy (e.g., changes in employment, employment opportunities, and job characteristics), especially in certain geographic areas; by interaction between economic factors and such social factors as family structure, community support, and religiosity; by the duration of economic hardship; and by programs designed to alleviate economic deprivation and other social stressors.**
- **The research should consider study designs, measurement strategies, and analytic methods that can strengthen causal inferences and conclusions. Examples include well-designed longitudinal cohort studies with individual-level data linked to time-varying environmental data measured at multiple levels (e.g., states, neighborhoods, families), and approaches that capitalize on natural or quasi-experiments that can be leveraged to identify etiologic (causal) factors and policy impacts.**

Finally, some broad work in the area of U.S. health and mortality contrasts social and economic policy regimes in the United States with those in European and other high-income countries around the world. Much of this work suggests that differences in population health and mortality between the United States and its high-income peers may be due to weaker social and economic policy supports for individuals in the United States (Avendano and Kawachi, 2014; Bambra and Beckfield, 2012; Beckfield and Bambra, 2016; House, 2015; Hummer and Hamilton, 2019; IOM and NRC, 2013). To date, however, this promising area of inquiry has not been explored as rigorously as should be the case. In addition, this area of research should include state- and substate-level examination of the relationship of social policy to health and mortality in the United States, given the suggestion of promising recent work that wide health and mortality disparities in the United States may be driven by differences in social and health policies across state and local areas (Montez et al., 2020).

RECOMMENDATION 11-7: The National Institutes of Health and other public and private research entities should support a program of cross-national research aimed at understanding why trends and disparities in working-age mortality have unfolded differently in the United States and in other high-income countries. This program of research should

- **examine long-terms trends and disparities, beginning in the 1950s;**
- **include not only transdisciplinary studies of etiology (causation pathways) but also policy research to evaluate the effectiveness of policy approaches in other countries and their potential adaptability to the United States; and**
- **include a complementary domestic research portfolio focused on understanding long-term changes within the United States at the state and substate levels, beginning in the 1980s when these gaps began to widen.**

LESSONS FROM THE COVID-19 PANDEMIC

The COVID-19 pandemic had a devastating impact on mortality in 2020. As this report goes into print, researchers are only beginning to uncover the full impact of this virus. For many people, this pandemic marks a clear break with the past; it has changed people's daily lives in ways previously unimaginable. In many ways, however, COVID-19 has simply reinforced and exacerbated the impact of existing social and economic inequalities within the United States. It has underscored and reinforced the importance of key themes articulated throughout this report by illustrating the ways in which economic conditions and socioeconomic inequalities make certain population groups and geographic areas more vulnerable to COVID-19.

First, this report documents increased working-age mortality from drug poisoning (Chapter 7) and cardiometabolic diseases (Chapter 9), such as hypertension, diabetes, and obesity, that during the pandemic received attention as risk factors for COVID-19 morbidity and mortality and defined vulnerable groups in need of prioritized care (Ssentongo et al., 2020; Wang et al., 2021). The increased prevalence of cardiometabolic diseases in the U.S. working-age population highlighted in this report may help explain the unexpectedly high COVID-19 death toll seen among young and middle-age adults. Moreover, there is some preliminary evidence that the stressors of the pandemic led to an increase in substance use (both alcohol and drugs) as a coping mechanism (Czeisler et al., 2020; Pollard, Tucker, and Green, 2020; United Nations Office on Drugs and Crime [UNODC], 2020), potentially foreshadowing future increases in mortality from these causes.

Second, this report examines geographic and socioeconomic disparities in health among the U.S. population, the growth in social division and income inequality, and the potential association of these disparities with trends in working-age mortality. Similarly, the pandemic exposed the heightened vulnerability of certain geographic areas (e.g., hard-hit states, rural areas, low-income neighborhoods and communities) and the economically disadvantaged to COVID-19 (Chen and Krieger, 2020; Cheng, Sun,

and Monnat, 2020; Mueller et al., 2021). Low-income individuals were disproportionately represented among service and front-line workers with the greatest exposure to the virus and were less likely than more-advantaged groups to be able to work from home, adhere to social distancing guidelines, and sustain their families (e.g., to avoid food and housing insecurity) amid a devastated economy (Weiss and Paasche-Orlow, 2020). They were also more likely to have comorbidities (Cutler, Meara, and Richards-Shubik, 2011; Pampel, Krueger, and Denney, 2010) associated with more severe COVID-19 illness (CDC, 2020a).

Third, this report documents large disparities in mortality among people of color. These disparities are reflected in the disproportionately high rates of infection, hospitalization, and death from COVID-19 experienced by Blacks and Hispanics (Ford, Reber, and Reeves, 2020; Gold et al., 2020; National Center for Health Statistics [NCHS], 2021). Although significant racial/ethnic disparities were observed at older ages, the greatest disparities occurred among younger adults (Ford, Reber, and Reeves, 2020)—so great that they exceeded overall disparities in all-cause mortality among working-age adults (NCHS, 2021). Although these racial/ethnic disparities were undoubtedly due at least in part to the geographic concentration of the initial surge in infections in large, racially and ethnically diverse central metropolitan areas, such as New York City, San Francisco, Seattle, and Los Angeles, the virus subsequently spread to less-populated and less-diverse areas of the country. By January 2021, both case and mortality rates for COVID-19 were higher in nonmetropolitan than in metropolitan counties (Ullrich and Mueller, 2021). And even in nonmetropolitan areas, large racial/ethnic disparities persisted (Cheng, Sun, and Monnat, 2020; Ford, Reber, and Reeves, 2020).

Finally, this report points to the role of health care—both access to health insurance and providers and the barriers to care delivery faced by underserved patients—in shaping progress and setbacks in working-age mortality. The pandemic and the particular difficulties experienced by the U.S. health care system (and public health infrastructure) in comparison with peer countries (Bilinski and Emanuel, 2020) underscore the barriers the public (especially marginalized groups) faced in accessing care (from COVID-19 testing, to vaccination, to intensive care) and the limited capacity of the care delivery system to absorb surges in hospitalization and other health care demands.

Thus, the COVID-19 pandemic has drawn attention to long-standing social and economic inequalities that leave some populations vulnerable when new health threats emerge. It has also highlighted the important role that public policy can play in achieving health equity. The public witnessed vivid illustrations of not only how policy decisions affected the nation's epidemic curve but also how state trends were influenced by the decisions

of governors, legislators, and state courts, including states' preemption of the ability of cities to enact their own mandates to prevent the spread of the virus (Haddow et al., 2020; Treskon and Docter, 2020; Wagner, Rainwater, and Carter, 2020). All these issues received greater visibility during the COVID-19 pandemic and inspired calls for policy action to address them.

CONCLUSION

The United States is losing far too many lives far too early. The rise in working-age mortality documented in this report represents a crisis, one that threatens the future of the nation's families, workforce, economy, and national security, and one that requires action even if the evidence is imperfect or only suggestive of causal effects and solutions. This chapter has offered policy conclusions and recommendations toward addressing this crisis. At the same time, given the potential for unintended consequences of even the best-intended policy actions, it is also crucial to design policies carefully to account for potential risks, continue to monitor outcomes, generate better evidence, and adjust policies over time. The research implications of this report, also highlighted in this chapter, provide direction to this end.

TABLE 11-1 Recommendations and Policy Conclusions

	Opioids, Other Drugs, and Alcohol
POLICY CONCLUSION 7-1	Economic policies are needed to address the larger economic and social strains and dislocations that made communities that experienced economic decline over the past four decades vulnerable to opioids and other drugs. This effort may require a holistic approach to development that involves federal, state, and local governments as well as a range of private-sector actors.

continued

TABLE 11-1 Continued

	Opioids, Other Drugs, and Alcohol
RECOMMENDATION 7-1	Policy makers should implement policies that better address the U.S. addiction and overdose crisis and prevent future crises. In general, the most effective interventions target both risk and protective factors at multiple levels, including the individual, family, community, and society. • The Food and Drug Administration, the Drug Enforcement Administration, and other federal and state regulatory agencies should strengthen regulatory control and monitoring of the development, marketing, distribution, and dispensing of prescription drugs. • The pharmaceutical industry (including manufacturers, distributors, dispensers, and trade associations) should develop and fund stronger internal standards, regulatory structures, and procedures for surveillance and prevention of activities that could result in misuse, addiction, or other harms among users of its products. It should also develop stronger sanctions for violation of these standards. • Federal, state, and local governments should invest in programs that focus on substance use as a public health issue and pursue alternatives to arrest and incarceration. Such programs should be aimed at reducing barriers to and encouraging entry into substance use disorder treatment. • Medicaid and state and local government agencies (e.g., health departments, social services, public schools) should expand access to and improve the quality of substance use prevention, treatment, recovery, and harm reduction programs, as well as mental health counseling and treatment for people with substance use disorders. Substance use prevention programs should begin early, focus on life skills training and prosocial development rather than on fear, and be targeted to children and adolescents most at risk of early initiation of drug and alcohol use (e.g., those living in neighborhoods of low socioeconomic status, those who have suffered adverse childhood experiences).

RECOMMENDATION 7-2 Federal agencies, in partnership with private foundations and other funding entities, should support research on the effectiveness of behavioral health interventions in reducing mental illness and its consequences; on improved methods for delivering mental health and substance use treatment, harm reduction products and services (e.g., naloxone, medication-assisted therapies, needle exchange programs, safe injection sites), and recovery services; and on the extent to which inadequate access to these products and services has contributed to rising working-age mortality from substance use and suicide.

RECOMMENDATION 7-3 The National Institutes of Health, the Substance Abuse and Mental Health Services Administration, the Centers for Disease Control and Prevention, the Food and Drug Administration, and other relevant federal agencies should support research to address the gaps in knowledge regarding the underlying causes of the rise in drug poisoning, alcohol-related death, and suicide. Specifically, this research should be focused on

- the mechanisms underlying physicians' and patients' unintended responses to tighter regulation of drugs with a high risk of misuse and addiction, such as cases in which individuals dependent on prescription opioids were pushed to markets for heroin and fentanyl, and the identification of strategies for preventing those unintended consequences;
- whether changes over time in alcohol consumption (including types of alcoholic beverages, frequency of drinking, and volume of consumption), in advertising and promotion of alcohol, in cultural acceptance of alcohol use, and in concurrent use of drugs and alcohol have contributed to increases in alcohol-related mortality rates; and
- whether the various multilevel mechanisms that explain demographic and geographic differences and temporal changes in drug use are the same as or different from those that drive demographic and geographic differences and temporal changes in alcohol use and suicide.

continued

TABLE 11-1 Continued

	Suicide
RECOMMENDATION 8-1	Federal agencies, in partnership with private foundations and other funding entities, should support research on lethal means of suicide aimed at better understanding the increase in use of different suicide modalities, how modalities differ by sex, and what factors might precipitate the choices made. Research on the role of gun control laws and gun availability is particularly warranted, with attention paid to the causal effect of changes in gun control laws and gun availability on trends in suicide mortality.
	Cardiometabolic Diseases
RECOMMENDATION 9-1	Federal agencies, in partnership with private foundations and other funding entities, should support research that evaluates the effectiveness of programs and policies designed to improve U.S. cardiometabolic health and that considers the impact of changes at multiple levels of analysis: • At the individual level, research should continue to evaluate the effectiveness of programs and policies that promote consumption of healthy foods (e.g., mandatory labeling of food ingredients or components, fruit and vegetable subsidies) and the adoption of healthy lifestyles (e.g., subsidies for sports activities; urban development that prioritizes walking, biking, and transit). Likewise, research should continue to evaluate the effectiveness of programs and policies that discourage the consumption of poor-quality foods (e.g., sugar and soda taxes, nutritional standards and dietary guidelines from the U.S. Department of Agriculture and the U.S. Department of Health and Human Services) and unhealthy lifestyles (e.g., insurance rating based on poor health habits such as smoking, zoning laws for fast food restaurants and alcohol outlets). • At the societal level, research should consider systemic changes in food production, workplace systems, and transportation and other societal-level changes in the United States that foster and sustain obesogenic environments and sedentary lifestyles to determine the pathways through which such environments have deleterious consequences for population health.

RECOMMENDATION 9-2	Federal agencies, in partnership with private foundations and other funding entities, should support research that uses experimental designs and takes advantage of existing neighborhood experimental projects to examine the causal role of factors in the obesogenic environment and determine which have the greatest role in the rise in obesity prevalence and body mass index levels.
POLICY CONCLUSION 9-1	To reduce the per capita calorie consumption and body mass index levels of the U.S. population, policy makers will need to implement laws and regulations that preserve a healthy balance between the rights of the food industry, advertisers, grocers, and restaurants to enjoy free market competition and the public health imperative to limit the promotion and consumption of foods and beverages that contribute to obesity.
RECOMMENDATION 9-3	Designers of obesity prevention programs should focus on developing programs that start early in life and target children and adolescents most at risk of obesity (e.g., racial/ethnic minorities, females, people living in poverty and in neighborhoods of low socioeconomic status) and those who are overweight or gaining weight, thus intervening before obesity trajectories become set throughout adulthood.
RECOMMENDATION 9-4	To improve systems for delivering preventive care (e.g., smoking cessation) and existing treatments for hypertension, diabetes, and heart disease, federal agencies, in partnership with private foundations and other funding entities, should support research focused on better understanding the barriers to prevention and control of cardiometabolic disease faced by individuals—especially less-educated and lower-income populations—and evaluating potential solutions for removing those barriers.
	Cross-Cutting Themes
RECOMMENDATION 11-1	Given recent findings regarding largely better health and lower mortality among working-age adults who live in states that have expanded Medicaid under the Affordable Care Act, the 12 states that have not yet expanded access to Medicaid should do so as soon as possible. The National Institutes of Health and private foundations should also support research to analyze the long-term effects of Medicaid expansion on the health and mortality of the working-age population.

TABLE 11-1 Continued

	Cross-Cutting Themes
RECOMMENDATION 11-2	Federal agencies, in partnership with private foundations and other funding entities, should support research that tracks physical pain and the various psychosocial indicators, including stress, distress, despair, hopelessness, coping, resilience, and grit, that increase and/or decrease the risk of unhealthy behaviors related to substance use at the population level; explores relationships between these indicators and various causes of mortality and morbidity; and examines how trends in these indicators and their associations with mortality and morbidity vary by demographic group, socioeconomic status, and geography.
RECOMMENDATION 11-3	Researchers should more frequently use the International Classification of Diseases (ICD)-10 codes for multiple causes of death in their examination, analysis, and explanations of mortality trends and disparities in order to better identify the various factors that act together in causing death and how those factors and their combinations change over time.
RECOMMENDATION 11-4	The National Institutes of Health and other government and private research funders invested in understanding the structural and policy determinants of health should support a robust research program aimed at identifying the macro-level historical and contemporary drivers (e.g., social, economic, cultural, policy) of health and mortality inequities and the mediators (e.g., environmental, socioeconomic, health care, biological, psychological, behavioral) through which these drivers operate to create and sustain persistent racial/ethnic, socioeconomic (income and education), and geographic (including rural–urban, regional, and across- and within-state) disparities in U.S. working-age mortality. Particular emphasis should be on understanding policy solutions that may be effective in reducing and eliminating inequities in health and well-being.
RECOMMENDATION 11-5	Given the potential connection of daily stressors to substance use, suicide, and cardiometabolic disease and mortality, federal agencies, in partnership with private foundations and other funding entities, should support research that documents the sources of increasing stress in the lives of Americans (e.g., student debt, foreclosures, job instability, economic insecurity, family instability) and identifies those groups most affected by increasing stress (e.g., the poor, immigrants, young adults, racial/ethnic minorities, women, those living in rural areas, the long-term unemployed, those without a 4-year college degree).

POLICY CONCLUSION 11-1 To reduce and ultimately eliminate racial/ethnic and other socioeconomic inequalities that continue to drive racial/ethnic disparities in U.S. working-age mortality, policy makers and decision makers at all levels of society will need to dismantle structural racism and discriminatory policies of exclusion (in such areas as education, employment and pay, housing, lending, civic participation, criminal justice, and health care) and be intentional in ensuring that new social and economic policies serve to eliminate, and not perpetuate, the social and economic inequalities to which racial/ethnic minority groups have long been exposed.

RECOMMENDATION 11-6 Federal agencies, in partnership with private foundations and other funding entities, should support quantitative and qualitative interdisciplinary research on how factors defined at multiple levels (e.g., nation, state, community, family, individual) relate to working-age mortality, especially to deaths involving drug and alcohol use, suicide, and cardiometabolic disease.

- This research should examine how mortality due to drugs and alcohol, suicide, and cardiometabolic disease varies by individual-level demographic characteristics (including sex, race and ethnicity, and socioeconomic status), economic and social factors (e.g., social integration, unemployment, income inequality, public policy), and various levels of geographic characteristics that may change over time (e.g., geographic characteristics of counties, state and local jurisdictions, labor markets, and neighborhood environments).
- The research should be designed to uncover protective and predisposing factors unique to specific population subgroups that can inform policies designed to reduce disparities in working-age mortality.
- The research should explore how mortality is affected by long-term changes in the economy (e.g., changes in employment, employment opportunities, and job characteristics), especially in certain geographic areas; by interaction between economic factors and such social factors as family structure, community support, and religiosity; by the duration of economic hardship; and by programs designed to alleviate economic deprivation and other social stressors.
- The research should consider study designs, measurement strategies, and analytic methods that can strengthen causal inferences and conclusions. Examples include well-designed longitudinal cohort studies with individual-level data linked to time-varying environmental data measured at multiple levels (e.g., states, neighborhoods, families), and approaches that capitalize on natural or quasi-experiments that can be leveraged to identify etiologic (causal) factors and policy impacts.

TABLE 11-1 Continued

	Cross-Cutting Themes
RECOMMENDATION 11-7	The National Institutes of Health and other public and private research entities should support a program of cross-national research aimed at understanding why trends and disparities in working-age mortality have unfolded differently in the United States and in other high-income countries. This program of research should • examine long-terms trends and disparities, beginning in the 1950s; • include not only transdisciplinary studies of etiology (causation pathways) but also policy research to evaluate the effectiveness of policy approaches in other countries and their potential adaptability to the United States; and • include a complementary domestic research portfolio focused on understanding long-term changes within the United States at the state and substate levels, beginning in the 1980s when these gaps began to widen.
	Data Needs
RECOMMENDATION 5-1	The National Center for Health Statistics (NCHS), state vital statistics offices, and local-area health agencies should work together to develop a plan and set of activities for improving the accuracy of reporting on U.S. death certificates of educational attainment, American Indian and Alaska Native identity, and multiple causes of death. NCHS should also continue to conduct or facilitate studies on the accuracy of reporting on U.S. death certificates of educational attainment (particularly as such reports may vary across states and local areas) and American Indian and Alaska Native identity (particularly as such reports may vary across states, tribal affiliations, and local areas).
RECOMMENDATION 5-2	The National Center for Health Statistics and the National Institutes of Health should undertake and/or fund studies to evaluate state- and local-level variation in cause-of-death coding practices, explore how such variation may contribute to observed mortality trends, and make recommendations for reducing such variation.
RECOMMENDATION 5-3	The National Center for Health Statistics should include Asians in its regular reports on life expectancy estimates and trends in the United States and make an item on place of birth available to researchers in the public-use files, even if such information is at first categorical (e.g., foreign-born vs. U.S.-born) rather than granular.

RECOMMENDATION 5-4	To enable robust research on rural–urban trends in health and mortality, the National Institutes of Health and other research agencies and funders should support the oversampling of rural populations on national health and social surveys, including both existing (e.g., Health and Retirement Study, Behavioral Risk Factor Surveillance System, National Longitudinal Study of Adolescent to Adult Health [Add Health], National Survey on Drug Use and Health, National Health Interview Survey, National Health and Nutrition Examination Survey) and new surveys.
RECOMMENDATION 7-4	The Substance Abuse and Mental Health Services Administration should add to the publicly accessible version of the National Survey on Drug Use and Health U.S. Census region or U.S. Census division categories and the nine-category U.S. Department of Agriculture Economic Research Service rural–urban continuum codes or National Center for Health Statistics urban influence codes.
RECOMMENDATION 7-5	The National Institute of Mental Health and other relevant federal agencies should develop a research program to identify innovative and cost-effective methods for conducting periodic or ongoing population surveys of important mental health conditions. The research agenda should include measuring access to and uptake of behavioral health care services (e.g., mental health counseling, substance use disorder treatment) and the effects of such services on mental health outcomes and other important outcomes, such as those in the social, cognitive, and functional domains. These national surveys should be linked where possible to medical record and claims data, as well as to other important sources, such as education and social service information, while carefully protecting respondent confidentiality.
RECOMMENDATION 7-6	Questions about adverse childhood experiences should be added to the core of the Behavioral Risk Factor Surveillance System (so that the questions are asked in every state in every year), as well as to other relevant national health surveys, such as the National Health Interview Survey and the National Survey on Drug Use and Health. To advance understanding of the mechanisms and control of these experiences, this information should be improved by facilitating maximal record linkage of cohort findings to available social, military, medical, psychiatric, environmental, and law enforcement records.
RECOMMENDATION 8-2	Directors and funders of longitudinal studies should routinely link these survey data to the National Death Index to support a life-course approach to the study of mental health and suicide mortality.

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Appendix A

Mortality Data Analyses: Review Process and Detailed Mortality Rate Tables

REVIEW PROCESS FOR MORTALITY DATA CODING AND ANALYSIS

Checking International Classification of Diseases (ICD)-9 and ICD-10 Code Categorizations

Shannon Monnat compiled a list of ICD-9 and ICD-10 codes for the 20 specific cause-of-death categories included in this report. Irma Elo and Ryan Masters and two graduate research assistants checked this list for completeness and accuracy of categorization. The committee edited the list to ensure that it was exhaustive (included all ICD-9 and ICD-10 codes) and mutually exclusive (no code was included in more than one category). The specific ICD-9 and ICD-10 codes for each cause-of-death category are presented in Chapter 5.

Checking for Accuracy of Death Counts for Cause-of-Death Categories

The committee conducted analyses using the restricted mortality files (death certificates) from the National Center for Health Statistics. After assigning decedents to one of 20 cause-of-death categories based on their underlying cause of death, Shannon Monnat tabulated the total number of deaths in each of the 20 cause-of-death categories by year (1990–2017), sex, and 10-year age group (25–34, 35–44, 45–54, and 55–64). She then compared those tabulations from the restricted mortality data with the

total counts for each of the 20 cause-of-death categories in CDC WONDER (the public online mortality database) to ensure that the annual and subgroup-specific counts from the restricted mortality data equated to the counts derived from CDC WONDER. A graduate research assistant replicated this procedure. The committee member then repeated this procedure to further disaggregate counts by race (non-Hispanic [NH] White, NH Black, Hispanic, Asian, American Indian/Alaska Native) and compare them with the CDC WONDER data. This check was available only for 1999–2017 because Hispanic ethnicity was not included in CDC WONDER prior to 1999.

Checking for Accuracy of Age-Adjustment

Two committee members, Shannon Monnat and Irma Elo, independently calculated all of the subgroup-specific age-adjusted mortality rates for all-cause mortality and the 20 cause-of-death categories presented in this report. The two committee members independently arrived at identical age-adjusted mortality rates, which are those presented in the report.

CAUSE-SPECIFIC MORTALITY ESTIMATES BY SEX, AGE GROUP, AND RACE AND ETHNICITY

TABLE A-1 Cause-Specific Mortality Rates (Deaths/100,000): Non-Hispanic White Males Ages 25-44

	1990-93	1994-96	1997-99	2000-02	2003-05	2006-08	2009-11	2012-14	2015-17
All-Cause Mortality	212.29	216.23	184.10	188.82	190.08	190.73	186.89	190.47	222.62
Cause-Specific Mortality									
Infectious & Parasitic Diseases									
HIV/AIDS	35.83	36.31	9.06	7.01	5.51	3.74	2.08	1.53	1.05
Non-HIV/AIDS	3.62	3.98	3.31	3.14	2.94	2.60	2.45	2.35	2.57
Cancers									
Liver	0.49	0.61	0.62	0.57	0.56	0.51	0.51	0.47	0.52
Lung	4.34	4.11	4.00	3.82	3.47	2.79	2.26	1.90	1.61
All Other Cancers	20.90	20.38	19.30	18.28	17.39	16.51	16.56	15.82	15.55
Cardio and Metabolic Diseases									
Endocrine, Nutrition, & Metabolic Diseases	5.12	5.50	5.14	5.65	5.94	5.95	6.20	6.58	7.00
Hypertensive Heart Disease	1.08	1.39	1.60	1.97	2.58	2.90	3.24	3.41	3.86
Ischemic & Other Circulatory Diseases	30.76	30.93	29.67	28.76	28.48	26.98	25.74	24.48	24.52
Substance Use and Mental Health									
Drug Poisonings	9.55	13.00	15.08	19.09	24.86	31.71	34.39	39.48	62.43
Alcohol-Induced*	4.81	4.95	4.41	4.08	3.83	4.66	5.33	5.61	6.70
Suicide	23.09	24.37	23.82	23.65	24.32	25.57	27.62	28.92	32.30
Mental & Behavioral Disorders	3.84	4.24	4.48	4.76	5.15	4.01	3.42	3.85	5.10

continued

TABLE A-1 Continued

	1990-93	1994-96	1997-99	2000-02	2003-05	2006-08	2009-11	2012-14	2015-17
Other Body System Diseases & Disorders									
Nervous System	3.11	3.23	3.31	3.77	4.06	4.19	4.41	4.39	4.63
Genitourinary System	1.01	0.96	1.00	1.14	1.24	1.32	1.19	1.17	1.29
Respiratory System	4.49	4.40	4.17	4.16	4.01	3.95	4.35	4.37	4.23
Digestive System*	5.50	5.33	5.18	5.23	4.88	4.46	4.43	4.58	4.59
Other Causes of Death									
Homicides	8.12	7.12	5.97	5.39	5.18	5.57	4.78	4.88	5.66
Transport Injuries	27.21	25.55	24.96	26.11	26.47	26.21	21.28	20.90	22.22
Other External Causes	12.69	12.54	12.16	11.66	11.34	10.67	10.02	9.76	10.43
All Other Causes of Death	6.72	7.36	6.85	10.58	7.87	6.45	6.62	6.00	6.38

*Changes in the International Classification of Diseases (ICD)-10 code for diseases of the digestive system in 2006 affected the comparability of both digestive system deaths and alcohol-induced deaths before and after that year. More information is provided in Chapter 5.
NOTE: The table shows all-cause and cause-specific mortality rates (in deaths per 100,000 people in the population) among non-Hispanic White males ages 25–44. Mortality rates within each age group are age-adjusted by single-year of age to match the age distribution of the U.S. population in 2000 in order to improve comparability over time. These causes of death are exhaustive of all underlying cause-of-death codes and are based on the ICD-9 (1990–1998) and ICD-10 (1999–2017) codes. More information about the classification of causes of death can be found in Chapter 5.
SOURCE: Data from https://www.cdc.gov/nchs/nvss/nvss-restricted-data.htm.

TABLE A-2 Cause-Specific Mortality Rates (Deaths/100,000): Non-Hispanic Black Males Ages 25-44

	1990-93	1994-96	1997-99	2000-02	2003-05	2006-08	2009-11	2012-14	2015-17
All-Cause Mortality	582.82	575.02	407.48	373.59	352.47	329.79	286.19	281.67	319.86
Cause-Specific Mortality									
Infectious & Parasitic Diseases									
HIV/AIDS	134.82	174.99	68.52	56.25	43.76	31.89	19.68	12.58	10.16
Non-HIV/AIDS	16.23	14.90	10.56	7.61	6.90	6.16	5.33	4.63	4.78
Cancers									
Liver	1.84	2.11	1.72	1.63	1.52	1.42	1.16	1.25	1.18
Lung	10.43	9.16	7.55	6.87	4.97	3.47	2.34	2.37	2.36
All Other Cancers	31.45	29.61	26.74	23.02	21.33	20.41	19.17	17.46	17.42
Cardio and Metabolic Diseases									
Endocrine, Nutrition, & Metabolic Diseases	13.04	14.48	12.35	11.68	12.83	13.60	12.99	13.69	15.39
Hypertensive Heart Disease	9.98	10.25	10.45	10.77	11.98	12.88	13.16	13.52	14.97
Ischemic & Other Circulatory Diseases	76.27	70.33	63.20	57.62	56.41	53.75	49.32	49.66	49.51
Substance Use and Mental Health									
Drug Poisonings	19.45	22.19	19.75	17.65	17.02	18.18	13.71	16.20	32.54
Alcohol-Induced*	13.17	8.40	5.57	4.34	3.04	2.78	2.87	2.83	3.26
Suicide	17.11	17.04	14.78	13.94	13.70	13.72	13.58	14.35	15.67
Mental & Behavioral Disorders	14.37	11.57	8.33	7.27	5.95	4.24	2.98	2.85	3.53

continued

TABLE A-2 Continued

	1990-93	1994-96	1997-99	2000-02	2003-05	2006-08	2009-11	2012-14	2015-17
Other Body System Diseases & Disorders									
Nervous System	6.82	5.99	5.74	6.34	6.06	6.11	6.10	6.88	7.36
Genitourinary System	7.59	6.46	5.47	4.77	5.26	5.25	4.80	4.44	4.86
Respiratory System	19.29	16.02	13.23	10.84	9.47	8.48	8.36	8.02	8.52
Digestive System*	15.93	13.65	9.83	8.69	7.29	5.77	5.25	4.90	4.79
Other Causes of Death									
Homicides	92.84	76.20	57.02	57.80	62.88	63.23	56.12	57.93	69.24
Transport Injuries	34.30	31.80	31.92	31.64	31.32	30.89	24.87	25.62	30.37
Other External Causes	23.88	19.69	17.11	14.89	13.66	12.73	11.17	10.64	11.50
All Other Causes of Death	24.03	20.18	17.65	19.99	17.11	14.84	13.23	11.85	12.46

*Changes in the International Classification of Diseases (ICD)-10 code for diseases of the digestive system in 2006 affected the comparability of both digestive system deaths and alcohol-induced deaths before and after that year. More information is provided in Chapter 5.
NOTE: The table shows all-cause and cause-specific mortality rates (in deaths per 100,000 people in the population) among non-Hispanic Black males ages 25–44. Mortality rates within each age group are age-adjusted by single-year of age to match the age distribution of the U.S. population in 2000 in order to improve comparability over time. These causes of death are exhaustive of all underlying cause-of-death codes and are based on the ICD-9 (1990–1998) and ICD-10 (1999–2017) codes. More information about the classification of causes of death can be found in Chapter 5.
SOURCE: Data from https://www.cdc.gov/nchs/nvss/nvss-restricted-data.htm.

TABLE A-3 Cause-Specific Mortality Rates (Deaths/100,000): Hispanic Males Ages 25-44

	1990-93	1994-96	1997-99	2000-02	2003-05	2006-08	2009-11	2012-14	2015-17
All-Cause Mortality	287.25	263.37	183.15	170.32	161.19	150.07	129.11	126.49	144.89
Cause-Specific Mortality									
Infectious & Parasitic Diseases									
HIV/AIDS	70.91	72.57	21.34	14.99	11.26	7.75	4.66	2.79	2.38
Non-HIV/AIDS	6.84	6.48	5.59	4.47	4.16	3.31	2.76	2.51	2.32
Cancers									
Liver	0.83	0.90	0.82	0.88	0.83	0.70	0.61	0.60	0.54
Lung	2.08	1.89	1.63	1.32	1.17	0.90	0.88	0.72	0.73
All Other Cancers	15.65	14.75	13.57	12.99	12.25	12.08	11.76	11.99	11.95
Cardio and Metabolic Diseases									
Endocrine, Nutrition, & Metabolic Diseases	4.65	4.75	3.69	3.99	4.03	4.24	4.30	4.68	5.09
Hypertensive Heart Disease	1.64	1.71	1.73	2.09	2.33	2.46	2.45	2.90	3.07
Ischemic & Other Circulatory Diseases	23.58	22.01	19.64	17.85	18.47	17.29	15.68	15.04	16.19
Substance Use and Mental Health									
Drug Poisonings	16.26	17.41	16.17	13.16	13.16	13.71	11.98	13.58	22.39
Alcohol-Induced*	12.60	10.72	8.50	7.33	6.18	6.82	7.21	6.83	7.78
Suicide	14.42	13.76	11.20	10.79	10.85	10.95	10.66	11.07	12.94
Mental & Behavioral Disorders	7.33	7.19	6.27	5.37	5.35	3.74	2.35	2.56	2.92

continued

TABLE A-3 Continued

	1990-93	1994-96	1997-99	2000-02	2003-05	2006-08	2009-11	2012-14	2015-17
Other Body System Diseases & Disorders									
Nervous System	2.53	2.14	2.00	2.05	2.36	2.32	2.23	2.23	2.72
Genitourinary System	1.46	1.03	1.08	1.14	1.12	1.09	1.23	1.11	1.14
Respiratory System	6.30	4.86	3.80	3.01	2.89	2.71	3.64	3.05	2.97
Digestive System*	8.58	7.29	6.80	5.86	5.28	4.75	4.25	3.92	3.95
Other Causes of Death									
Homicides	38.69	27.36	18.17	16.28	16.87	15.76	12.45	11.21	12.75
Transport Injuries	30.75	27.05	24.13	26.32	25.76	23.27	17.25	17.23	19.41
Other External Causes	13.90	12.10	11.09	10.97	10.67	10.46	8.44	8.10	8.68
All Other Causes of Death	8.26	7.43	5.94	9.45	6.21	5.77	4.30	4.39	4.96

*Changes in the International Classification of Diseases (ICD)-10 code for diseases of the digestive system in 2006 affected the comparability of both digestive system deaths and alcohol-induced deaths before and after that year. More information is provided in Chapter 5.

NOTE: The table shows all-cause and cause-specific mortality rates (in deaths per 100,000 people in the population) among Hispanic males ages 25–44. Mortality rates within each age group are age-adjusted by single-year of age to match the age distribution of the U.S. population in 2000 in order to improve comparability over time. These causes of death are exhaustive of all underlying cause-of-death codes and are based on the ICD-9 (1990–1998) and ICD-10 (1999–2017) codes. More information about the classification of causes of death can be found in Chapter 5.

SOURCE: Data from https://www.cdc.gov/nchs/nvss/nvss-restricted-data.htm.

TABLE A-4 Cause-Specific Mortality Rates (Deaths/100,000): Non-Hispanic White Females Ages 25-44

	1990-93	1994-96	1997-99	2000-02	2003-05	2006-08	2009-11	2012-14	2015-17
All-Cause Mortality	88.93	93.74	94.27	99.49	102.11	102.97	104.62	108.04	121.90
Cause-Specific Mortality									
Infectious & Parasitic Diseases									
HIV/AIDS	2.09	3.65	1.52	1.42	1.24	0.93	0.62	0.42	0.37
Non-HIV/AIDS	1.43	1.75	1.89	2.06	2.20	2.10	2.16	2.20	2.41
Cancers									
Liver	0.24	0.28	0.30	0.27	0.28	0.32	0.31	0.35	0.39
Lung	3.07	3.08	3.35	3.51	3.21	2.70	2.32	1.88	1.48
All Other Cancers	26.62	25.39	23.97	22.98	21.61	20.52	20.00	19.50	19.19
Cardio and Metabolic Diseases									
Endocrine, Nutrition, & Metabolic Diseases	3.06	3.35	3.51	3.88	3.99	4.11	4.20	4.55	4.92
Hypertensive Heart Disease	0.33	0.46	0.49	0.78	0.97	1.11	1.22	1.41	1.65
Ischemic & Other Circulatory Diseases	11.88	12.69	13.12	13.26	13.34	12.77	12.48	12.52	13.09
Substance Use and Mental Health									
Drug Poisonings	4.10	5.11	6.72	9.60	13.92	17.60	20.10	22.42	30.36
Alcohol-Induced*	1.71	1.87	1.97	2.04	1.91	2.31	2.72	3.03	3.83
Suicide	4.64	4.77	4.95	4.64	4.78	5.20	5.77	6.62	7.85
Mental & Behavioral Disorders	1.24	1.44	1.69	1.91	2.04	1.92	1.65	2.11	3.08

continued

TABLE A-4 Continued

	1990-93	1994-96	1997-99	2000-02	2003-05	2006-08	2009-11	2012-14	2015-17
Other Body System Diseases & Disorders									
Nervous System	2.28	2.32	2.50	2.76	2.80	3.16	3.20	3.33	3.53
Genitourinary System	0.71	0.79	0.88	1.03	1.09	1.07	1.17	1.11	1.18
Respiratory System	3.03	3.47	3.61	3.72	3.70	3.71	4.27	4.33	4.18
Digestive System*	2.68	2.57	2.84	3.24	3.21	3.01	3.11	3.30	3.75
Other Causes of Death									
Homicides	3.36	3.14	2.86	2.72	2.58	2.47	2.37	2.28	2.59
Transport Injuries	9.60	9.91	10.10	9.89	9.86	9.21	7.66	7.48	8.22
Other External Causes	2.59	2.77	2.98	2.98	3.12	3.10	3.18	3.21	3.49
All Other Causes of Death	4.26	4.92	5.01	6.81	6.25	5.66	6.11	5.99	6.38

*Changes in the International Classification of Diseases (ICD)-10 code for diseases of the digestive system in 2006 affected the comparability of both digestive system deaths and alcohol-induced deaths before and after that year. More information is provided in Chapter 5.
NOTE: The table shows all-cause and cause-specific mortality rates (in deaths per 100,000 people in the population) among non-Hispanic White females ages 25–44. Mortality rates within each age group are age-adjusted by single-year of age to match the age distribution of the U.S. population in 2000 in order to improve comparability over time. These causes of death are exhaustive of all underlying cause-of-death codes and are based on the ICD-9 (1990–1998) and ICD-10 (1999–2017) codes. More information about the classification of causes of death can be found in Chapter 5.
SOURCE: Data from https://www.cdc.gov/nchs/nvss/nvss-restricted-data.htm.

TABLE A-5 Cause-Specific Mortality Rates (Deaths/100,000): Non-Hispanic Black Females Ages 25-44

	1990-93	1994-96	1997-99	2000-02	2003-05	2006-08	2009-11	2012-14	2015-17
All-Cause Mortality	243.70	256.23	218.70	210.98	198.28	180.02	161.93	153.79	162.89
Cause-Specific Mortality									
Infectious & Parasitic Diseases									
HIV/AIDS	32.04	51.84	28.14	27.51	23.88	19.34	12.08	8.13	5.89
Non-HIV/AIDS	8.75	8.22	7.86	5.83	5.39	4.94	4.50	3.88	4.26
Cancers									
Liver	0.59	0.57	0.60	0.64	0.53	0.45	0.56	0.42	0.53
Lung	4.59	4.32	4.64	4.25	3.67	2.88	1.84	1.74	1.19
All Other Cancers	41.80	39.68	37.95	35.17	33.24	30.93	29.94	27.72	27.61
Cardio and Metabolic Diseases									
Endocrine, Nutrition, & Metabolic Diseases	8.34	9.43	9.09	8.90	9.45	9.82	9.47	9.93	11.31
Hypertensive Heart Disease	5.65	6.01	6.13	6.58	7.44	7.15	7.52	7.05	7.51
Ischemic & Other Circulatory Diseases	42.31	42.84	39.67	37.51	35.35	31.54	28.47	27.13	27.90
Substance Use and Mental Health									
Drug Poisonings	6.72	7.95	7.55	8.27	9.40	8.85	8.11	9.02	13.88
Alcohol-Induced*	5.38	3.63	2.63	2.02	1.48	1.40	1.49	1.58	2.15
Suicide	2.42	2.29	1.87	1.90	1.96	1.76	2.13	2.39	2.83
Mental & Behavioral Disorders	5.30	4.47	3.65	3.17	2.68	2.22	1.39	1.47	1.78

continued

TABLE A-5 Continued

	1990-93	1994-96	1997-99	2000-02	2003-05	2006-08	2009-11	2012-14	2015-17
Other Body System Diseases & Disorders									
Nervous System	4.21	4.32	4.14	4.74	5.18	5.14	5.10	5.53	5.93
Genitourinary System	4.01	3.48	3.66	4.35	4.57	4.31	4.10	3.71	4.10
Respiratory System	11.40	11.35	10.41	8.48	7.43	6.84	7.19	6.83	6.47
Digestive System*	8.18	6.69	6.09	5.91	5.20	4.54	4.22	4.17	4.02
Other Causes of Death									
Homicides	19.64	17.01	12.96	11.47	9.82	9.27	7.58	7.18	8.11
Transport Injuries	9.83	10.35	10.42	9.67	8.96	8.15	7.02	7.18	8.20
Other External Causes	5.15	4.99	4.19	4.03	3.79	3.45	3.28	3.20	3.36
All Other Causes of Death	17.41	16.81	17.05	20.58	18.86	17.05	15.94	15.52	15.84

*Changes in the International Classification of Diseases (ICD)-10 code for diseases of the digestive system in 2006 affected the comparability of both digestive system deaths and alcohol-induced deaths before and after that year. More information is provided in Chapter 5.
NOTE: The table shows all-cause and cause-specific mortality rates (in deaths per 100,000 people in the population) among non-Hispanic Black females ages 25–44. Mortality rates within each age group are age-adjusted by single-year of age to match the age distribution of the U.S. population in 2000 in order to improve comparability over time. These causes of death are exhaustive of all underlying cause-of-death codes and are based on the ICD-9 (1990–1998) and ICD-10 (1999–2017) codes. More information about the classification of causes of death can be found in Chapter 5.
SOURCE: Data from https://www.cdc.gov/nchs/nvss/nvss-restricted-data.htm.

TABLE A-6 Cause-Specific Mortality Rates (Deaths/100,000): Hispanic Females Ages 25-44

	1990-93	1994-96	1997-99	2000-02	2003-05	2006-08	2009-11	2012-14	2015-17
All-Cause Mortality	89.67	90.46	75.75	74.45	69.10	64.48	60.86	60.75	66.32
Cause-Specific Mortality									
Infectious & Parasitic Diseases									
HIV/AIDS	11.74	16.05	5.86	4.71	3.51	2.47	1.32	0.81	0.54
Non-HIV/AIDS	2.57	2.48	2.33	2.12	2.13	1.74	1.69	1.44	1.41
Cancers									
Liver	0.29	0.30	0.29	0.34	0.31	0.33	0.26	0.33	0.39
Lung	1.03	1.00	1.10	1.00	0.88	0.86	0.82	0.70	0.71
All Other Cancers	21.79	20.98	19.87	19.64	17.39	17.59	16.20	16.84	17.24
Cardio and Metabolic Diseases									
Endocrine, Nutrition, & Metabolic Diseases	2.50	2.92	2.76	2.60	2.73	2.46	2.80	2.87	2.88
Hypertensive Heart Disease	0.80	0.61	0.70	0.80	0.80	0.81	0.90	0.89	1.00
Ischemic & Other Circulatory Diseases	10.17	9.02	8.92	8.84	8.00	7.10	6.69	6.59	7.07
Substance Use and Mental Health									
Drug Poisonings	2.91	3.34	3.53	3.72	4.31	4.51	5.19	5.71	7.25
Alcohol-Induced*	2.00	1.91	1.75	1.47	1.38	1.47	1.58	1.70	2.50
Suicide	2.24	1.98	1.56	1.51	1.50	1.56	1.71	2.02	2.66
Mental & Behavioral Disorders	1.29	1.45	1.16	1.23	1.19	0.97	0.63	0.65	0.83

continued

TABLE A-6 Continued

	1990-93	1994-96	1997-99	2000-02	2003-05	2006-08	2009-11	2012-14	2015-17
Other Body System Diseases & Disorders									
Nervous System	1.54	1.40	1.54	1.47	1.55	1.63	1.67	1.73	1.80
Genitourinary System	0.98	0.92	0.93	1.04	0.99	0.86	0.94	0.89	0.99
Respiratory System	3.47	3.03	2.67	2.31	2.14	1.85	2.60	2.09	1.93
Digestive System*	2.50	2.55	2.45	2.44	2.23	2.31	1.99	1.97	2.14
Other Causes of Death									
Homicides	6.26	5.12	3.83	3.61	3.35	3.08	2.70	2.31	2.75
Transport Injuries	8.49	7.85	7.56	7.48	7.31	6.37	5.01	4.95	5.70
Other External Causes	1.79	1.80	1.86	1.67	1.68	1.35	1.39	1.38	1.41
All Other Causes of Death	5.33	5.74	5.09	6.44	5.72	5.14	4.78	4.89	5.13

*Changes in the International Classification of Diseases (ICD)-10 code for diseases of the digestive system in 2006 affected the comparability of both digestive system deaths and alcohol-induced deaths before and after that year. More information is provided in Chapter 5.

NOTE: The table shows all-cause and cause-specific mortality rates (in deaths per 100,000 people in the population) among Hispanic females ages 25–44. Mortality rates within each age group are age-adjusted by single-year of age to match the age distribution of the U.S. population in 2000 in order to improve comparability over time. These causes of death are exhaustive of all underlying cause-of-death codes and are based on the ICD-9 (1990–1998) and ICD-10 (1999–2017) codes. More information about the classification of causes of death can be found in Chapter 5.

SOURCE: Data from https://www.cdc.gov/nchs/nvss/nvss-restricted-data.htm.

TABLE A-7 Cause-Specific Mortality Rates (Deaths/100,000): Non-Hispanic White Males Ages 45-54

	1990-93	1994-96	1997-99	2000-02	2003-05	2006-08	2009-11	2012-14	2015-17
All-Cause Mortality	520.80	514.60	485.49	498.00	511.08	508.07	500.12	491.17	493.39
Cause-Specific Mortality									
Infectious & Parasitic Diseases									
HIV/AIDS	25.76	25.35	7.73	7.91	7.67	6.74	5.37	4.68	3.81
Non-HIV/AIDS	6.54	7.92	10.13	14.12	16.35	15.23	13.85	12.63	11.03
Cancers									
Liver	2.54	3.21	4.47	5.81	7.16	7.23	7.18	6.37	5.03
Lung	52.60	45.42	39.22	36.28	35.68	32.95	30.38	26.45	21.22
All Other Cancers	91.52	89.68	86.74	84.65	79.74	77.32	76.18	73.87	70.23
Cardio and Metabolic Diseases									
Endocrine, Nutrition, & Metabolic Diseases	14.95	16.48	17.22	19.01	20.68	20.83	21.55	22.24	24.93
Hypertensive Heart Disease	5.61	6.99	7.33	8.12	10.46	12.11	13.37	14.74	16.00
Ischemic & Other Circulatory Diseases	177.57	168.47	152.90	141.28	135.41	127.61	119.49	114.03	110.92
Substance Use and Mental Health									
Drug Poisonings	4.39	7.59	11.63	16.94	24.87	31.77	33.83	36.87	47.73
Alcohol-Induced*	13.33	14.37	15.07	15.83	16.25	18.71	20.30	21.49	21.83
Suicide	22.36	22.56	22.51	24.19	26.14	29.19	32.92	33.80	35.21
Mental & Behavioral Disorders	7.35	8.48	9.44	10.63	12.76	11.51	10.38	10.78	12.69

continued

TABLE A-7 Continued

	1990-93	1994-96	1997-99	2000-02	2003-05	2006-08	2009-11	2012-14	2015-17
Other Body System Diseases & Disorders									
Nervous System	7.01	7.36	8.08	9.56	10.52	10.73	11.19	11.53	11.98
Genitourinary System	3.18	3.28	3.59	4.30	4.79	5.16	4.89	5.00	5.62
Respiratory System	18.32	18.41	17.91	18.20	19.36	19.68	20.92	20.70	20.26
Digestive System*	17.23	17.69	19.56	22.85	23.58	22.76	22.79	21.83	19.94
Other Causes of Death									
Homicides	6.26	4.91	4.47	4.03	4.29	4.51	4.23	4.31	4.78
Transport Injuries	20.37	20.63	21.41	22.95	24.73	24.73	21.91	21.56	22.13
Other External Causes	14.19	14.39	14.74	15.52	16.91	17.26	17.07	16.50	15.99
All Other Causes of Death	9.74	11.40	11.35	15.83	13.71	12.03	12.33	11.81	12.04

*Changes in the International Classification of Diseases (ICD)-10 code for diseases of the digestive system in 2006 affected the comparability of both digestive system deaths and alcohol-induced deaths before and after that year. More information is provided in Chapter 5.

NOTE: The table shows all-cause and cause-specific mortality rates (in deaths per 100,000 people in the population) among non-Hispanic White males ages 45–54. Mortality rates within each age group are age-adjusted by single-year of age to match the age distribution of the U.S. population in 2000 in order to improve comparability over time. These causes of death are exhaustive of all underlying cause-of-death codes and are based on the ICD-9 (1990–1998) and ICD-10 (1999–2017) codes. More information about the classification of causes of death can be found in Chapter 5.

SOURCE: Data from https://www.cdc.gov/nchs/nvss/nvss-restricted-data.htm.

TABLE A-8 Cause-Specific Mortality Rates (Deaths/100,000): Non-Hispanic Black Males Ages 45-54

	1990-93	1994-96	1997-99	2000-02	2003-05	2006-08	2009-11	2012-14	2015-17
All-Cause Mortality	1236.32	1241.31	1082.49	1015.41	968.97	861.30	738.48	688.14	697.04
Cause-Specific Mortality									
Infectious & Parasitic Diseases									
HIV/AIDS	98.09	151.77	80.01	83.62	75.58	61.62	41.69	29.65	20.96
Non-HIV/AIDS	30.71	30.38	33.53	34.44	33.07	29.30	23.30	20.49	18.09
Cancers									
Liver	7.90	10.09	13.34	15.22	16.92	14.65	12.44	9.09	7.51
Lung	108.93	91.83	79.02	69.00	61.54	53.14	43.80	34.74	23.84
All Other Cancers	176.23	160.08	148.35	128.95	122.15	109.62	97.74	92.07	85.03
Cardio and Metabolic Diseases									
Endocrine, Nutrition, & Metabolic Diseases	43.04	47.42	45.02	43.53	42.50	40.47	38.38	40.57	46.95
Hypertensive Heart Disease	46.34	51.22	47.56	45.77	50.87	47.26	46.39	47.08	48.20
Ischemic & Other Circulatory Diseases	357.62	346.51	311.24	276.94	262.86	229.70	200.62	187.13	188.32
Substance Use and Mental Health									
Drug Poisonings	12.92	23.59	27.42	30.20	32.13	34.83	25.55	28.00	48.91
Alcohol-Induced*	36.81	32.65	24.76	19.84	16.54	14.49	13.39	12.22	12.04
Suicide	12.65	10.60	9.54	9.32	8.97	9.21	9.74	9.79	10.64
Mental & Behavioral Disorders	32.90	30.44	25.30	24.08	21.81	15.93	11.85	10.29	10.69

continued

TABLE A-8 Continued

	1990-93	1994-96	1997-99	2000-02	2003-05	2006-08	2009-11	2012-14	2015-17
Other Body System Diseases & Disorders									
Nervous System	12.75	12.62	13.88	14.30	14.55	14.95	14.55	14.97	16.07
Genitourinary System	20.19	19.43	20.81	22.62	22.87	20.06	17.78	16.65	18.74
Respiratory System	52.38	49.92	42.97	37.30	35.67	31.73	28.13	28.17	27.31
Digestive System*	45.89	43.93	42.76	43.25	39.05	31.81	26.41	22.44	19.39
Other Causes of Death									
Homicides	45.41	36.06	27.04	25.91	26.20	25.69	21.72	23.30	27.69
Transport Injuries	31.78	29.17	30.42	29.34	29.16	27.49	21.89	23.35	27.25
Other External Causes	31.46	30.46	27.83	24.43	24.59	21.96	18.49	16.20	17.58
All Other Causes of Death	32.33	33.12	31.70	37.35	31.94	27.39	24.61	21.95	21.84

*Changes in the International Classification of Diseases (ICD)-10 code for diseases of the digestive system in 2006 affected the comparability of both digestive system deaths and alcohol-induced deaths before and after that year. More information is provided in Chapter 5.
NOTE: The table shows all-cause and cause-specific mortality rates (in deaths per 100,000 people in the population) among non-Hispanic Black males ages 45–54. Mortality rates within each age group are age-adjusted by single-year of age to match the age distribution of the U.S. population in 2000 in order to improve comparability over time. These causes of death are exhaustive of all underlying cause-of-death codes and are based on the ICD-9 (1990–1998) and ICD-10 (1999–2017) codes. More information about the classification of causes of death can be found in Chapter 5.
SOURCE: Data from https://www.cdc.gov/nchs/nvss/nvss-restricted-data.htm.

TABLE A-9 Cause-Specific Mortality Rates (Deaths/100,000): Hispanic Males Ages 45-54

	1990-93	1994-96	1997-99	2000-02	2003-05	2006-08	2009-11	2012-14	2015-17
All-Cause Mortality	520.73	524.78	456.60	442.64	434.57	402.41	365.31	345.63	344.90
Cause-Specific Mortality									
Infectious & Parasitic Diseases									
HIV/AIDS	57.24	72.71	24.33	24.95	23.04	16.70	12.02	8.62	6.36
Non-HIV/AIDS	11.55	13.18	16.25	20.95	23.46	20.48	17.42	15.33	12.28
Cancers									
Liver	5.32	7.01	9.28	10.23	12.61	11.37	11.19	9.54	8.93
Lung	18.58	19.14	16.35	14.37	12.95	10.59	9.34	7.94	5.51
All Other Cancers	64.00	62.22	64.60	59.06	57.07	54.29	51.61	50.38	48.70
Cardio and Metabolic Diseases									
Endocrine, Nutrition, & Metabolic Diseases	18.19	23.71	20.91	20.11	20.95	19.86	18.76	19.97	20.72
Hypertensive Heart Disease	7.92	10.16	9.06	9.39	10.32	10.78	11.72	11.24	11.99
Ischemic & Other Circulatory Diseases	128.06	121.10	114.24	100.21	95.46	86.08	76.97	71.29	70.47
Substance Use and Mental Health									
Drug Poisonings	10.16	14.23	15.87	15.07	16.96	19.31	16.49	17.88	24.45
Alcohol-Induced*	37.36	37.65	32.29	30.24	27.16	28.48	26.96	26.75	27.02
Suicide	14.04	13.25	10.49	10.86	10.41	10.57	11.00	10.55	11.59
Mental & Behavioral Disorders	14.66	13.26	13.72	11.81	12.96	9.34	7.64	7.36	8.26

continued

TABLE A-9 Continued

	1990-93	1994-96	1997-99	2000-02	2003-05	2006-08	2009-11	2012-14	2015-17
Other Body System Diseases & Disorders									
Nervous System	5.60	4.24	4.94	5.69	5.40	6.36	6.17	6.14	7.13
Genitourinary System	5.28	4.33	4.80	5.45	6.56	6.91	6.20	5.82	6.14
Respiratory System	17.25	15.78	13.86	12.54	12.34	11.61	11.99	11.62	10.39
Digestive System*	29.07	26.74	28.32	29.81	28.65	25.41	24.58	22.76	18.61
Other Causes of Death									
Homicides	22.98	16.23	10.64	9.36	10.01	8.67	7.43	6.54	6.89
Transport Injuries	27.37	24.04	22.03	22.92	22.16	21.52	16.06	15.35	17.55
Other External Causes	15.85	14.23	14.49	13.63	15.09	13.79	12.56	12.27	13.24
All Other Causes of Death	10.26	11.56	10.09	15.98	11.01	10.30	9.21	8.29	8.69

*Changes in the International Classification of Diseases (ICD)-10 code for diseases of the digestive system in 2006 affected the comparability of both digestive system deaths and alcohol-induced deaths before and after that year. More information is provided in Chapter 5.
NOTE: The table shows all-cause and cause-specific mortality rates (in deaths per 100,000 people in the population) among Hispanic males ages 45–54. Mortality rates within each age group are age-adjusted by single-year of age to match the age distribution of the U.S. population in 2000 in order to improve comparability over time. These causes of death are exhaustive of all underlying cause-of-death codes and are based on the ICD-9 (1990–1998) and ICD-10 (1999–2017) codes. More information about the classification of causes of death can be found in Chapter 5.
SOURCE: Data from https://www.cdc.gov/nchs/nvss/nvss-restricted-data.htm.

TABLE A-10 Cause-Specific Mortality Rates (Deaths/100,000): Non-Hispanic White Females Ages 45-54

	1990-93	1994-96	1997-99	2000-02	2003-05	2006-08	2009-11	2012-14	2015-17
All-Cause Mortality	295.15	291.38	281.50	285.49	292.83	299.24	305.63	308.79	314.97
Cause-Specific Mortality									
Infectious & Parasitic Diseases									
HIV/AIDS	1.07	1.73	0.84	1.11	1.35	1.34	0.99	0.92	0.71
Non-HIV/AIDS	3.79	4.43	5.82	7.11	8.56	8.66	8.74	8.53	8.45
Cancers									
Liver	1.37	1.45	1.48	1.50	1.79	1.82	2.12	2.08	2.14
Lung	33.42	30.71	27.21	25.61	25.63	26.32	26.15	23.67	19.34
All Other Cancers	111.06	105.79	98.64	93.64	86.92	82.79	80.92	78.32	76.40
Cardio and Metabolic Diseases									
Endocrine, Nutrition, & Metabolic Diseases	10.04	10.80	11.33	12.37	12.53	12.16	12.37	13.38	14.76
Hypertensive Heart Disease	2.34	2.60	2.89	3.38	4.08	4.73	5.67	6.15	7.30
Ischemic & Other Circulatory Diseases	60.58	59.60	55.30	51.97	51.36	50.27	48.32	48.85	49.74
Substance Use and Mental Health									
Drug Poisonings	4.14	4.83	6.58	10.56	17.01	23.99	27.24	29.43	33.26
Alcohol-Induced*	4.21	4.58	4.57	5.17	6.13	7.53	9.47	10.28	11.73
Suicide	5.48	5.29	5.23	5.04	5.90	6.24	7.25	8.54	9.10
Mental & Behavioral Disorders	2.25	2.75	2.80	3.26	4.51	4.62	4.45	5.07	5.92

continued

TABLE A-10 Continued

	1990-93	1994-96	1997-99	2000-02	2003-05	2006-08	2009-11	2012-14	2015-17
Other Body System Diseases & Disorders									
Nervous System	6.03	6.66	7.48	8.33	8.67	8.95	9.15	9.39	9.70
Genitourinary System	2.54	2.65	2.89	3.39	3.73	3.82	3.83	3.86	4.51
Respiratory System	14.45	15.04	14.87	15.08	15.75	17.10	20.06	20.73	20.87
Digestive System*	9.47	9.21	9.64	11.26	11.68	12.36	13.05	13.82	13.70
Other Causes of Death									
Homicides	2.48	2.09	1.82	1.93	1.82	2.03	2.01	1.93	2.10
Transport Injuries	8.54	8.71	8.78	8.66	9.11	8.71	7.35	7.23	8.04
Other External Causes	4.15	4.11	4.58	4.97	5.76	5.93	6.34	6.29	6.64
All Other Causes of Death	7.70	8.36	8.77	11.15	10.53	9.85	10.16	10.30	10.55

*Changes in the International Classification of Diseases (ICD)-10 code for diseases of the digestive system in 2006 affected the comparability of both digestive system deaths and alcohol-induced deaths before and after that year. More information is provided in Chapter 5.

NOTE: The table shows all-cause and cause-specific mortality rates (in deaths per 100,000 people in the population) among non-Hispanic White females ages 45–54. Mortality rates within each age group are age-adjusted by single-year of age to match the age distribution of the U.S. population in 2000 in order to improve comparability over time. These causes of death are exhaustive of all underlying cause-of-death codes and are based on the ICD-9 (1990–1998) and ICD-10 (1999–2017) codes. More information about the classification of causes of death can be found in Chapter 5.

SOURCE: Data from https://www.cdc.gov/nchs/nvss/nvss-restricted-data.htm.

TABLE A-11 Cause-Specific Mortality Rates (Deaths/100,000): Non-Hispanic Black Females Ages 45-54

	1990-93	1994-96	1997-99	2000-02	2003-05	2006-08	2009-11	2012-14	2015-17
All-Cause Mortality	627.87	630.08	595.63	595.62	581.29	541.03	492.12	461.96	455.25
Cause-Specific Mortality									
Infectious & Parasitic Diseases									
HIV/AIDS	15.70	31.83	21.65	26.47	28.16	26.60	18.76	14.02	11.37
Non-HIV/AIDS	16.04	17.69	20.11	19.17	19.81	19.09	16.28	14.70	14.21
Cancers									
Liver	2.30	2.62	2.73	3.43	3.56	3.64	3.96	3.38	3.19
Lung	39.66	35.38	34.23	32.30	33.08	32.18	28.10	24.06	16.35
All Other Cancers	163.83	157.83	147.49	140.78	133.30	122.97	117.97	110.82	107.15
Cardio and Metabolic Diseases									
Endocrine, Nutrition, & Metabolic Diseases	35.09	36.40	35.48	35.67	33.78	30.07	28.98	30.23	31.95
Hypertensive Heart Disease	28.67	28.82	26.21	26.43	29.67	28.80	27.35	26.98	27.15
Ischemic & Other Circulatory Diseases	180.18	173.09	161.18	154.27	142.36	128.38	110.42	102.79	101.56
Substance Use and Mental Health									
Drug Poisonings	4.01	5.74	7.92	10.94	14.77	16.75	15.65	16.17	22.56
Alcohol-Induced*	11.24	9.59	7.84	7.41	5.76	5.63	6.28	6.19	6.58
Suicide	1.86	1.75	1.59	1.63	1.49	1.49	1.53	2.00	1.70
Mental & Behavioral Disorders	7.92	6.94	7.08	6.56	7.17	5.99	5.11	4.44	4.46

continued

TABLE A-11 Continued

	1990-93	1994-96	1997-99	2000-02	2003-05	2006-08	2009-11	2012-14	2015-17
Other Body System Diseases & Disorders									
Nervous System	9.31	10.03	10.59	12.75	12.73	12.95	12.02	13.22	13.72
Genitourinary System	12.57	14.13	13.87	15.18	15.18	15.50	14.50	12.23	12.86
Respiratory System	29.91	30.22	28.75	26.94	26.98	25.52	25.82	24.94	24.42
Digestive System*	22.27	20.49	19.23	20.96	19.77	17.98	15.58	15.33	14.24
Other Causes of Death									
Homicides	7.82	6.93	6.19	5.53	6.13	5.76	5.14	5.24	4.67
Transport Injuries	8.73	9.86	10.33	9.75	9.33	8.43	6.85	6.85	7.90
Other External Causes	8.04	7.96	7.53	7.32	7.80	7.07	6.91	6.03	6.53
All Other Causes of Death	22.73	22.81	25.62	32.12	30.48	26.23	24.91	22.35	22.66

*Changes in the International Classification of Diseases (ICD)-10 code for diseases of the digestive system in 2006 affected the comparability of both digestive system deaths and alcohol-induced deaths before and after that year. More information is provided in Chapter 5.

NOTE: The table shows all-cause and cause-specific mortality rates (in deaths per 100,000 people in the population) among non-Hispanic Black females ages 45–54. Mortality rates within each age group are age-adjusted by single-year of age to match the age distribution of the U.S. population in 2000 in order to improve comparability over time. These causes of death are exhaustive of all underlying cause-of-death codes and are based on the ICD-9 (1990–1998) and ICD-10 (1999–2017) codes. More information about the classification of causes of death can be found in Chapter 5.

SOURCE: Data from https://www.cdc.gov/nchs/nvss/nvss-restricted-data.htm.

TABLE A-12 Cause-Specific Mortality Rates (Deaths/100,000): Hispanic Females Ages 45-54

	1990-93	1994-96	1997-99	2000-02	2003-05	2006-08	2009-11	2012-14	2015-17
All-Cause Mortality	243.72	242.44	229.90	226.07	215.40	204.23	193.48	188.15	184.45
Cause-Specific Mortality									
Infectious & Parasitic Diseases									
HIV/AIDS	7.38	12.87	5.98	6.32	5.61	4.38	3.24	2.20	1.33
Non-HIV/AIDS	5.38	7.00	7.86	8.32	8.97	8.46	7.98	7.49	6.13
Cancers									
Liver	1.90	1.88	2.35	2.22	3.02	2.86	2.31	2.42	2.24
Lung	8.09	7.38	7.23	7.38	7.09	6.71	6.27	5.48	4.92
All Other Cancers	85.51	83.22	78.16	75.29	69.26	65.86	63.19	62.88	60.52
Cardio and Metabolic Diseases									
Endocrine, Nutrition, & Metabolic Diseases	12.99	15.63	14.94	14.55	14.27	12.76	10.97	10.98	11.82
Hypertensive Heart Disease	4.06	3.67	4.04	3.82	4.89	5.07	4.68	4.31	4.73
Ischemic & Other Circulatory Diseases	53.33	47.03	44.35	41.55	37.33	34.15	31.32	29.21	28.78
Substance Use and Mental Health									
Drug Poisonings	2.24	2.87	3.81	4.43	6.62	7.39	8.19	9.21	9.05
Alcohol-Induced*	6.07	5.94	4.70	5.18	4.10	5.86	6.87	6.49	6.95
Suicide	1.78	1.82	1.89	1.94	1.48	1.89	1.81	2.25	2.40
Mental & Behavioral Disorders	1.63	1.94	2.34	1.99	2.65	2.23	1.87	2.13	2.03

continued

TABLE A-12 Continued

	1990-93	1994-96	1997-99	2000-02	2003-05	2006-08	2009-11	2012-14	2015-17
Other Body System Diseases & Disorders									
Nervous System	3.30	3.16	3.61	3.92	4.49	4.28	3.96	4.85	5.00
Genitourinary System	4.48	3.85	4.24	4.43	4.26	4.60	4.36	3.95	4.33
Respiratory System	10.53	10.09	10.57	8.86	8.81	7.42	8.95	7.21	8.00
Digestive System*	10.94	10.90	10.90	10.80	10.62	10.84	10.24	10.41	8.82
Other Causes of Death									
Homicides	3.84	3.82	2.69	2.77	2.32	2.16	1.89	1.53	2.02
Transport Injuries	8.92	8.38	8.45	9.01	7.40	6.61	5.24	4.99	5.59
Other External Causes	2.59	2.70	2.78	2.98	3.26	2.89	3.01	2.78	2.55
All Other Causes of Death	8.77	8.28	9.01	10.32	8.94	7.80	7.13	7.38	7.23

*Changes in the International Classification of Diseases (ICD)-10 code for diseases of the digestive system in 2006 affected the comparability of both digestive system deaths and alcohol-induced deaths before and after that year. More information is provided in Chapter 5.

NOTE: The table shows all-cause and cause-specific mortality rates (in deaths per 100,000 people in the population) among Hispanic females ages 45–54. Mortality rates within each age group are age-adjusted by single-year of age to match the age distribution of the U.S. population in 2000 in order to improve comparability over time. These causes of death are exhaustive of all underlying cause-of-death codes and are based on the ICD-9 (1990–1998) and ICD-10 (1999–2017) codes. More information about the classification of causes of death can be found in Chapter 5.

SOURCE: Data from https://www.cdc.gov/nchs/nvss/nvss-restricted-data.htm.

TABLE A-13 Cause-Specific Mortality Rates (Deaths/100,000): Non-Hispanic White Males Ages 55-64

	1990-93	1994-96	1997-99	2000-02	2003-05	2006-08	2009-11	2012-14	2015-17
All-Cause Mortality	1345.21	1285.26	1209.87	1148.36	1093.17	1053.41	1036.31	1061.51	1085.81
Cause-Specific Mortality									
Infectious & Parasitic Diseases									
HIV/AIDS	10.16	10.32	3.45	3.92	3.91	3.89	3.67	3.61	3.49
Non-HIV/AIDS	13.68	14.82	16.12	19.25	22.62	25.61	28.81	31.70	29.26
Cancers									
Liver	8.75	10.51	10.36	11.28	12.94	16.40	21.23	25.59	25.79
Lung	198.91	180.50	166.02	151.82	135.58	119.16	107.07	99.68	90.90
All Other Cancers	271.79	264.68	255.22	247.11	234.17	224.16	215.61	211.11	206.16
Cardio and Metabolic Diseases									
Endocrine, Nutrition, & Metabolic Diseases	36.03	41.98	43.77	47.64	50.32	49.46	49.15	51.96	56.26
Hypertensive Heart Disease	15.20	16.27	16.34	16.13	19.37	21.33	24.01	28.50	32.61
Ischemic & Other Circulatory Diseases	517.49	479.54	432.67	381.81	335.84	302.15	280.07	277.70	279.65
Substance Use and Mental Health									
Drug Poisonings	2.58	2.72	3.72	4.83	8.19	13.03	17.60	22.56	31.42
Alcohol-Induced*	20.73	19.56	18.70	18.21	18.64	21.76	24.77	28.56	31.58
Suicide	24.75	22.75	21.73	21.96	22.96	25.39	28.52	31.27	32.79
Mental & Behavioral Disorders	11.37	12.08	11.30	11.22	13.83	14.74	15.16	18.09	21.01

continued

TABLE A-13 Continued

	1990-93	1994-96	1997-99	2000-02	2003-05	2006-08	2009-11	2012-14	2015-17
Other Body System Diseases & Disorders									
Nervous System	15.71	16.95	18.12	21.32	22.55	23.25	24.49	26.11	28.44
Genitourinary System	10.42	10.68	11.53	12.80	14.21	14.37	14.18	14.08	16.02
Respiratory System	85.70	82.17	81.51	76.51	74.92	72.92	73.39	77.12	81.10
Digestive System*	39.05	36.64	36.22	37.54	38.27	40.03	43.46	46.17	44.79
Other Causes of Death									
Homicides	4.76	4.12	3.50	3.19	2.96	3.02	2.94	2.96	3.42
Transport Injuries	20.00	20.02	21.44	21.46	22.52	21.97	20.28	20.64	23.30
Other External Causes	19.63	18.88	19.38	19.41	20.38	21.43	22.35	23.53	25.58
All Other Causes of Death	18.49	20.06	18.77	20.94	19.00	19.33	19.55	20.57	22.24

*Changes in the International Classification of Diseases (ICD)-10 code for diseases of the digestive system in 2006 affected the comparability of both digestive system deaths and alcohol-induced deaths before and after that year. More information is provided in Chapter 5.
NOTE: The table shows all-cause and cause-specific mortality rates (in deaths per 100,000 people in the population) among non-Hispanic White males ages 55–64. Mortality rates within each age group are age-adjusted by single-year of age to match the age distribution of the U.S. population in 2000 in order to improve comparability over time. These causes of death are exhaustive of all underlying cause-of-death codes and are based on the ICD-9 (1990–1998) and ICD-10 (1999–2017) codes. More information about the classification of causes of death can be found in Chapter 5.
SOURCE: Data from https://www.cdc.gov/nchs/nvss/nvss-restricted-data.htm.

TABLE A-14 Cause-Specific Mortality Rates (Deaths/100,000): Non-Hispanic Black Males Ages 55-64

	1990-93	1994-96	1997-99	2000-02	2003-05	2006-08	2009-11	2012-14	2015-17
All-Cause Mortality	2466.82	2371.54	2225.07	2074.04	1978.86	1838.06	1714.49	1669.12	1669.83
Cause-Specific Mortality									
Infectious & Parasitic Diseases									
HIV/AIDS	44.75	72.67	43.15	45.70	48.99	47.64	40.12	33.56	30.07
Non-HIV/AIDS	45.06	45.92	50.60	55.82	60.12	63.22	62.58	61.40	54.76
Cancers									
Liver	18.95	19.83	21.62	24.07	30.31	41.20	52.91	57.53	49.60
Lung	320.63	289.64	255.66	224.26	207.75	176.90	158.61	141.83	119.64
All Other Cancers	461.08	440.19	418.43	382.11	348.27	325.19	304.26	283.14	274.10
Cardio and Metabolic Diseases									
Endocrine, Nutrition, & Metabolic Diseases	92.87	104.37	108.46	110.79	111.13	100.68	97.21	97.72	103.79
Hypertensive Heart Disease	91.66	95.98	93.72	88.12	94.77	93.62	94.99	96.74	100.22
Ischemic & Other Circulatory Diseases	869.66	815.72	767.30	698.18	630.21	557.46	489.95	471.84	474.12
Substance Use and Mental Health									
Drug Poisonings	4.79	7.96	10.74	11.97	17.14	24.63	25.41	32.05	55.90
Alcohol-Induced*	41.88	34.98	31.92	24.02	23.09	21.73	23.58	23.96	25.36
Suicide	10.50	9.96	9.06	8.09	8.35	7.69	7.92	7.98	8.17
Mental & Behavioral Disorders	38.14	35.22	29.74	27.68	26.76	26.93	23.59	25.92	26.84

continued

TABLE A-14 Continued

	1990-93	1994-96	1997-99	2000-02	2003-05	2006-08	2009-11	2012-14	2015-17
Other Body System Diseases & Disorders									
Nervous System	21.91	21.19	23.44	25.50	27.97	28.29	30.00	31.40	33.96
Genitourinary System	47.87	46.92	49.81	54.03	59.84	56.18	50.77	49.23	50.37
Respiratory System	136.43	127.44	118.65	103.26	100.17	92.44	85.21	89.15	92.21
Digestive System*	72.29	67.50	65.20	67.05	64.63	62.94	62.07	60.66	54.58
Other Causes of Death									
Homicides	29.74	23.88	17.44	14.73	14.19	15.34	13.66	13.87	17.05
Transport Injuries	31.16	28.81	30.60	29.20	27.64	25.75	22.49	24.60	29.09
Other External Causes	43.80	40.67	36.08	33.22	32.89	29.63	28.71	28.88	30.69
All Other Causes of Death	43.67	42.70	43.44	46.24	44.63	40.59	40.45	37.65	39.35

*Changes in the International Classification of Diseases (ICD)-10 code for diseases of the digestive system in 2006 affected the comparability of both digestive system deaths and alcohol-induced deaths before and after that year. More information is provided in Chapter 5.
NOTE: The table shows all-cause and cause-specific mortality rates (in deaths per 100,000 people in the population) among non-Hispanic Black males ages 55–64. Mortality rates within each age group are age-adjusted by single-year of age to match the age distribution of the U.S. population in 2000 in order to improve comparability over time. These causes of death are exhaustive of all underlying cause-of-death codes and are based on the ICD-9 (1990–1998) and ICD-10 (1999–2017) codes. More information about the classification of causes of death can be found in Chapter 5.
SOURCE: Data from https://www.cdc.gov/nchs/nvss/nvss-restricted-data.htm.

TABLE A-15 Cause-Specific Mortality Rates (Deaths/100,000): Hispanic Males Ages 55-64

	1990-93	1994-96	1997-99	2000-02	2003-05	2006-08	2009-11	2012-14	2015-17
All-Cause Mortality	1090.13	1067.21	986.68	955.66	914.69	857.53	815.63	812.17	805.96
Cause-Specific Mortality									
Infectious & Parasitic Diseases									
HIV/AIDS	30.35	39.18	15.30	14.94	13.82	11.83	10.02	8.68	7.86
Non-HIV/AIDS	19.35	19.15	22.67	28.62	30.22	35.88	37.28	37.42	31.13
Cancers									
Liver	16.59	20.86	21.28	22.31	27.47	29.76	34.90	39.12	35.64
Lung	77.14	68.66	63.45	60.85	54.85	45.20	40.28	35.07	30.51
All Other Cancers	194.86	188.97	185.79	178.90	170.98	161.06	155.66	149.62	143.86
Cardio and Metabolic Diseases									
Endocrine, Nutrition, & Metabolic Diseases	54.32	65.59	68.36	67.08	68.35	58.60	56.56	54.60	58.01
Hypertensive Heart Disease	21.36	24.92	22.73	22.15	24.33	24.42	24.82	25.69	29.08
Ischemic & Other Circulatory Diseases	385.36	357.27	325.10	298.89	269.47	237.90	210.88	201.09	200.92
Substance Use and Mental Health									
Drug Poisonings	4.25	5.94	6.12	5.71	9.10	10.85	11.72	14.45	19.36
Alcohol-Induced*	50.36	49.43	44.84	40.55	35.60	37.63	39.94	42.33	43.74
Suicide	16.28	12.93	11.08	11.44	10.60	10.09	11.15	11.15	10.78
Mental & Behavioral Disorders	15.28	15.74	13.96	14.34	13.79	13.19	10.96	13.19	13.83

continued

TABLE A-15 Continued

	1990-93	1994-96	1997-99	2000-02	2003-05	2006-08	2009-11	2012-14	2015-17
Other Body System Diseases & Disorders									
Nervous System	10.49	10.27	10.38	12.91	14.08	13.75	14.49	15.67	17.62
Genitourinary System	13.55	12.59	17.22	17.44	19.86	20.22	19.53	16.91	19.06
Respiratory System	51.06	49.47	45.76	45.16	41.64	39.31	37.53	40.43	37.73
Digestive System*	52.63	51.20	48.17	48.48	48.80	47.86	46.54	50.63	44.78
Other Causes of Death									
Homicides	14.04	12.79	7.85	6.65	5.77	6.10	4.86	4.70	5.47
Transport Injuries	27.08	27.11	23.25	23.53	21.50	20.36	16.33	18.62	20.08
Other External Causes	20.54	18.56	18.72	17.20	18.70	18.55	18.45	17.39	19.69
All Other Causes of Death	15.26	16.58	14.64	18.52	15.76	14.97	13.72	15.41	16.80

*Changes in the International Classification of Diseases (ICD)-10 code for diseases of the digestive system in 2006 affected the comparability of both digestive system deaths and alcohol-induced deaths before and after that year. More information is provided in Chapter 5.
NOTE: The table shows all-cause and cause-specific mortality rates (in deaths per 100,000 people in the population) among Hispanic males ages 55–64. Mortality rates within each age group are age-adjusted by single-year of age to match the age distribution of the U.S. population in 2000 in order to improve comparability over time. These causes of death are exhaustive of all underlying cause-of-death codes and are based on the ICD-9 (1990–1998) and ICD-10 (1999–2017) codes. More information about the classification of causes of death can be found in Chapter 5.
SOURCE: Data from https://www.cdc.gov/nchs/nvss/nvss-restricted-data.htm.

TABLE A-16 Cause Specific Mortality Rates (Deaths/100,000): Non-Hispanic White Females Ages 55-64

	1990-93	1994-96	1997-99	2000-02	2003-05	2006-08	2009-11	2012-14	2015-17
All-Cause Mortality	767.20	760.35	744.90	725.14	690.00	649.92	626.15	636.17	659.91
Cause-Specific Mortality									
Infectious & Parasitic Diseases									
HIV/AIDS	0.67	0.78	0.36	0.50	0.51	0.54	0.59	0.65	0.58
Non-HIV/AIDS	9.40	10.32	12.47	14.30	16.11	17.22	18.35	19.98	19.68
Cancers									
Liver	3.79	4.18	4.20	4.17	4.38	4.84	5.59	6.91	7.52
Lung	104.57	104.83	102.16	99.36	92.37	82.07	72.55	71.31	67.85
All Other Cancers	248.27	240.99	230.35	220.33	208.23	193.52	184.21	176.72	173.33
Cardio and Metabolic Diseases									
Endocrine, Nutrition, & Metabolic Diseases	28.45	31.60	32.36	33.23	33.03	31.19	29.35	29.91	32.24
Hypertensive Heart Disease	7.93	8.26	8.04	8.65	9.80	10.15	10.80	13.00	15.27
Ischemic & Other Circulatory Diseases	207.31	198.87	186.79	167.74	146.38	129.17	117.39	115.02	120.51
Substance Use and Mental Health									
Drug Poisonings	3.45	3.04	3.68	5.00	7.70	11.54	14.79	18.66	22.56
Alcohol-Induced*	6.52	6.16	5.53	5.53	5.72	6.64	8.17	9.83	12.65
Suicide	5.16	4.39	4.30	4.45	4.47	5.06	5.51	6.41	7.59
Mental & Behavioral Disorders	3.81	4.48	4.24	4.37	5.44	6.15	6.89	8.26	8.86

continued

TABLE A-16 Continued

	1990-93	1994-96	1997-99	2000-02	2003-05	2006-08	2009-11	2012-14	2015-17
Other Body System Diseases & Disorders									
Nervous System	12.82	14.18	15.53	19.06	19.94	20.73	21.09	22.35	24.01
Genitourinary System	7.80	8.13	9.55	11.09	11.28	11.50	11.26	11.09	12.16
Respiratory System	60.25	62.77	65.78	64.67	63.50	59.10	59.99	62.93	68.84
Digestive System*	24.41	23.28	24.17	25.14	24.69	25.03	25.64	27.80	29.13
Other Causes of Death									
Homicides	1.72	1.51	1.36	1.42	1.31	1.36	1.39	1.45	1.57
Transport Injuries	9.29	9.71	10.22	9.59	9.23	8.26	6.65	6.74	7.12
Other External Causes	6.97	7.15	7.63	8.24	8.63	9.00	9.25	10.27	11.29
All Other Causes of Death	14.62	15.72	16.17	18.30	17.27	16.86	16.70	16.88	17.14

*Changes in the International Classification of Diseases (ICD)-10 code for diseases of the digestive system in 2006 affected the comparability of both digestive system deaths and alcohol-induced deaths before and after that year. More information is provided in Chapter 5.
NOTE: The table shows all-cause and cause-specific mortality rates (in deaths per 100,000 people in the population) among non-Hispanic White females ages 55–64. Mortality rates within each age group are age-adjusted by single-year of age to match the age distribution of the U.S. population in 2000 in order to improve comparability over time. These causes of death are exhaustive of all underlying cause-of-death codes and are based on the ICD-9 (1990–1998) and ICD-10 (1999–2017) codes. More information about the classification of causes of death can be found in Chapter 5.
SOURCE: Data from https://www.cdc.gov/nchs/nvss/nvss-restricted-data.htm.

TABLES A-17 Cause-Specific Mortality Rates (Deaths/100,000): Non-Hispanic Black Females Ages 55-64

	1990-93	1994-96	1997-99	2000-02	2003-05	2006-08	2009-11	2012-14	2015-17
All-Cause Mortality	1367.98	1340.86	1302.88	1229.51	1156.01	1062.93	996.15	988.17	1002.3
Cause-Specific Mortality									
Infectious & Parasitic Diseases									
HIV/AIDS	7.36	14.50	10.04	11.21	12.48	13.98	13.37	11.91	10.70
Non-HIV/AIDS	28.04	31.17	36.63	39.45	38.46	37.74	37.44	38.42	36.95
Cancers									
Liver	6.52	7.14	7.11	7.31	7.90	8.35	10.74	12.42	13.48
Lung	108.58	104.81	101.66	96.17	91.33	80.77	76.84	73.06	68.08
All Other Cancers	342.25	332.47	322.64	297.41	283.77	268.72	255.12	247.79	242.89
Cardio and Metabolic Diseases									
Endocrine, Nutrition, & Metabolic Diseases	92.73	102.92	103.15	92.53	89.30	79.47	70.27	68.49	70.97
Hypertensive Heart Disease	61.95	58.98	59.81	55.61	54.25	51.20	50.24	50.81	53.22
Ischemic & Other Circulatory Diseases	476.99	448.57	416.31	383.56	334.85	287.27	253.53	242.34	244.29
Substance Use and Mental Health									
Drug Poisonings	1.59	2.08	2.54	3.05	5.17	8.08	9.80	12.69	18.80
Alcohol-Induced*	12.18	11.60	8.26	7.85	6.86	6.06	7.13	7.71	9.26
Suicide	1.98	1.53	1.47	1.40	1.31	0.95	1.06	1.32	1.57
Mental & Behavioral Disorders	8.39	7.35	6.44	6.20	6.59	7.92	7.05	8.68	9.35

continued

TABLE A-17 Continued

	1990-93	1994-96	1997-99	2000-02	2003-05	2006-08	2009-11	2012-14	2015-17
Other Body System Diseases & Disorders									
Nervous System	15.27	15.56	19.57	21.65	23.52	24.29	24.51	26.00	27.98
Genitourinary System	35.72	36.12	39.27	42.55	43.89	40.94	35.45	33.83	36.48
Respiratory System	67.09	69.43	68.85	63.97	59.76	57.30	57.59	65.94	72.12
Digestive System*	41.75	37.22	37.57	36.64	34.61	32.58	31.59	32.85	31.68
Other Causes of Death									
Homicides	5.66	4.69	3.57	3.00	3.11	2.93	2.77	2.71	3.50
Transport Injuries	10.44	9.73	10.77	8.31	9.13	8.13	6.55	6.72	7.06
Other External Causes	12.72	12.67	12.87	12.61	11.27	11.01	11.35	11.41	11.22
All Other Causes of Death	30.77	32.31	34.37	39.03	38.46	35.23	33.76	33.08	32.72

*Changes in the International Classification of Diseases (ICD)-10 code for diseases of the digestive system in 2006 affected the comparability of both digestive system deaths and alcohol-induced deaths before and after that year. More information is provided in Chapter 5.
NOTE: The table shows all-cause and cause-specific mortality rates (in deaths per 100,000 people in the population) among non-Hispanic Black females ages 55–64. Mortality rates within each age group are age-adjusted by single-year of age to match the age distribution of the U.S. population in 2000 in order to improve comparability over time. These causes of death are exhaustive of all underlying cause-of-death codes and are based on the ICD-9 (1990–1998) and ICD-10 (1999–2017) codes. More information about the classification of causes of death can be found in Chapter 5.
SOURCE: Data from https://www.cdc.gov/nchs/nvss/nvss-restricted-data.htm.

TABLE A-18 Cause-Specific Mortality Rates (Deaths/100,000): Hispanic Females Ages 55-64

	1990-93	1994-96	1997-99	2000-02	2003-05	2006-08	2009-11	2012-14	2015-17
All-Cause Mortality	596.84	590.57	570.90	542.74	514.90	478.95	449.44	443.92	440.99
Cause-Specific Mortality									
Infectious & Parasitic Diseases									
HIV/AIDS	4.38	7.14	3.40	4.38	3.13	3.00	2.75	1.96	1.88
Non-HIV/AIDS	13.41	15.09	16.86	16.85	19.07	19.45	20.12	19.51	16.70
Cancers									
Liver	6.17	6.99	7.20	7.88	8.12	8.08	8.52	10.45	9.96
Lung	26.69	25.70	25.23	23.59	21.99	20.87	19.55	19.67	18.45
All Other Cancers	178.44	173.30	166.91	162.39	154.13	149.09	140.81	137.20	135.29
Cardio and Metabolic Diseases									
Endocrine, Nutrition, & Metabolic Diseases	50.36	55.92	55.65	52.42	47.74	40.38	34.07	35.54	35.63
Hypertensive Heart Disease	12.34	12.72	11.96	13.01	13.34	13.04	11.03	11.64	13.04
Ischemic & Other Circulatory Diseases	175.91	167.98	157.64	136.62	121.40	104.41	92.16	84.03	81.51
Substance Use and Mental Health									
Drug Poisonings	2.15	2.33	1.90	2.27	3.00	4.27	5.20	5.86	7.54
Alcohol-Induced*	8.38	7.22	6.71	5.78	6.02	5.99	7.22	7.95	9.27
Suicide	1.92	1.52	1.40	1.00	1.31	1.04	1.60	1.68	1.97
Mental & Behavioral Disorders	3.03	2.83	2.51	2.65	2.35	3.44	3.40	4.01	4.02

continued

TABLE A-18 Continued

	1990-93	1994-96	1997-99	2000-02	2003-05	2006-08	2009-11	2012-14	2015-17
Other Body System Diseases & Disorders									
Nervous System	7.38	8.79	8.37	10.02	10.94	11.22	11.39	10.94	13.44
Genitourinary System	12.33	11.14	12.47	14.99	15.49	16.35	13.82	13.09	12.98
Respiratory System	29.20	28.34	29.09	27.48	26.79	24.27	25.17	26.08	27.01
Digestive System*	33.06	30.19	29.48	27.19	27.89	26.97	27.12	28.01	26.52
Other Causes of Death									
Homicides	2.54	1.90	1.76	1.74	1.36	1.27	1.39	1.35	1.24
Transport Injuries	10.70	11.77	10.74	9.95	9.75	7.12	6.09	6.59	6.45
Other External Causes	5.09	5.03	6.03	5.66	6.05	5.85	5.45	4.96	5.56
All Other Causes of Death	13.37	14.69	15.60	16.86	15.02	12.84	12.58	13.41	12.53

*Changes in the International Classification of Diseases (ICD)-10 code for diseases of the digestive system in 2006 affected the comparability of both digestive system deaths and alcohol-induced deaths before and after that year. More information is provided in Chapter 5.

NOTE: The table shows all-cause and cause-specific mortality rates (in deaths per 100,000 people in the population) among Hispanic females ages 55–64. Mortality rates within each age group are age-adjusted by single-year of age to match the age distribution of the U.S. population in 2000 in order to improve comparability over time. These causes of death are exhaustive of all underlying cause-of-death codes and are based on the ICD-9 (1990–1998) and ICD-10 (1999–2017) codes. More information about the classification of causes of death can be found in Chapter 5.

SOURCE: Data from https://www.cdc.gov/nchs/nvss/nvss-restricted-data.htm.

CAUSE-SPECIFIC MORTALITY ESTIMATES BY SEX, AGE GROUP, RACE AND ETHNICITY, AND METROPOLITAN AREA STATUS

TABLE A-19 Cause-Specific Mortality Rates (Deaths/100,000): Non-Hispanic White Males Ages 25-64 Living in Large Central Metropolitan Areas

	Ages 25-44				Ages 45-64			
	1990-93	2000-02	2009-11	2015-17	1990-93	2000-02	2009-11	2015-17
All-Cause Mortality	224.09	175.70	162.81	198.33	838.81	726.52	654.79	643.75
Cause-Specific Mortality								
Infectious & Parasitic Diseases								
HIV/AIDS	52.48	7.17	1.77	0.89	31.00	7.56	4.61	3.29
Non-HIV/AIDS	4.48	4.64	2.58	2.12	10.24	18.32	19.59	16.44
Cancers								
Liver	0.50	0.59	0.50	0.50	5.39	8.37	12.65	12.11
Lung	4.16	3.27	1.87	1.28	106.61	75.66	52.39	40.02
All Other Cancers	21.09	16.91	14.80	13.61	156.34	142.78	120.81	109.37
Cardio and Metabolic Diseases								
Endocrine, Nutrition, & Metabolic Diseases	5.17	5.00	5.16	5.41	22.65	29.16	29.27	32.16
Hypertensive Heart Disease	1.26	2.26	3.32	3.61	11.38	13.26	19.21	22.46
Ischemic & Other Circulatory Diseases	29.10	25.30	21.03	20.02	299.82	221.27	163.46	153.04

continued

TABLE A-19 Continued

	Ages 25-44				Ages 45-64			
	1990-93	2000-02	2009-11	2015-17	1990-93	2000-02	2009-11	2015-17
Substance Use and Mental Health								
Drug Poisonings	12.13	21.29	34.08	66.45	4.64	14.66	29.50	44.80
Alcohol-Induced*	5.43	3.62	3.73	4.69	16.76	16.23	18.76	20.81
Suicide	21.80	20.65	23.75	26.79	22.31	21.80	29.74	31.05
Mental & Behavioral Disorders	4.18	5.45	3.61	5.09	9.00	11.88	12.51	14.93
Other Body System Diseases & Disorders								
Nervous System	3.01	3.33	3.72	3.64	10.24	13.58	15.21	16.40
Genitourinary System	1.02	1.04	0.95	0.89	6.09	7.28	7.76	8.39
Respiratory System	4.68	3.73	3.69	3.19	41.05	36.88	35.16	34.84
Digestive System*	5.99	4.70	3.71	3.84	26.40	27.15	28.16	25.25
Other Causes of Death								
Homicides	8.29	4.66	3.77	4.28	5.95	2.90	2.65	2.95
Transport Injuries	21.39	19.46	15.37	16.34	16.42	17.81	16.42	17.27
Other External Causes	10.44	9.21	8.72	9.13	13.99	14.51	18.08	18.58
All Other Causes of Death	7.47	13.42	6.66	6.57	22.54	25.46	18.87	19.59

*Changes in the International Classification of Diseases (ICD)-10 code for diseases of the digestive system in 2006 affected the comparability of both digestive system deaths and alcohol-induced deaths before and after that year. More information is provided in Chapter 5.

NOTE: The table shows the all-cause and cause-specific mortality rates among non-Hispanic White working-age males in large central metropolitan areas by age group (25–44 and 45–64). These causes of death are exhaustive of all underlying cause-of-death codes and are based on the International Classification of Diseases (ICD)-9 (1990–1998) and ICD-10 (1999–2017) codes. Mortality rates within each age group are age-adjusted by single-year of age groups to match the age distribution of the U.S. population in 2000 in order to improve comparability over time. More information about the classification of causes of death can be found in Chapter 5.

SOURCE: Data from https://www.cdc.gov/nchs/nvss/nvss-restricted-data.htm.

TABLE A-20 Cause-Specific Mortality Rates (Deaths/100,000): Non-Hispanic White Males Ages 25-64 Living in Large Fringe Metropolitan Areas

	Ages 25-44				Ages 45-64			
	1990-93	2000-02	2009-11	2015-17	1990-93	2000-02	2009-11	2015-17
All-Cause Mortality	196.33	194.49	197.98	238.70	830.48	756.98	731.08	767.65
Cause-Specific Mortality								
Infectious & Parasitic Diseases								
HIV/AIDS	24.13	5.00	1.57	0.83	11.49	3.64	3.32	3.07
Non-HIV/AIDS	3.11	5.01	3.30	3.23	8.89	17.59	22.13	21.09
Cancers								
Liver	0.46	0.50	0.59	0.44	4.62	8.01	13.26	14.64
Lung	4.35	4.05	2.39	1.80	110.53	83.18	62.84	51.78
All Other Cancers	20.61	18.33	16.67	15.28	149.76	143.27	129.20	122.41
Cardio and Metabolic Diseases								
Endocrine, Nutrition, & Metabolic Diseases	4.72	5.89	6.42	7.74	23.43	30.72	33.03	40.14
Hypertensive Heart Disease	0.90	1.96	3.32	4.22	8.81	11.40	17.71	24.09
Ischemic & Other Circulatory Diseases	30.26	29.45	27.71	24.98	307.50	232.57	183.54	181.24
Substance Use and Mental Health								
Drug Poisonings	8.52	20.64	37.53	67.36	3.22	13.28	29.61	45.95
Alcohol-Induced*	4.69	4.21	4.46	6.08	16.56	17.98	21.52	26.39

continued

TABLE A-20 Continued

	Ages 25-44				Ages 45-64			
	1990-93	2000-02	2009-11	2015-17	1990-93	2000-02	2009-11	2015-17
Suicide	23.07	25.07	29.19	35.35	24.03	24.20	31.83	36.07
Mental & Behavioral Disorders	3.54	4.42	3.31	5.20	8.45	10.67	13.01	17.99
Other Body System Diseases & Disorders								
Nervous System	3.11	4.24	4.53	5.25	10.94	14.53	17.26	19.91
Genitourinary System	0.91	1.14	1.29	1.39	5.90	7.46	8.89	10.01
Respiratory System	4.27	4.20	4.42	4.61	45.28	42.37	43.17	47.64
Digestive System*	5.09	4.95	4.98	5.03	25.62	27.80	33.39	32.18
Other Causes of Death								
Homicides	7.65	5.25	4.88	6.01	5.03	3.42	3.06	4.11
Transport Injuries	27.85	27.22	21.96	24.01	20.27	22.80	21.26	23.67
Other External Causes	11.96	11.87	11.42	12.21	15.53	16.91	21.15	22.25
All Other Causes of Death	7.17	11.10	8.04	7.68	24.62	25.17	21.90	23.01

*Changes in the International Classification of Diseases (ICD)-10 code for diseases of the digestive system in 2006 affected the comparability of both digestive system deaths and alcohol-induced deaths before and after that year. More information is provided in Chapter 5.

NOTE: The table shows the all-cause and cause-specific mortality rates among non-Hispanic White working-age males in large fringe metropolitan areas by age group (25–44 and 45–64). These causes of death are exhaustive of all underlying cause-of-death codes and are based on the International Classification of Diseases (ICD)-9 (1990–1998) and ICD-10 (1999–2017) codes. Mortality rates within each age group are age-adjusted by single-year of age groups to match the age distribution of the U.S. population in 2000 in order to improve comparability over time. More information about the classification of causes of death can be found in Chapter 5.

SOURCE: Data from https://www.cdc.gov/nchs/nvss/nvss-restricted-data.htm.

TABLE A-21 Cause-Specific Mortality Rates (Deaths/100,000): Non-Hispanic White Males Ages 25–64 Living in Small/Medium Metropolitan Areas

	Ages 25-44				Ages 45-64			
	1990-93	2000-02	2009-11	2015-17	1990-93	2000-02	2009-11	2015-17
All-Cause Mortality	195.06	197.63	207.13	236.00	865.54	782.03	767.40	812.83
Cause-Specific Mortality								
Infectious & Parasitic Diseases								
HIV/AIDS	16.73	3.67	1.34	0.55	7.67	2.99	2.66	2.53
Non-HIV/AIDS	2.70	4.56	3.31	3.33	8.92	16.09	21.34	21.09
Cancers								
Liver	0.48	0.57	0.29	0.65	4.68	7.23	12.78	14.30
Lung	4.52	4.27	2.67	1.90	117.10	86.61	67.06	55.95
All Other Cancers	20.02	18.32	16.77	15.66	152.06	146.06	131.39	129.79
Cardio and Metabolic Diseases								
Endocrine, Nutrition, & Metabolic Diseases	5.05	6.40	7.57	8.38	25.16	32.92	36.35	42.71
Hypertensive Heart Disease	0.93	1.47	2.85	3.67	7.33	8.86	16.28	22.13
Ischemic & Other Circulatory Diseases	31.52	32.00	29.50	27.40	327.81	250.03	201.16	203.12

continued

TABLE A-21 Continued

	Ages 25-44				Ages 45-64			
	1990-93	2000-02	2009-11	2015-17	1990-93	2000-02	2009-11	2015-17
Substance Use and Mental Health								
Drug Poisonings	5.74	16.12	33.33	52.49	2.74	10.60	25.26	35.29
Alcohol-Induced*	4.23	4.22	3.94	6.14	16.19	16.91	22.07	27.25
Suicide	25.21	26.74	32.29	37.93	24.05	24.53	33.68	38.16
Mental & Behavioral Disorders	3.17	4.22	3.76	5.61	8.77	10.47	13.09	19.40
Other Body System Diseases & Disorders								
Nervous System	3.79	4.35	5.24	5.99	11.14	15.25	18.56	21.51
Genitourinary System	1.01	1.36	1.60	1.75	6.50	8.25	9.59	11.40
Respiratory System	4.40	4.71	4.57	5.38	51.33	45.19	48.51	53.74
Digestive System*	4.92	4.68	5.07	5.02	25.11	27.34	33.88	34.94
Other Causes of Death								
Homicides	7.28	4.83	4.81	6.75	5.38	3.38	3.34	3.75
Transport Injuries	32.36	31.55	26.14	25.66	23.76	26.16	25.22	28.05
Other External Causes	15.21	13.84	13.58	13.67	17.81	19.43	23.56	23.37
All Other Causes of Death	5.80	9.75	8.50	8.09	22.03	23.73	21.64	24.36

*Changes in the International Classification of Diseases (ICD)-10 code for diseases of the digestive system in 2006 affected the comparability of both digestive system deaths and alcohol-induced deaths before and after that year. More information is provided in Chapter 5.

NOTE: The table shows the all-cause and cause-specific mortality rates among non-Hispanic White working-age males in small/medium metropolitan areas by age group (25–44 and 45–64). These causes of death are exhaustive of all underlying cause-of-death codes and are based on the International Classification of Diseases (ICD)-9 (1990–1998) and ICD-10 (1999–2017) codes. Mortality rates within each age group are age-adjusted by single-year of age groups to match the age distribution of the U.S. population in 2000 in order to improve comparability over time. More information about the classification of causes of death can be found in Chapter 5.

SOURCE: Data from https://www.cdc.gov/nchs/nvss/nvss-restricted-data.htm.

TABLE A-22 Cause-Specific Mortality Rates (Deaths/100,000): Non-Hispanic White Males Ages 25–64 Living in Nonmetropolitan Areas

	Ages 25-44				Ages 45-64			
	1990-93	2000-02	2009-11	2015-17	1990-93	2000-02	2009-11	2015-17
All-Cause Mortality	206.06	224.03	234.74	267.70	878.35	820.79	816.05	872.60
Cause-Specific Mortality								
Infectious & Parasitic Diseases								
HIV/AIDS	11.17	3.00	1.22	0.78	4.97	2.26	2.06	1.88
Non-HIV/AIDS	2.24	4.15	3.41	3.81	7.92	15.66	22.33	22.52
Cancers								
Liver	0.50	0.64	0.60	0.61	4.65	7.49	12.74	14.40
Lung	4.65	5.07	3.08	2.21	117.06	93.49	76.36	65.99
All Other Cancers	20.40	19.90	18.06	17.33	149.42	148.59	136.53	136.76
Cardio and Metabolic Diseases								
Endocrine, Nutrition, & Metabolic Diseases	5.48	7.06	8.23	10.00	23.84	31.80	38.30	45.71
Hypertensive Heart Disease	0.82	1.47	3.20	4.24	6.27	8.06	14.46	21.78
Ischemic & Other Circulatory Diseases	35.27	37.45	35.24	35.69	341.08	273.22	224.44	228.81
Substance Use and Mental Health								
Drug Poisonings	5.20	15.91	34.84	51.40	2.17	8.78	24.85	32.85
Alcohol-Induced*	3.64	3.58	3.84	5.45	15.58	15.59	19.53	24.67
Suicide	25.93	29.88	34.65	42.09	24.65	26.23	33.34	39.21
Mental & Behavioral Disorders	2.88	3.83	2.85	4.68	8.50	9.44	10.55	14.73

continued

TABLE A-22 Continued

	Ages 25-44				Ages 45-64			
	1990-93	2000-02	2009-11	2015-17	1990-93	2000-02	2009-11	2015-17
Other Body System Diseases & Disorders								
Nervous System	3.64	4.23	5.84	6.06	11.01	14.94	17.55	21.14
Genitourinary System	1.11	1.33	1.58	2.07	5.94	8.53	9.72	12.21
Respiratory System	4.21	5.30	6.23	6.13	51.63	49.04	53.04	61.50
Digestive System*	4.47	4.84	5.55	6.03	24.39	27.20	34.44	36.88
Other Causes of Death								
Homicides	8.52	5.63	5.19	7.12	5.54	3.70	3.76	4.69
Transport Injuries	41.58	42.55	35.42	36.06	28.11	32.05	32.06	33.14
Other External Causes	19.35	18.30	16.55	16.87	22.72	22.14	26.11	27.66
All Other Causes of Death	5.01	9.91	9.15	9.06	22.88	22.58	23.87	26.05

*Changes in the International Classification of Diseases (ICD)-10 code for diseases of the digestive system in 2006 affected the comparability of both digestive system deaths and alcohol-induced deaths before and after that year. More information is provided in Chapter 5.

NOTE: The table shows the all-cause and cause-specific mortality rates among non-Hispanic White working-age males in nonmetropolitan areas by age group (25–44 and 45–64). These causes of death are exhaustive of all underlying cause-of-death codes and are based on the International Classification of Diseases (ICD)-9 (1990–1998) and ICD-10 (1999–2017) codes. Mortality rates within each age group are age-adjusted by single-year of age groups to match the age distribution of the U.S. population in 2000 in order to improve comparability over time. More information about the classification of causes of death can be found in Chapter 5.

SOURCE: Data from https://www.cdc.gov/nchs/nvss/nvss-restricted-data.htm.

TABLE A-23 Cause-Specific Mortality Rates (Deaths/100,000): Non-Hispanic Black Males Ages 25-64 Living in Large Central Metropolitan Areas

	Ages 25-44				Ages 45-64			
	1990-93	2000-02	2009-11	2015-17	1990-93	2000-02	2009-11	2015-17
All-Cause Mortality	642.70	393.44	286.44	314.95	1728.58	1425.35	1090.57	1033.27
Cause-Specific Mortality								
Infectious & Parasitic Diseases								
HIV/AIDS	170.11	51.37	15.75	8.07	98.39	67.48	35.21	20.67
Non-HIV/AIDS	19.21	20.89	10.72	6.96	40.42	60.33	50.32	36.09
Cancers								
Liver	1.84	1.77	1.12	1.29	13.45	20.33	29.68	23.33
Lung	10.27	6.46	2.05	2.17	188.71	122.98	81.38	54.43
All Other Cancers	31.51	22.20	17.94	16.59	258.54	219.22	167.41	146.98
Cardio and Metabolic Diseases								
Endocrine, Nutrition, & Metabolic Diseases	13.91	11.65	12.68	14.29	61.12	66.32	56.92	64.70
Hypertensive Heart Disease	10.75	12.21	13.69	14.94	70.58	69.25	70.79	71.86
Ischemic & Other Circulatory Diseases	73.80	55.13	45.98	45.46	536.65	424.38	297.21	280.36
Substance Use and Mental Health								
Drug Poisonings	25.17	24.32	16.79	36.22	12.83	33.21	32.50	63.80
Alcohol-Induced*	14.20	3.74	1.81	2.13	41.04	20.26	14.20	14.11
Suicide	17.59	14.18	13.74	15.56	12.04	9.03	9.19	9.40

continued

TABLE A-23 Continued

	Ages 25-44				Ages 45-64			
	1990-93	2000-02	2009-11	2015-17	1990-93	2000-02	2009-11	2015-17
Mental & Behavioral Disorders	15.49	8.00	2.97	3.46	35.16	26.64	16.81	16.20
Other Body System Diseases & Disorders								
Nervous System	6.85	6.21	5.74	6.96	16.17	17.65	19.27	21.31
Genitourinary System	8.52	4.58	4.37	4.03	32.02	33.97	27.72	28.14
Respiratory System	21.84	11.76	8.27	7.99	86.59	61.99	47.78	49.13
Digestive System*	17.24	7.77	4.85	4.40	57.51	50.06	39.46	30.14
Other Causes of Death								
Homicides	107.53	66.40	61.42	73.44	42.01	20.25	17.89	22.57
Transport Injuries	27.57	27.09	21.67	26.17	26.35	24.30	17.83	23.55
Other External Causes	21.84	13.60	10.67	11.62	33.89	24.88	22.04	23.03
All Other Causes of Death	27.45	24.10	14.22	13.21	65.10	52.81	36.96	33.45

*Changes in the International Classification of Diseases (ICD)-10 code for diseases of the digestive system in 2006 affected the comparability of both digestive system deaths and alcohol-induced deaths before and after that year. More information is provided in Chapter 5.
NOTE: The table shows the all-cause and cause-specific mortality rates among non-Hispanic Black working-age males in large central metropolitan areas by age group (25–44 and 45–64). These causes of death are exhaustive of all underlying cause-of-death codes and are based on the International Classification of Diseases (ICD)-9 (1990–1998) and ICD-10 (1999–2017) codes. Mortality rates within each age group are age-adjusted by single-year of age groups to match the age distribution of the U.S. population in 2000 in order to improve comparability over time. More information about the classification of causes of death can be found in Chapter 5.
SOURCE: Data from https://www.cdc.gov/nchs/nvss/nvss-restricted-data.htm.

TABLE A-24 Cause-Specific Mortality Rates (Deaths/100,000): Non-Hispanic Black Males Ages 25-64 Living in Large Fringe Metropolitan Areas

	Ages 25-44				Ages 45-64			
	1990-93	2000-02	2009-11	2015-17	1990-93	2000-02	2009-11	2015-17
All-Cause Mortality	477.67	361.79	293.62	336.96	1622.77	1413.10	1145.14	1128.13
Cause-Specific Mortality								
Infectious & Parasitic Diseases								
HIV/AIDS	88.98	36.78	13.09	7.48	45.42	42.86	26.51	19.22
Non-HIV/AIDS	12.33	20.09	9.56	7.44	28.03	53.09	47.14	40.28
Cancers								
Liver	1.94	1.39	1.47	0.76	10.07	17.62	29.41	28.74
Lung	9.79	6.35	2.83	2.39	191.65	133.70	94.50	67.56
All Other Cancers	29.67	22.16	19.17	16.19	252.68	220.05	174.67	158.43
Cardio and Metabolic Diseases								
Endocrine, Nutrition, & Metabolic Diseases	11.41	11.94	13.90	16.22	62.84	77.21	67.59	75.97
Hypertensive Heart Disease	8.17	8.87	11.62	15.30	53.24	51.55	60.16	63.57
Ischemic & Other Circulatory Diseases	72.59	63.10	53.09	55.17	549.16	442.99	319.44	313.83
Substance Use and Mental Health								
Drug Poisonings	10.89	15.61	13.56	33.79	4.56	16.25	24.38	43.56
Alcohol-Induced*	11.64	4.02	2.06	2.19	36.36	21.98	19.07	17.95
Suicide	16.05	15.25	14.75	17.14	10.58	9.48	8.63	10.14

continued

TABLE A-24 Continued

	Ages 25-44				Ages 45-64			
	1990-93	2000-02	2009-11	2015-17	1990-93	2000-02	2009-11	2015-17
Mental & Behavioral Disorders	11.23	6.46	2.98	3.30	32.06	24.05	15.78	18.51
Other Body System Diseases & Disorders								
Nervous System	6.44	7.13	7.04	7.50	17.47	20.33	22.34	26.43
Genitourinary System	6.33	5.18	5.49	6.83	26.77	34.55	33.28	34.13
Respiratory System	14.12	10.20	8.78	9.28	81.97	65.43	52.72	57.01
Digestive System*	12.90	6.65	5.60	5.44	53.40	48.76	44.05	37.54
Other Causes of Death								
Homicides	69.75	47.72	51.07	69.10	30.27	19.46	15.41	21.02
Transport Injuries	37.67	36.62	29.08	34.63	35.74	34.13	26.18	32.24
Other External Causes	25.01	14.89	13.62	12.57	34.07	27.75	23.76	22.62
All Other Causes of Death	20.78	21.36	14.84	14.24	66.42	51.85	40.12	39.35

*Changes in the International Classification of Diseases (ICD)-10 code for diseases of the digestive system in 2006 affected the comparability of both digestive system deaths and alcohol-induced deaths before and after that year. More information is provided in Chapter 5.
NOTE: The table shows the all-cause and cause-specific mortality rates among non-Hispanic Black working-age males in large fringe metropolitan areas by age group (25–44 and 45–64). These causes of death are exhaustive of all underlying cause-of-death codes and are based on the International Classification of Diseases (ICD)-9 (1990–1998) and ICD-10 (1999–2017) codes. Mortality rates within each age group are age-adjusted by single-year of age groups to match the age distribution of the U.S. population in 2000 in order to improve comparability over time. More information about the classification of causes of death can be found in Chapter 5.
SOURCE: Data from https://www.cdc.gov/nchs/nvss/nvss-restricted-data.htm.

TABLE A-25 Cause-Specific Mortality Rates (Deaths/100,000): Non-Hispanic Black Males Ages 25-64 Living in Small/Medium Metropolitan Areas

	Ages 25-44				Ages 45-64			
	1990-93	2000-02	2009-11	2015-17	1990-93	2000-02	2009-11	2015-17
All-Cause Mortality	430.49	311.60	277.68	323.99	1717.77	1451.65	1171.27	1176.07
Cause-Specific Mortality								
Infectious & Parasitic Diseases								
HIV/AIDS	52.66	29.46	11.48	7.16	25.25	27.21	23.06	15.19
Non-HIV/AIDS	9.09	15.68	10.30	6.29	31.43	46.98	47.39	40.39
Cancers								
Liver	1.29	1.30	1.14	1.06	9.68	15.89	25.58	22.57
Lung	9.28	9.94	3.65	3.23	205.67	136.72	101.20	73.72
All Other Cancers	27.86	22.58	18.36	16.38	275.19	232.50	175.52	167.14
Cardio and Metabolic Diseases								
Endocrine, Nutrition, & Metabolic Diseases	9.68	10.98	14.27	20.18	68.16	81.36	69.09	73.94
Hypertensive Heart Disease	8.29	6.68	11.88	14.24	50.08	53.45	53.85	61.03
Ischemic & Other Circulatory Diseases	76.93	56.31	59.01	56.46	602.37	481.43	354.24	348.03
Substance Use and Mental Health								
Drug Poisonings	8.22	9.41	15.84	32.79	3.15	12.27	17.00	32.16
Alcohol-Induced*	11.69	4.54	2.23	2.88	36.39	22.89	17.55	17.78
Suicide	18.57	13.16	11.45	15.87	9.68	7.67	8.45	10.12
Mental & Behavioral Disorders	11.47	5.75	2.63	4.75	36.44	24.49	15.97	20.81

continued

TABLE A-25 Continued

	Ages 25-44				Ages 45-64			
	1990-93	2000-02	2009-11	2015-17	1990-93	2000-02	2009-11	2015-17
Other Body System Diseases & Disorders								
Nervous System	7.62	6.60	6.62	9.27	15.85	24.91	24.46	27.16
Genitourinary System	4.84	4.92	6.48	5.27	31.66	39.22	38.14	38.80
Respiratory System	13.58	9.53	8.03	9.48	80.06	65.93	53.51	64.19
Digestive System*	12.75	7.59	5.43	5.16	55.37	48.27	39.82	44.11
Other Causes of Death								
Homicides	61.65	29.88	34.04	50.85	35.33	14.60	12.50	18.18
Transport Injuries	40.43	34.79	26.83	35.61	41.17	32.17	26.50	34.54
Other External Causes	29.30	15.80	12.26	13.24	39.95	37.05	27.79	24.76
All Other Causes of Death	15.30	16.70	15.75	13.82	64.90	46.64	39.62	41.46

*Changes in the International Classification of Diseases (ICD)-10 code for diseases of the digestive system in 2006 affected the comparability of both digestive system deaths and alcohol-induced deaths before and after that year. More information is provided in Chapter 5.

NOTE: The table shows the all-cause and cause-specific mortality rates among non-Hispanic Black working-age males in small/medium metropolitan areas by age group (25–44 and 45–64). These causes of death are exhaustive of all underlying cause-of-death codes and are based on the International Classification of Diseases (ICD)-9 (1990–1998) and ICD-10 (1999–2017) codes. Mortality rates within each age group are age-adjusted by single-year of age groups to match the age distribution of the U.S. population in 2000 in order to improve comparability over time. More information about the classification of causes of death can be found in Chapter 5.

SOURCE: Data from https://www.cdc.gov/nchs/nvss/nvss-restricted-data.htm.

TABLE A-26 Cause-Specific Mortality Rates (Deaths/100,000): Non-Hispanic Black Males Ages 25-64 Living in Nonmetropolitan Areas

	Ages 25-44				Ages 45-64			
	1990-93	2000-02	2009-11	2015-17	1990-93	2000-02	2009-11	2015-17
All-Cause Mortality	466.44	340.00	287.46	316.37	1809.05	1555.88	1249.46	1255.89
Cause-Specific Mortality								
Infectious & Parasitic Diseases								
HIV/AIDS	45.87	26.10	11.99	6.62	23.65	29.47	19.55	15.67
Non-HIV/AIDS	8.44	15.55	11.05	7.48	26.20	46.72	44.95	40.55
Cancers								
Liver	1.75	1.48	0.88	1.21	9.61	13.11	21.53	22.35
Lung	11.38	8.09	2.56	2.86	206.54	164.85	114.67	89.43
All Other Cancers	30.87	22.75	19.24	16.94	263.02	239.57	194.65	179.39
Cardio and Metabolic Diseases								
Endocrine, Nutrition, & Metabolic Diseases	11.45	12.24	12.68	17.72	67.63	75.68	73.61	85.40
Hypertensive Heart Disease	8.30	8.84	13.10	14.31	49.04	48.37	51.91	64.03
Ischemic & Other Circulatory Diseases	89.95	65.93	57.02	59.13	682.93	540.28	382.93	383.06
Substance Use and Mental Health								
Drug Poisonings	5.36	7.22	9.13	18.70	2.10	7.05	12.70	21.32
Alcohol-Induced*	10.91	5.42	2.51	1.99	32.70	23.51	16.68	18.74

continued

TABLE A-26 Continued

	Ages 25-44				Ages 45-64			
	1990-93	2000-02	2009-11	2015-17	1990-93	2000-02	2009-11	2015-17
Suicide	15.47	12.70	12.46	13.78	13.21	9.09	9.09	10.38
Mental & Behavioral Disorders	12.09	5.58	3.50	3.32	36.19	24.74	15.88	17.46
Other Body System Diseases & Disorders								
Nervous System	7.67	6.03	6.36	8.32	17.60	19.59	23.12	26.56
Genitourinary System	5.19	5.35	5.31	6.09	32.36	39.74	38.79	40.84
Respiratory System	14.61	8.40	8.89	10.10	85.96	70.42	61.73	63.51
Digestive System*	11.99	8.22	7.10	5.78	49.26	50.26	42.27	39.82
Other Causes of Death								
Homicides	59.19	29.62	36.33	45.83	35.17	16.35	12.84	17.73
Transport Injuries	65.66	49.86	37.25	45.41	52.20	50.48	38.81	45.42
Other External Causes	35.07	23.42	14.59	15.41	54.61	40.05	31.33	31.91
All Other Causes of Death	15.20	17.19	15.52	15.37	69.07	46.55	42.43	42.31

*Changes in the International Classification of Diseases (ICD)-10 code for diseases of the digestive system in 2006 affected the comparability of both digestive system deaths and alcohol-induced deaths before and after that year. More information is provided in Chapter 5.

NOTE: The table shows the all-cause and cause-specific mortality rates among non-Hispanic Black working-age males in nonmetropolitan areas by age group (25–44 and 45–64). These causes of death are exhaustive of all underlying cause-of-death codes and are based on the International Classification of Diseases (ICD)-9 (1990–1998) and ICD-10 (1999–2017) codes. Mortality rates within each age group are age-adjusted by single-year of age groups to match the age distribution of the U.S. population in 2000 in order to improve comparability over time. More information about the classification of causes of death can be found in Chapter 5.

SOURCE: Data from https://www.cdc.gov/nchs/nvss/nvss-restricted-data.htm.

TABLE A-27 Cause-Specific Mortality Rates (Deaths/100,000): Hispanic Males Ages 25-64 Living in Large Central Metropolitan Areas

	Ages 25-44				Ages 45-64			
	1990-93	2000-02	2009-11	2015-17	1990-93	2000-02	2009-11	2015-17
All-Cause Mortality	305.04	163.68	121.72	136.69	743.83	622.96	513.87	493.84
Cause-Specific Mortality								
Infectious & Parasitic Diseases								
HIV/AIDS	88.22	14.75	3.87	2.15	60.69	22.23	10.55	6.39
Non-HIV/AIDS	7.68	6.32	3.47	2.39	14.33	25.58	25.46	18.08
Cancers								
Liver	0.89	0.79	0.64	0.50	9.66	14.16	19.06	17.87
Lung	2.27	1.35	0.91	0.68	41.33	32.67	21.33	14.98
All Other Cancers	15.69	12.43	11.33	11.64	106.46	100.49	86.84	80.78
Cardio and Metabolic Diseases								
Endocrine, Nutrition, & Metabolic Diseases	4.45	3.50	3.87	4.78	27.92	33.89	30.76	32.78
Hypertensive Heart Disease	1.82	2.25	2.75	3.18	15.17	15.61	17.27	19.69
Ischemic & Other Circulatory Diseases	23.02	17.32	15.08	14.96	224.38	173.55	124.68	115.63
Substance Use and Mental Health								
Drug Poisonings	16.81	13.63	12.04	22.82	8.31	12.59	14.81	23.45
Alcohol-Induced*	12.83	6.55	5.09	5.81	44.29	32.03	29.14	29.50

continued

TABLE A-27 Continued

	Ages 25-44				Ages 45-64			
	1990-93	2000-02	2009-11	2015-17	1990-93	2000-02	2009-11	2015-17
Suicide	13.10	9.73	9.59	11.31	14.84	10.99	10.50	10.44
Mental & Behavioral Disorders	7.43	5.61	2.31	2.95	14.87	12.63	8.74	10.20
Other Body System Diseases & Disorders								
Nervous System	2.57	1.74	2.16	2.41	7.01	7.95	8.76	10.01
Genitourinary System	1.58	1.11	1.13	1.07	7.93	9.98	10.26	10.32
Respiratory System	6.68	2.65	3.35	2.66	29.95	24.20	20.69	19.12
Digestive System*	8.33	4.86	3.58	3.36	36.55	32.41	27.39	23.45
Other Causes of Death								
Homicides	43.95	16.30	11.70	12.09	22.27	7.47	5.42	4.64
Transport Injuries	25.97	21.96	14.62	17.31	22.96	19.55	13.39	16.03
Other External Causes	12.99	9.99	9.57	9.31	16.20	13.62	15.02	16.56
All Other Causes of Death	8.75	10.85	4.67	5.33	18.70	21.35	13.82	13.93

*Changes in the International Classification of Diseases (ICD)-10 code for diseases of the digestive system in 2006 affected the comparability of both digestive system deaths and alcohol-induced deaths before and after that year. More information is provided in Chapter 5.

NOTE: The table shows the all-cause and cause-specific mortality rates among Hispanic working-age males in large central metropolitan areas by age group (25–44 and 45–64). These causes of death are exhaustive of all underlying cause-of-death codes and are based on the International Classification of Diseases (ICD)-9 (1990–1998) and ICD-10 (1999–2017) codes. Mortality rates within each age group are age-adjusted by single-year of age groups to match the age distribution of the U.S. population in 2000 in order to improve comparability over time. More information about the classification of causes of death can be found in Chapter 5.

SOURCE: Data from https://www.cdc.gov/nchs/nvss/nvss-restricted-data.htm.

TABLE A-28 Cause-Specific Mortality Rates (Deaths/100,000): Hispanic Males Ages 25-64 Living in Large Fringe Metropolitan Areas

	Ages 25-44				Ages 45-64			
	1990-93	2000-02	2009-11	2015-17	1990-93	2000-02	2009-11	2015-17
All-Cause Mortality	245.27	180.83	142.50	165.00	731.25	692.76	592.62	604.94
Cause-Specific Mortality								
Infectious & Parasitic Diseases								
HIV/AIDS	33.50	9.96	3.91	1.62	15.41	10.33	6.94	5.23
Non-HIV/AIDS	5.07	6.75	3.78	3.65	15.97	31.38	32.12	27.31
Cancers								
Liver	0.64	1.03	0.52	0.59	9.74	18.04	23.99	22.48
Lung	1.47	0.95	0.89	0.99	40.13	32.06	21.07	15.66
All Other Cancers	15.03	12.98	11.67	11.75	115.85	107.89	91.72	88.17
Cardio and Metabolic Diseases								
Endocrine, Nutrition, & Metabolic Diseases	4.95	5.13	4.76	5.31	40.74	47.08	37.80	41.69
Hypertensive Heart Disease	1.34	1.83	1.93	3.02	9.79	14.27	18.12	18.87
Ischemic & Other Circulatory Diseases	24.37	18.42	17.05	19.21	226.10	186.49	137.68	132.09
Substance Use and Mental Health								
Drug Poisonings	17.35	16.47	15.64	25.79	9.05	15.53	19.73	28.04
Alcohol-Induced*	11.76	7.42	5.34	6.73	38.34	40.81	30.02	35.12

continued

TABLE A-28 Continued

	Ages 25-44				Ages 45-64			
	1990-93	2000-02	2009-11	2015-17	1990-93	2000-02	2009-11	2015-17
Suicide	16.55	11.92	11.66	15.08	13.30	9.34	12.20	13.16
Mental & Behavioral Disorders	6.67	4.45	2.33	2.79	15.81	13.09	8.90	10.97
Other Body System Diseases & Disorders								
Nervous System	2.77	3.07	2.41	3.33	9.43	9.49	10.33	14.51
Genitourinary System	1.22	1.36	1.42	1.33	11.11	10.24	13.83	12.38
Respiratory System	5.52	3.92	3.96	3.85	32.00	25.97	23.61	24.96
Digestive System*	9.29	6.31	6.02	5.00	41.42	43.96	44.21	41.78
Other Causes of Death								
Homicides	27.33	14.10	12.47	14.18	12.39	5.69	5.10	6.37
Transport Injuries	38.25	32.04	20.51	22.97	33.49	28.38	19.33	23.48
Other External Causes	14.11	11.81	10.12	11.12	19.67	16.00	17.52	19.61
All Other Causes of Death	8.09	10.92	6.10	6.68	21.52	26.72	18.40	23.08

*Changes in the International Classification of Diseases (ICD)-10 code for diseases of the digestive system in 2006 affected the comparability of both digestive system deaths and alcohol-induced deaths before and after that year. More information is provided in Chapter 5.

NOTE: The table shows the all-cause and cause-specific mortality rates among Hispanic working-age males in large fringe metropolitan areas by age group (25–44 and 45–64). These causes of death are exhaustive of all underlying cause-of-death codes and are based on the International Classification of Diseases (ICD)-9 (1990–1998) and ICD-10 (1999–2017) codes. Mortality rates within each age group are age-adjusted by single-year of age groups to match the age distribution of the U.S. population in 2000 in order to improve comparability over time. More information about the classification of causes of death can be found in Chapter 5.

SOURCE: Data from https://www.cdc.gov/nchs/nvss/nvss-restricted-data.htm.

TABLE A-29 Cause-Specific Mortality Rates (Deaths/100,000): Hispanic Males Ages 25-64 Living in Small/Medium Metropolitan Areas

	Ages 25-44				Ages 45-64			
	1990-93	2000-02	2009-11	2015-17	1990-93	2000-02	2009-11	2015-17
All-Cause Mortality	220.74	175.49	137.31	156.08	699.50	640.03	568.27	571.72
Cause-Specific Mortality								
Infectious & Parasitic Diseases								
HIV/AIDS	22.58	6.23	2.44	1.66	15.01	7.59	3.64	2.83
Non-HIV/AIDS	4.64	6.29	4.60	3.30	13.21	25.77	26.57	25.19
Cancers								
Liver	1.02	0.41	0.35	0.57	8.82	14.72	21.83	21.83
Lung	0.52	1.11	0.59	0.68	38.92	33.45	19.13	14.89
All Other Cancers	15.91	13.33	10.88	11.98	104.84	94.12	84.83	82.38
Cardio and Metabolic Diseases								
Endocrine, Nutrition, & Metabolic Diseases	3.81	5.03	5.75	6.16	46.44	47.70	39.39	37.03
Hypertensive Heart Disease	0.87	1.90	1.00	1.96	6.80	7.88	12.66	13.46
Ischemic & Other Circulatory Diseases	21.01	18.76	16.57	15.37	214.03	171.72	130.43	130.88
Substance Use and Mental Health								
Drug Poisonings	13.37	12.01	13.92	21.67	2.98	9.99	15.82	21.58
Alcohol-Induced*	10.92	7.76	6.52	7.13	36.29	35.87	33.62	36.70

continued

TABLE A-29 Continued

	Ages 25-44				Ages 45-64			
	1990-93	2000-02	2009-11	2015-17	1990-93	2000-02	2009-11	2015-17
Suicide	14.39	13.88	13.80	17.07	16.98	12.71	11.02	11.65
Mental & Behavioral Disorders	7.84	4.69	2.31	2.96	12.67	16.33	9.39	13.75
Other Body System Diseases & Disorders								
Nervous System	1.82	2.16	2.14	4.11	6.63	8.42	12.07	12.62
Genitourinary System	0.92	0.75	1.35	1.10	9.00	8.02	11.96	13.30
Respiratory System	3.82	4.42	4.39	3.86	30.66	25.52	24.03	25.54
Digestive System*	8.04	5.58	4.67	6.15	42.45	43.56	41.29	35.76
Other Causes of Death								
Homicides	21.02	10.78	8.70	9.33	10.33	3.35	6.44	5.44
Transport Injuries	45.45	39.31	21.27	22.82	38.93	31.26	22.54	23.76
Other External Causes	16.54	14.35	12.71	12.79	24.43	18.21	21.30	22.78
All Other Causes of Death	6.24	6.74	3.36	5.41	20.10	23.84	20.30	20.35

*Changes in the International Classification of Diseases (ICD)-10 code for diseases of the digestive system in 2006 affected the comparability of both digestive system deaths and alcohol-induced deaths before and after that year. More information is provided in Chapter 5.

NOTE: The table shows the all-cause and cause-specific mortality rates among Hispanic working-age males in small/medium metropolitan areas by age group (25–44 and 45–64). These causes of death are exhaustive of all underlying cause-of-death codes and are based on the International Classification of Diseases (ICD)-9 (1990–1998) and ICD-10 (1999–2017) codes. Mortality rates within each age group are age-adjusted by single-year of age groups to match the age distribution of the U.S. population in 2000 in order to improve comparability over time. More information about the classification of causes of death can be found in Chapter 5.

SOURCE: Data from https://www.cdc.gov/nchs/nvss/nvss-restricted-data.htm.

TABLE A-30 Cause-Specific Mortality Rates (Deaths/100,000): Hispanic Males Ages 25-64 Living in Nonmetropolitan Areas

	Ages 25-44				Ages 45-64			
	1990-93	2000-02	2009-11	2015-17	1990-93	2000-02	2009-11	2015-17
All-Cause Mortality	241.11	191.64	155.40	165.45	783.61	683.79	626.17	614.95
Cause-Specific Mortality								
Infectious & Parasitic Diseases								
HIV/AIDS	16.62	4.93	2.41	1.73	8.80	6.33	3.33	3.66
Non-HIV/AIDS	3.20	7.62	4.62	2.68	13.78	27.84	33.38	27.58
Cancers								
Liver	0.33	1.42	0.64	0.82	11.00	14.54	22.72	25.15
Lung	2.05	1.90	0.70	0.41	47.40	30.09	23.54	17.85
All Other Cancers	13.54	12.29	9.68	9.57	102.79	101.03	95.02	82.79
Cardio and Metabolic Diseases								
Endocrine, Nutrition, & Metabolic Diseases	5.55	4.49	5.84	6.64	44.16	50.63	41.81	43.41
Hypertensive Heart Disease	0.68	1.02	1.70	2.98	6.43	6.32	11.36	13.22
Ischemic & Other Circulatory Diseases	24.20	17.98	16.09	20.09	282.44	190.95	144.47	150.71
Substance Use and Mental Health								
Drug Poisonings	9.03	11.54	14.37	20.06	2.27	8.81	13.82	17.55
Alcohol-Induced*	11.99	6.72	5.22	5.15	39.67	31.67	32.75	32.61

continued

TABLE A-30 Continued

	Ages 25-44				Ages 45-64			
	1990-93	2000-02	2009-11	2015-17	1990-93	2000-02	2009-11	2015-17
Suicide	23.62	17.19	16.23	20.61	18.52	16.14	13.93	15.15
Mental & Behavioral Disorders	6.24	4.87	2.68	2.92	12.63	11.79	9.61	9.49
Other Body System Diseases & Disorders								
Nervous System	2.22	2.35	2.57	3.29	9.18	10.48	11.04	13.46
Genitourinary System	1.04	1.00	1.53	1.27	7.61	11.98	14.25	15.32
Respiratory System	5.00	2.61	4.94	3.02	30.60	32.75	27.39	27.13
Digestive System*	7.00	6.68	5.65	5.19	42.43	44.14	51.48	44.77
Other Causes of Death								
Homicides	23.30	14.06	8.30	9.73	13.89	8.95	4.45	5.62
Transport Injuries	54.92	46.56	31.63	29.10	46.67	37.62	29.46	28.23
Other External Causes	25.56	18.80	15.85	15.24	23.31	21.50	24.68	23.34
All Other Causes of Death	4.98	7.62	4.77	4.95	20.05	20.23	17.68	17.90

*Changes in the International Classification of Diseases (ICD)-10 code for diseases of the digestive system in 2006 affected the comparability of both digestive system deaths and alcohol-induced deaths before and after that year. More information is provided in Chapter 5.

NOTE: The table shows the all-cause and cause-specific mortality rates among Hispanic working-age males in nonmetropolitan areas by age group (25–44 and 45–64). These causes of death are exhaustive of all underlying cause-of-death codes and are based on the International Classification of Diseases (ICD)-9 (1990–1998) and ICD-10 (1999–2017) codes. Mortality rates within each age group are age-adjusted by single-year of age groups to match the age distribution of the U.S. population in 2000 in order to improve comparability over time. More information about the classification of causes of death can be found in Chapter 5.

SOURCE: Data from https://www.cdc.gov/nchs/nvss/nvss-restricted-data.htm.

TABLE A-31 Cause-Specific Mortality Rates (Deaths/100,000): Non-Hispanic White Females Ages 25-64 Living in Large Central Metropolitan Areas

	Ages 25-44				Ages 45-64			
	1990-93	2000-02	2009-11	2015-17	1990-93	2000-02	2009-11	2015-17
All-Cause Mortality	87.28	91.51	89.26	103.91	483.81	443.70	397.17	396.31
Cause-Specific Mortality								
Infectious & Parasitic Diseases								
HIV/AIDS	2.97	1.47	0.48	0.26	1.29	1.06	0.71	0.60
Non-HIV/AIDS	1.52	2.21	1.98	1.93	6.05	10.10	11.80	11.13
Cancers								
Liver	0.22	0.22	0.25	0.35	2.32	2.50	3.47	4.08
Lung	3.15	3.09	1.85	1.17	64.49	53.87	39.93	32.86
All Other Cancers	26.04	21.96	18.27	17.32	168.96	142.66	118.01	106.95
Cardio and Metabolic Diseases								
Endocrine, Nutrition, & Metabolic Diseases	2.80	3.31	3.29	3.64	16.04	18.74	16.23	17.64
Hypertensive Heart Disease	0.37	0.79	1.23	1.32	5.30	6.07	8.13	9.97
Ischemic & Other Circulatory Diseases	11.07	11.29	9.75	9.77	113.91	89.23	64.58	63.42
Substance Use and Mental Health								
Drug Poisonings	4.70	9.93	18.35	29.43	4.45	9.02	21.91	28.10
Alcohol-Induced*	2.03	1.96	2.14	2.98	5.97	5.35	8.06	10.65

continued

TABLE A-31 Continued

	Ages 25-44				Ages 45-64			
	1990-93	2000-02	2009-11	2015-17	1990-93	2000-02	2009-11	2015-17
Suicide	4.55	4.46	5.14	6.79	5.48	4.78	6.40	7.90
Mental & Behavioral Disorders	1.32	2.16	1.85	2.85	2.99	4.07	5.48	6.89
Other Body System Diseases & Disorders								
Nervous System	2.26	2.41	2.61	2.78	8.50	12.08	12.95	13.34
Genitourinary System	0.69	0.88	0.91	0.89	4.67	5.96	5.98	6.15
Respiratory System	2.96	3.33	3.39	3.05	31.38	31.33	29.43	30.67
Digestive System*	2.79	2.95	2.48	3.14	15.44	15.37	16.07	16.39
Other Causes of Death								
Homicides	3.29	2.04	1.65	1.87	2.09	1.31	1.23	1.46
Transport Injuries	7.68	7.04	5.08	5.43	7.56	7.10	5.22	5.64
Other External Causes	2.25	2.50	2.92	3.21	4.80	5.72	7.38	8.12
All Other Causes of Death	4.60	7.49	5.66	5.70	12.12	17.39	14.21	14.35

*Changes in the International Classification of Diseases (ICD)-10 code for diseases of the digestive system in 2006 affected the comparability of both digestive system deaths and alcohol-induced deaths before and after that year. More information is provided in Chapter 5.

NOTE: The table shows the all-cause and cause-specific mortality rates among non-Hispanic White working-age females in large central metropolitan areas by age group (25–44 and 45–64). These causes of death are exhaustive of all underlying cause-of-death codes and are based on the International Classification of Diseases (ICD)-9 (1990–1998) and ICD-10 (1999–2017) codes. Mortality rates within each age group are age-adjusted by single-year of age groups to match the age distribution of the U.S. population in 2000 in order to improve comparability over time. More information about the classification of causes of death can be found in Chapter 5.

SOURCE: Data from https://www.cdc.gov/nchs/nvss/nvss-restricted-data.htm.

TABLE A-32 Cause-Specific Mortality Rates (Deaths/100,000): Non-Hispanic White Females Ages 25-64 Living in Large Fringe Metropolitan Areas

	Ages 25-44				Ages 45-64			
	1990-93	2000-02	2009-11	2015-17	1990-93	2000-02	2009-11	2015-17
All-Cause Mortality	87.77	103.48	111.15	132.37	474.45	460.73	440.00	471.08
Cause-Specific Mortality								
Infectious & Parasitic Diseases								
HIV/AIDS	1.50	1.05	0.40	0.32	0.74	0.53	0.66	0.50
Non-HIV/AIDS	1.41	2.45	2.58	2.62	6.37	10.12	13.16	13.80
Cancers								
Liver	0.21	0.29	0.40	0.46	2.26	2.51	3.54	4.22
Lung	3.03	3.84	2.60	1.51	59.72	53.84	46.06	39.53
All Other Cancers	25.99	22.89	20.09	19.62	158.44	139.81	117.35	113.37
Cardio and Metabolic Diseases								
Endocrine, Nutrition, & Metabolic Diseases	3.11	4.43	4.49	5.16	17.86	20.87	19.65	23.04
Hypertensive Heart Disease	0.30	0.76	1.16	1.84	4.35	5.55	7.75	11.03
Ischemic & Other Circulatory Diseases	11.70	13.69	13.09	13.65	115.94	97.14	75.73	79.28
Substance Use and Mental Health								
Drug Poisonings	4.06	11.07	22.65	34.69	3.83	9.46	24.62	31.74
Alcohol-Induced*	1.76	2.05	2.49	3.88	5.27	5.74	8.97	12.47

continued

TABLE A-32 Continued

	Ages 25-44				Ages 45-64			
	1990-93	2000-02	2009-11	2015-17	1990-93	2000-02	2009-11	2015-17
Suicide	4.66	4.71	6.08	9.14	5.38	4.97	6.89	9.24
Mental & Behavioral Disorders	1.03	1.65	1.45	3.28	2.83	3.51	5.51	7.41
Other Body System Diseases & Disorders								
Nervous System	2.37	2.87	3.47	3.80	9.17	13.04	14.18	16.71
Genitourinary System	0.70	1.10	1.21	1.34	4.47	6.66	6.89	8.02
Respiratory System	3.16	4.02	4.51	4.64	33.39	36.68	37.00	42.63
Digestive System*	2.53	3.07	3.59	4.11	15.49	16.18	19.65	21.59
Other Causes of Death								
Homicides	3.27	2.68	2.33	2.51	2.18	1.57	1.56	1.66
Transport Injuries	9.72	10.20	7.90	8.44	8.54	8.70	6.85	7.64
Other External Causes	2.50	2.77	3.63	3.99	5.23	6.14	7.99	9.62
All Other Causes of Death	4.78	7.89	7.01	7.39	13.00	17.71	15.98	17.58

*Changes in the International Classification of Diseases (ICD)-10 code for diseases of the digestive system in 2006 affected the comparability of both digestive system deaths and alcohol-induced deaths before and after that year. More information is provided in Chapter 5.

NOTE: The table shows the all-cause and cause-specific mortality rates among non-Hispanic White working-age females in large fringe metropolitan areas by age group (25–44 and 45–64). These causes of death are exhaustive of all underlying cause-of-death codes and are based on the International Classification of Diseases (ICD)-9 (1990–1998) and ICD-10 (1999–2017) codes. Mortality rates within each age group are age-adjusted by single-year of age groups to match the age distribution of the U.S. population in 2000 in order to improve comparability over time. More information about the classification of causes of death can be found in Chapter 5.

SOURCE: Data from https://www.cdc.gov/nchs/nvss/nvss-restricted-data.htm.

TABLE A-33 Cause-Specific Mortality Rates (Deaths/100,000): Non-Hispanic White Females Ages 25-64 Living in Small/Medium Metropolitan Areas

	Ages 25-44				Ages 45-64			
	1990-93	2000-02	2009-11	2015-17	1990-93	2000-02	2009-11	2015-17
All-Cause Mortality	90.17	106.27	116.45	132.61	490.11	473.83	462.99	503.41
Cause-Specific Mortality								
Infectious & Parasitic Diseases								
HIV/AIDS	1.12	0.82	0.41	0.22	0.49	0.30	0.65	0.40
Non-HIV/AIDS	1.29	2.59	2.56	2.71	5.96	10.04	12.99	14.61
Cancers								
Liver	0.27	0.40	0.23	0.35	2.16	2.69	3.43	4.38
Lung	2.72	3.85	2.57	1.70	60.94	56.88	47.38	43.17
All Other Cancers	26.69	23.38	19.78	19.10	162.49	138.88	120.56	116.44
Cardio and Metabolic Diseases								
Endocrine, Nutrition, & Metabolic Diseases	3.69	4.37	5.22	5.84	19.03	22.33	22.80	25.78
Hypertensive Heart Disease	0.22	0.69	1.27	1.79	4.04	4.59	7.39	10.60
Ischemic & Other Circulatory Diseases	12.33	15.29	15.35	15.53	124.44	105.13	85.43	91.68
Substance Use and Mental Health								
Drug Poisonings	3.45	9.34	20.90	29.03	3.46	7.56	23.11	29.07
Alcohol-Induced*	1.45	2.14	2.41	3.81	4.38	5.31	8.16	12.11

continued

TABLE A-33 Continued

	Ages 25-44				Ages 45-64			
	1990-93	2000-02	2009-11	2015-17	1990-93	2000-02	2009-11	2015-17
Suicide	4.91	4.72	6.86	9.08	5.55	5.28	6.74	9.28
Mental & Behavioral Disorders	1.17	1.74	1.50	3.49	2.63	3.53	5.71	7.72
Other Body System Diseases & Disorders								
Nervous System	2.28	3.56	3.54	4.35	9.26	13.87	15.01	17.63
Genitourinary System	0.73	1.09	1.63	1.07	4.39	6.68	7.51	9.08
Respiratory System	3.12	3.61	4.84	5.20	34.82	38.06	40.92	48.31
Digestive System*	2.47	3.06	3.86	4.13	15.26	16.57	20.22	22.71
Other Causes of Death								
Homicides	3.50	2.44	2.51	2.43	2.28	1.54	1.64	2.00
Transport Injuries	11.54	12.15	9.09	10.18	9.95	10.38	8.20	9.60
Other External Causes	3.13	3.52	4.31	4.74	5.90	6.88	8.57	10.42
All Other Causes of Death	4.09	7.51	7.62	7.87	12.68	17.34	16.56	18.39

*Changes in the International Classification of Diseases (ICD)-10 code for diseases of the digestive system in 2006 affected the comparability of both digestive system deaths and alcohol-induced deaths before and after that year. More information is provided in Chapter 5.

NOTE: The table shows the all-cause and cause-specific mortality rates among non-Hispanic White working-age females in small/medium metropolitan areas by age group (25–44 and 45–64). These causes of death are exhaustive of all underlying cause-of-death codes and are based on the International Classification of Diseases (ICD)-9 (1990–1998) and ICD-10 (1999–2017) codes. Mortality rates within each age group are age-adjusted by single-year of age groups to match the age distribution of the U.S. population in 2000 in order to improve comparability over time. More information about the classification of causes of death can be found in Chapter 5.

SOURCE: Data from https://www.cdc.gov/nchs/nvss/nvss-restricted-data.htm.

TABLE A-34 Cause-Specific Mortality Rates (Deaths/100,000): Non-Hispanic White Females Ages 25-64 Living in Nonmetropolitan Areas

	Ages 25-44				Ages 45-64			
	1990-93	2000-02	2009-11	2015-17	1990-93	2000-02	2009-11	2015-17
All-Cause Mortality	94.04	117.75	136.50	156.66	485.17	495.14	503.68	557.48
Cause-Specific Mortality								
Infectious & Parasitic Diseases								
HIV/AIDS	0.71	0.64	0.39	0.28	0.31	0.31	0.43	0.44
Non-HIV/AIDS	1.26	2.49	2.96	3.83	5.65	10.32	14.41	16.24
Cancers								
Liver	0.32	0.36	0.42	0.42	2.58	2.68	3.63	4.90
Lung	3.02	4.32	3.32	2.31	56.92	57.26	53.46	51.17
All Other Cancers	27.36	24.48	22.62	21.70	156.03	142.35	124.78	122.62
Cardio and Metabolic Diseases								
Endocrine, Nutrition, & Metabolic Diseases	3.41	4.69	6.00	7.92	19.26	24.38	24.01	29.18
Hypertensive Heart Disease	0.28	0.84	1.30	2.27	3.19	4.47	6.87	11.26
Ischemic & Other Circulatory Diseases	14.07	17.76	18.32	20.84	131.94	116.68	99.54	108.28
Substance Use and Mental Health								
Drug Poisonings	2.72	8.96	23.41	29.94	2.62	7.39	22.36	29.54
Alcohol-Induced*	1.16	1.70	2.14	3.25	3.93	4.22	7.52	10.52

continued

TABLE A-34 Continued

	Ages 25-44				Ages 45-64			
	1990-93	2000-02	2009-11	2015-17	1990-93	2000-02	2009-11	2015-17
Suicide	4.75	5.21	6.70	8.81	4.85	4.49	6.60	9.11
Mental & Behavioral Disorders	0.86	1.65	1.41	3.25	2.16	3.18	4.99	6.93
Other Body System Diseases & Disorders								
Nervous System	2.64	3.29	4.41	4.93	9.07	12.62	15.40	18.26
Genitourinary System	0.78	1.36	1.62	1.96	4.88	7.35	8.27	10.01
Respiratory System	3.01	4.70	6.34	6.42	34.17	39.52	48.74	57.58
Digestive System*	2.32	3.16	3.98	4.91	14.60	17.06	20.27	25.70
Other Causes of Death								
Homicides	3.53	3.31	2.87	3.26	2.33	1.94	1.82	2.17
Transport Injuries	14.32	17.05	14.38	15.49	11.93	13.82	11.53	12.28
Other External Causes	3.54	4.56	5.09	5.35	6.21	7.34	10.02	11.26
All Other Causes of Death	3.98	7.19	8.79	9.52	12.54	17.75	19.02	20.04

*Changes in the International Classification of Diseases (ICD)-10 code for diseases of the digestive system in 2006 affected the comparability of both digestive system deaths and alcohol-induced deaths before and after that year. More information is provided in Chapter 5.

NOTE: The table shows the all-cause and cause-specific mortality rates among non-Hispanic White working-age females in nonmetropolitan areas by age group (25–44 and 45–64). These causes of death are exhaustive of all underlying cause-of-death codes and are based on the International Classification of Diseases (ICD)-9 (1990–1998) and ICD-10 (1999–2017) codes. Mortality rates within each age group are age-adjusted by single-year of age groups to match the age distribution of the U.S. population in 2000 in order to improve comparability over time. More information about the classification of causes of death can be found in Chapter 5.

SOURCE: Data from https://www.cdc.gov/nchs/nvss/nvss-restricted-data.htm.

TABLE A-35 Cause-Specific Mortality Rates (Deaths/100,000): Non-Hispanic Black Females Ages 25-64 Living in Large Central Metropolitan Areas

	Ages 25-44				Ages 45-64			
	1990-93	2000-02	2009-11	2015-17	1990-93	2000-02	2009-11	2015-17
All-Cause Mortality	252.59	209.52	152.18	150.31	919.69	833.20	668.61	640.65
Cause-Specific Mortality								
Infectious & Parasitic Diseases								
HIV/AIDS	39.64	24.37	8.47	4.27	15.65	20.69	13.70	9.04
Non-HIV/AIDS	9.32	12.14	7.72	5.24	21.89	31.61	28.03	23.68
Cancers								
Liver	0.59	0.63	0.52	0.58	4.10	5.05	6.87	7.21
Lung	4.81	4.33	1.87	1.27	70.98	58.58	47.20	36.37
All Other Cancers	40.86	33.70	29.15	26.60	235.40	199.30	168.50	157.62
Cardio and Metabolic Diseases								
Endocrine, Nutrition, & Metabolic Diseases	8.05	8.33	8.17	9.04	53.79	52.51	40.31	42.57
Hypertensive Heart Disease	6.04	6.90	7.50	7.17	45.35	40.70	38.34	38.36
Ischemic & Other Circulatory Diseases	39.76	34.66	24.61	23.87	283.58	231.55	154.48	144.00
Substance Use and Mental Health								
Drug Poisonings	8.25	10.75	9.18	14.93	3.69	10.07	15.73	24.85
Alcohol-Induced*	5.74	1.70	1.14	1.73	11.97	7.25	5.71	7.00

continued

TABLE A-35 Continued

	Ages 25-44				Ages 45-64			
	1990-93	2000-02	2009-11	2015-17	1990-93	2000-02	2009-11	2015-17
Suicide	2.65	1.93	2.41	2.91	2.00	1.74	1.43	1.74
Mental & Behavioral Disorders	5.68	3.55	1.51	1.89	7.94	7.04	6.11	6.50
Other Body System Diseases & Disorders								
Nervous System	4.16	4.37	4.61	5.31	11.53	15.93	15.82	17.81
Genitourinary System	4.16	4.18	3.30	3.46	22.01	24.17	19.92	19.68
Respiratory System	12.36	8.46	6.99	5.77	45.67	41.71	37.28	40.48
Digestive System*	8.37	5.01	3.75	3.63	30.35	24.80	21.10	19.52
Other Causes of Death								
Homicides	20.29	10.64	6.73	7.41	6.91	4.06	3.42	3.32
Transport Injuries	8.40	8.31	5.78	6.79	8.10	7.58	5.50	6.21
Other External Causes	4.87	3.67	3.29	3.31	9.26	8.36	7.98	8.03
All Other Causes of Death	18.59	21.89	15.47	15.15	29.50	40.48	31.17	26.67

*Changes in the International Classification of Diseases (ICD)-10 code for diseases of the digestive system in 2006 affected the comparability of both digestive system deaths and alcohol-induced deaths before and after that year. More information is provided in Chapter 5.

NOTE: The table shows the all-cause and cause-specific mortality rates among non-Hispanic Black working-age females in large central metropolitan areas by age group (25–44 and 45–64). These causes of death are exhaustive of all underlying cause-of-death codes and are based on the International Classification of Diseases (ICD)-9 (1990–1998) and ICD-10 (1999–2017) codes. Mortality rates within each age group are age-adjusted by single-year of age groups to match the age distribution of the U.S. population in 2000 in order to improve comparability over time. More information about the classification of causes of death can be found in Chapter 5.

SOURCE: Data from https://www.cdc.gov/nchs/nvss/nvss-restricted-data.htm..

TABLE A-36 Cause-Specific Mortality Rates (Deaths/100,000): Non-Hispanic Black Females Ages 25-64 Living in Large Fringe Metropolitan Areas

	Ages 25-44				Ages 45-64			
	1990-93	2000-02	2009-11	2015-17	1990-93	2000-02	2009-11	2015-17
All-Cause Mortality	218.15	205.00	169.97	180.52	889.80	852.66	708.02	701.81
Cause-Specific Mortality								
Infectious & Parasitic Diseases								
HIV/AIDS	21.39	16.24	7.25	4.44	6.97	10.71	11.01	9.80
Non-HIV/AIDS	7.91	12.46	8.92	5.91	17.84	31.49	29.88	26.51
Cancers								
Liver	0.60	0.68	0.70	0.44	2.97	4.80	6.98	7.93
Lung	4.06	4.24	1.81	0.91	61.72	57.09	48.77	38.33
All Other Cancers	38.86	35.19	28.23	27.61	215.40	195.10	163.90	152.49
Cardio and Metabolic Diseases								
Endocrine, Nutrition, & Metabolic Diseases	8.16	9.13	10.50	14.67	64.67	67.55	49.93	52.55
Hypertensive Heart Disease	4.81	5.86	7.15	8.22	32.88	33.58	31.59	35.59
Ischemic & Other Circulatory Diseases	40.98	39.28	33.42	33.05	301.26	249.12	174.82	171.12
Substance Use and Mental Health								
Drug Poisonings	4.68	7.99	8.47	15.05	2.37	7.16	13.69	17.66
Alcohol-Induced*	5.18	2.13	1.45	1.90	13.05	7.73	7.35	8.55

continued

TABLE A-36 Continued

	Ages 25-44				Ages 45-64			
	1990-93	2000-02	2009-11	2015-17	1990-93	2000-02	2009-11	2015-17
Suicide	2.25	1.85	1.67	2.93	1.73	1.36	1.28	1.78
Mental & Behavioral Disorders	4.16	2.38	0.79	1.65	8.47	5.16	5.47	6.48
Other Body System Diseases & Disorders								
Nervous System	4.37	5.51	5.89	7.16	11.30	18.33	19.39	21.48
Genitourinary System	3.28	4.40	5.40	4.71	18.90	28.09	26.87	25.33
Respiratory System	8.51	7.61	7.10	7.26	44.19	43.72	39.24	45.96
Digestive System*	7.29	5.59	4.45	4.68	28.37	28.03	23.61	23.29
Other Causes of Death								
Homicides	19.26	9.89	7.92	8.39	7.50	3.96	3.79	4.27
Transport Injuries	10.24	9.24	8.15	9.20	10.27	10.78	6.89	9.69
Other External Causes	5.04	4.05	3.28	4.35	8.85	10.29	9.48	9.44
All Other Causes of Death	17.12	21.27	17.42	18.00	31.11	38.61	34.10	33.53

*Changes in the International Classification of Diseases (ICD)-10 code for diseases of the digestive system in 2006 affected the comparability of both digestive system deaths and alcohol-induced deaths before and after that year. More information is provided in Chapter 5.

NOTE: The table shows the all-cause and cause-specific mortality rates among non-Hispanic Black working-age females in large fringe metropolitan areas by age group (25–44 and 45–64). These causes of death are exhaustive of all underlying cause-of-death codes and are based on the International Classification of Diseases (ICD)-9 (1990–1998) and ICD-10 (1999–2017) codes. Mortality rates within each age group are age-adjusted by single-year of age groups to match the age distribution of the U.S. population in 2000 in order to improve comparability over time. More information about the classification of causes of death can be found in Chapter 5.

SOURCE: Data from https://www.cdc.gov/nchs/nvss/nvss-restricted-data.htm.

TABLE A-37 Cause-Specific Mortality Rates (Deaths/100,000): Non-Hispanic Black Females Ages 25-64 Living in Small/Medium Metropolitan Areas

	Ages 25-44				Ages 45-64			
	1990-93	2000-02	2009-11	2015-17	1990-93	2000-02	2009-11	2015-17
All-Cause Mortality	217.28	211.02	183.95	184.40	948.43	874.21	743.91	762.33
Cause-Specific Mortality								
Infectious & Parasitic Diseases								
HIV/AIDS	11.73	14.17	7.01	4.70	5.69	9.09	7.77	7.36
Non-HIV/AIDS	8.04	10.57	9.18	5.73	21.74	30.21	29.65	33.05
Cancers								
Liver	0.49	0.57	0.51	0.48	3.71	5.00	5.39	5.90
Lung	4.72	3.91	2.30	1.20	66.07	55.59	47.56	39.12
All Other Cancers	40.52	37.26	28.60	29.42	237.09	194.15	171.68	165.56
Cardio and Metabolic Diseases								
Endocrine, Nutrition, & Metabolic Diseases	8.88	10.78	12.49	17.33	60.47	71.83	54.03	56.82
Hypertensive Heart Disease	4.26	4.65	7.43	8.47	39.47	32.62	34.27	34.99
Ischemic & Other Circulatory Diseases	48.93	44.68	38.09	32.25	324.87	272.19	196.43	196.95
Substance Use and Mental Health								
Drug Poisonings	3.16	5.85	7.62	13.82	1.27	4.44	9.78	15.28
Alcohol-Induced*	4.78	3.09	1.29	2.17	10.92	7.07	7.43	6.86

continued

TABLE A-37 Continued

	Ages 25-44				Ages 45-64			
	1990-93	2000-02	2009-11	2015-17	1990-93	2000-02	2009-11	2015-17
Suicide	1.53	1.86	1.35	2.37	1.87	0.99	1.03	0.88
Mental & Behavioral Disorders	3.66	2.37	1.92	1.73	9.92	8.05	5.45	6.42
Other Body System Diseases & Disorders								
Nervous System	4.84	5.15	6.88	7.29	13.46	13.74	18.28	23.69
Genitourinary System	3.51	4.86	6.31	4.96	20.46	30.94	29.24	29.21
Respiratory System	10.82	8.17	7.41	8.85	39.68	40.86	43.70	52.69
Digestive System*	7.11	5.87	5.79	5.03	30.19	27.47	25.36	26.47
Other Causes of Death								
Homicides	16.84	10.52	6.10	6.70	5.81	3.31	3.29	4.22
Transport Injuries	11.18	12.91	8.96	8.20	10.08	12.17	8.87	9.32
Other External Causes	6.76	4.54	4.55	3.65	14.40	12.24	10.72	9.73
All Other Causes of Death	15.53	19.24	20.15	20.06	31.27	42.26	34.00	37.81

*Changes in the International Classification of Diseases (ICD)-10 code for diseases of the digestive system in 2006 affected the comparability of both digestive system deaths and alcohol-induced deaths before and after that year. More information is provided in Chapter 5.

NOTE: The table shows the all-cause and cause-specific mortality rates among non-Hispanic Black working-age females in small/medium metropolitan areas by age group (25–44 and 45–64). These causes of death are exhaustive of all underlying cause-of-death codes and are based on the International Classification of Diseases (ICD)-9 (1990–1998) and ICD-10 (1999–2017) codes. Mortality rates within each age group are age-adjusted by single-year of age groups to match the age distribution of the U.S. population in 2000 in order to improve comparability over time. More information about the classification of causes of death can be found in Chapter 5.

SOURCE: Data from https://www.cdc.gov/nchs/nvss/nvss-restricted-data.htm.

TABLE A-38 Cause-Specific Mortality Rates (Deaths/100,000): Non-Hispanic Black Females Ages 25-64 Living in Nonmetropolitan Areas

	Ages 25-44				Ages 45-64			
	1990-93	2000-02	2009-11	2015-17	1990-93	2000-02	2009-11	2015-17
All-Cause Mortality	231.14	239.93	213.17	217.15	951.95	915.63	783.66	805.23
Cause-Specific Mortality								
Infectious & Parasitic Diseases								
HIV/AIDS	10.68	14.39	9.74	5.52	3.92	7.83	9.85	8.56
Non-HIV/AIDS	6.49	12.12	11.24	9.44	17.63	25.04	30.39	29.28
Cancers								
Liver	0.60	0.65	0.64	0.38	4.75	4.65	5.48	7.59
Lung	3.10	4.01	1.55	1.14	49.89	52.01	45.84	37.48
All Other Cancers	44.45	41.53	35.40	28.03	214.76	209.98	177.46	172.96
Cardio and Metabolic Diseases								
Endocrine, Nutrition, & Metabolic Diseases	9.67	11.73	15.76	18.90	72.21	71.91	65.39	68.63
Hypertensive Heart Disease	4.53	7.01	8.82	7.98	34.84	31.42	33.41	37.78
Ischemic & Other Circulatory Diseases	54.54	50.62	43.44	47.43	361.26	311.70	217.62	215.50
Substance Use and Mental Health								
Drug Poisonings	1.91	3.72	6.79	8.67	1.12	2.28	6.41	11.27
Alcohol-Induced*	4.90	2.49	1.23	1.31	9.90	8.91	6.50	6.64

continued

TABLE A-38 Continued

	Ages 25-44				Ages 45-64			
	1990-93	2000-02	2009-11	2015-17	1990-93	2000-02	2009-11	2015-17
Suicide	1.70	1.83	1.62	2.47	1.66	0.98	1.19	1.35
Mental & Behavioral Disorders	4.31	2.44	1.42	1.18	6.60	4.61	5.49	5.81
Other Body System Diseases & Disorders								
Nervous System	4.85	5.94	6.03	7.66	12.48	16.47	19.09	24.38
Genitourinary System	4.02	5.13	6.34	7.69	24.78	32.11	30.64	30.86
Respiratory System	9.55	10.70	9.51	9.20	41.08	39.08	41.47	54.55
Digestive System*	6.85	6.95	6.40	5.43	27.14	25.92	22.34	26.01
Other Causes of Death								
Homicides	17.55	10.62	7.62	8.92	6.78	4.67	4.51	3.65
Transport Injuries	18.21	18.89	13.46	19.05	16.09	15.36	13.86	12.80
Other External Causes	7.07	6.54	5.22	5.57	13.21	13.37	12.65	11.08
All Other Causes of Death	16.16	22.62	20.92	21.19	31.85	37.33	34.06	39.06

*Changes in the International Classification of Diseases (ICD)-10 code for diseases of the digestive system in 2006 affected the comparability of both digestive system deaths and alcohol-induced deaths before and after that year. More information is provided in Chapter 5.

NOTE: The table shows the all-cause and cause-specific mortality rates among non-Hispanic Black working-age females in nonmetropolitan areas by age group (25–44 and 45–64). These causes of death are exhaustive of all underlying cause-of-death codes and are based on the International Classification of Diseases (ICD)-9 (1990–1998) and ICD-10 (1999–2017) codes. Mortality rates within each age group are age-adjusted by single-year of age groups to match the age distribution of the U.S. population in 2000 in order to improve comparability over time. More information about the classification of causes of death can be found in Chapter 5.

SOURCE: Data from https://www.cdc.gov/nchs/nvss/nvss-restricted-data.htm.

TABLE A-39 Cause-Specific Mortality Rates (Deaths/100,000): Hispanic Females Ages 25-64 Living in Large Central Metropolitan Areas

	Ages 25-44				Ages 45-64			
	1990-93	2000-02	2009-11	2015-17	1990-93	2000-02	2009-11	2015-17
All-Cause Mortality	90.29	71.96	56.99	60.57	368.11	336.42	276.55	266.90
Cause-Specific Mortality								
Infectious & Parasitic Diseases								
HIV/AIDS	14.51	4.70	1.05	0.48	8.19	5.66	2.76	1.49
Non-HIV/AIDS	2.90	2.79	1.88	1.22	8.36	12.08	12.35	9.05
Cancers								
Liver	0.25	0.34	0.27	0.38	3.36	4.40	4.77	5.25
Lung	1.06	1.01	0.80	0.57	14.62	13.65	11.34	10.20
All Other Cancers	20.51	19.35	15.99	16.80	117.23	106.74	90.67	87.30
Cardio and Metabolic Diseases								
Endocrine, Nutrition, & Metabolic Diseases	2.21	2.29	2.47	2.39	22.72	26.39	18.19	18.86
Hypertensive Heart Disease	0.92	0.88	0.95	1.01	8.00	7.87	7.44	8.27
Ischemic & Other Circulatory Diseases	9.72	8.28	6.35	6.48	97.02	74.03	50.63	45.44
Substance Use and Mental Health								
Drug Poisonings	2.92	3.80	4.74	6.67	2.49	3.47	6.55	7.90
Alcohol-Induced*	2.07	1.36	1.22	1.95	7.60	5.27	6.45	7.32

continued

TABLE A-39 Continued

	Ages 25-44				Ages 45-64			
	1990-93	2000-02	2009-11	2015-17	1990-93	2000-02	2009-11	2015-17
Suicide	2.20	1.58	1.72	2.43	2.09	1.62	1.55	2.03
Mental & Behavioral Disorders	1.33	1.19	0.67	0.80	1.88	2.13	2.39	2.40
Other Body System Diseases & Disorders								
Nervous System	1.45	1.51	1.44	1.60	4.79	5.95	6.18	7.62
Genitourinary System	0.99	0.93	0.82	0.94	6.91	7.63	7.05	6.86
Respiratory System	3.60	2.36	2.29	1.78	17.93	15.94	14.33	14.24
Digestive System*	2.31	2.14	1.70	1.75	17.47	15.12	14.42	13.26
Other Causes of Death								
Homicides	6.70	3.15	2.39	2.29	3.58	1.91	1.30	1.21
Transport Injuries	6.93	6.01	4.02	4.50	8.64	8.22	4.43	4.87
Other External Causes	1.71	1.26	1.34	1.52	3.30	3.66	3.88	3.65
All Other Causes of Death	5.98	7.03	4.87	5.00	11.93	14.66	9.89	9.69

*Changes in the International Classification of Diseases (ICD)-10 code for diseases of the digestive system in 2006 affected the comparability of both digestive system deaths and alcohol-induced deaths before and after that year. More information is provided in Chapter 5.

NOTE: The table shows the all-cause and cause-specific mortality rates among Hispanic working-age females in large central metropolitan areas by age group (25–44 and 45–64). These causes of death are exhaustive of all underlying cause-of-death codes and are based on the International Classification of Diseases (ICD)-9 (1990–1998) and ICD-10 (1999–2017) codes. Mortality rates within each age group are age-adjusted by single-year of age groups to match the age distribution of the U.S. population in 2000 in order to improve comparability over time. More information about the classification of causes of death can be found in Chapter 5.

SOURCE: Data from https://www.cdc.gov/nchs/nvss/nvss-restricted-data.htm.

TABLE A-40 Cause-Specific Mortality Rates (Deaths/100,000): Hispanic Females Ages 25-64 Living in Large Fringe Metropolitan Areas

	Ages 25-44				Ages 45-64			
	1990-93	2000-02	2009-11	2015-17	1990-93	2000-02	2009-11	2015-17
All-Cause Mortality	84.27	79.31	66.67	77.35	402.60	378.29	325.52	327.26
Cause-Specific Mortality								
Infectious & Parasitic Diseases								
HIV/AIDS	6.03	2.60	0.85	0.43	1.65	3.23	1.39	1.04
Non-HIV/AIDS	1.77	2.66	2.18	2.19	9.08	14.45	15.83	13.86
Cancers								
Liver	0.31	0.40	0.30	0.42	4.51	3.66	4.37	5.63
Lung	0.97	0.93	0.75	0.92	16.26	12.07	10.86	9.58
All Other Cancers	22.43	19.31	15.82	16.97	122.30	110.41	96.54	94.24
Cardio and Metabolic Diseases								
Endocrine, Nutrition, & Metabolic Diseases	2.79	3.33	3.36	3.70	36.11	34.91	22.79	25.98
Hypertensive Heart Disease	0.55	0.61	0.87	1.05	5.86	7.05	6.65	8.00
Ischemic & Other Circulatory Diseases	11.21	10.30	7.16	8.47	108.58	88.62	62.90	56.56
Substance Use and Mental Health								
Drug Poisonings	2.77	4.21	7.03	9.14	1.34	4.81	9.05	11.70
Alcohol-Induced*	2.08	1.62	1.49	2.44	6.61	5.91	6.80	7.25

continued

TABLE A-40 Continued

	Ages 25-44				Ages 45-64			
	1990-93	2000-02	2009-11	2015-17	1990-93	2000-02	2009-11	2015-17
Suicide	2.25	1.53	1.59	2.84	1.08	1.45	2.43	2.58
Mental & Behavioral Disorders	0.89	1.63	0.56	0.93	2.58	2.67	2.48	3.40
Other Body System Diseases & Disorders								
Nervous System	1.80	1.30	2.08	2.34	5.42	6.76	7.80	10.00
Genitourinary System	0.90	1.09	1.24	1.02	8.58	10.79	9.58	9.30
Respiratory System	2.86	2.42	3.29	2.23	17.33	17.40	17.39	18.05
Digestive System*	2.57	2.61	2.58	3.00	23.32	20.09	21.48	20.86
Other Causes of Death								
Homicides	4.51	3.59	2.32	2.73	2.66	2.25	1.64	2.07
Transport Injuries	10.70	9.55	5.88	7.66	11.07	10.79	7.14	7.59
Other External Causes	1.74	2.43	1.81	1.92	3.84	3.83	4.45	4.87
All Other Causes of Death	5.15	7.18	5.51	6.96	14.42	17.16	13.96	14.71

*Changes in the International Classification of Diseases (ICD)-10 code for diseases of the digestive system in 2006 affected the comparability of both digestive system deaths and alcohol-induced deaths before and after that year. More information is provided in Chapter 5.

NOTE: The table shows the all-cause and cause-specific mortality rates among Hispanic working-age females in large fringe metropolitan areas by age group (25–44 and 45–64). These causes of death are exhaustive of all underlying cause-of-death codes and are based on the International Classification of Diseases (ICD)-9 (1990–1998) and ICD-10 (1999–2017) codes. Mortality rates within each age group are age-adjusted by single-year of age groups to match the age distribution of the U.S. population in 2000 in order to improve comparability over time. More information about the classification of causes of death can be found in Chapter 5.

SOURCE: Data from https://www.cdc.gov/nchs/nvss/nvss-restricted-data.htm.

TABLE A-41 Cause-Specific Mortality Rates (Deaths/100,000): Hispanic Females Ages 25-64 Living in Small/ Medium Metropolitan Areas

	Ages 25-44				Ages 45-64			
	1990-93	2000-02	2009-11	2015-17	1990-93	2000-02	2009-11	2015-17
All-Cause Mortality	86.95	74.88	67.59	77.73	416.57	360.21	324.47	309.09
Cause-Specific Mortality								
Infectious & Parasitic Diseases								
HIV/AIDS	3.28	1.86	0.78	0.31	1.07	1.35	1.37	0.48
Non-HIV/AIDS	1.44	2.49	2.31	1.76	6.37	11.45	14.37	13.89
Cancers								
Liver	0.39	0.25	0.10	0.31	3.11	6.36	5.19	4.60
Lung	0.94	0.55	1.18	1.46	17.87	16.75	10.28	11.48
All Other Cancers	22.82	16.89	13.88	17.18	123.42	104.18	87.21	82.49
Cardio and Metabolic Diseases								
Endocrine, Nutrition, & Metabolic Diseases	3.26	4.18	2.91	4.05	39.85	34.41	25.03	24.60
Hypertensive Heart Disease	0.22	0.53	0.58	0.78	5.58	5.17	6.89	6.31
Ischemic & Other Circulatory Diseases	8.56	8.67	7.27	6.97	111.60	79.19	66.42	53.50
Substance Use and Mental Health								
Drug Poisonings	3.63	5.95	7.92	10.85	1.43	2.75	9.11	9.58
Alcohol-Induced*	2.39	1.24	1.72	2.51	6.19	6.23	8.13	8.50

continued

TABLE A-41 Continued

	Ages 25-44				Ages 45-64			
	1990-93	2000-02	2009-11	2015-17	1990-93	2000-02	2009-11	2015-17
Suicide	2.62	1.41	1.66	3.93	1.82	0.80	1.65	3.65
Mental & Behavioral Disorders	0.30	0.60	0.54	0.71	4.25	2.33	2.79	4.34
Other Body System Diseases & Disorders								
Nervous System	2.25	1.45	2.11	1.89	5.51	8.70	8.50	9.07
Genitourinary System	0.65	0.99	1.34	1.18	10.51	8.51	10.71	9.48
Respiratory System	4.02	2.30	2.99	2.25	17.23	13.88	17.57	18.33
Digestive System*	3.38	2.44	3.07	2.63	25.91	19.21	22.66	19.76
Other Causes of Death								
Homicides	5.08	3.02	1.62	2.75	2.54	3.81	0.59	1.72
Transport Injuries	15.25	11.52	7.51	7.35	12.25	12.62	6.08	8.90
Other External Causes	2.71	3.25	2.49	2.63	5.14	5.72	5.35	4.90
All Other Causes of Death	3.76	5.28	5.62	6.23	14.94	16.80	14.58	13.52

*Changes in the International Classification of Diseases (ICD)-10 code for diseases of the digestive system in 2006 affected the comparability of both digestive system deaths and alcohol-induced deaths before and after that year. More information is provided in Chapter 5.

NOTE: The table shows the all-cause and cause-specific mortality rates among Hispanic working-age females in small/medium metropolitan areas by age group (25–44 and 45–64). These causes of death are exhaustive of all underlying cause-of-death codes and are based on the International Classification of Diseases (ICD)-9 (1990–1998) and ICD-10 (1999–2017) codes. Mortality rates within each age group are age-adjusted by single-year of age groups to match the age distribution of the U.S. population in 2000 in order to improve comparability over time. More information about the classification of causes of death can be found in Chapter 5.

SOURCE: Data from https://www.cdc.gov/nchs/nvss/nvss-restricted-data.htm.

TABLE A-42 Cause-Specific Mortality Rates (Deaths/100,000): Hispanic Females Ages 25-64 Living in Nonmetropolitan Areas

	Ages 25-44				Ages 45-64			
	1990-93	2000-02	2009-11	2015-17	1990-93	2000-02	2009-11	2015-17
All-Cause Mortality	92.35	81.31	79.70	89.30	450.12	399.44	355.23	364.25
Cause-Specific Mortality								
Infectious & Parasitic Diseases								
HIV/AIDS	1.50	0.99	0.89	0.54	0.73	0.98	0.37	0.94
Non-HIV/AIDS	1.44	3.41	2.65	2.06	10.52	11.87	17.40	14.51
Cancers								
Liver	0.41	0.30	0.06	0.55	3.30	5.40	4.73	5.45
Lung	0.71	1.12	0.85	1.01	19.55	16.56	15.50	12.40
All Other Cancers	23.89	19.98	16.38	16.61	136.69	107.84	93.04	88.98
Cardio and Metabolic Diseases								
Endocrine, Nutrition, & Metabolic Diseases	4.18	2.47	4.64	4.90	47.78	41.87	26.47	31.46
Hypertensive Heart Disease	0.25	0.46	0.64	0.84	4.86	4.98	5.87	7.05
Ischemic & Other Circulatory Diseases	11.47	9.92	8.02	9.40	120.74	98.39	70.64	72.99
Substance Use and Mental Health								
Drug Poisonings	2.55	5.41	7.72	10.46	1.97	4.83	9.81	9.48
Alcohol-Induced*	1.02	1.65	1.87	3.30	3.43	4.08	7.03	8.91

continued

TABLE A-42 Continued

	Ages 25-44				Ages 45-64			
	1990-93	2000-02	2009-11	2015-17	1990-93	2000-02	2009-11	2015-17
Suicide	2.36	0.72	2.17	3.98	1.22	1.77	1.76	2.40
Mental & Behavioral Disorders	1.49	0.84	0.34	0.98	2.09	2.15	3.14	4.65
Other Body System Diseases & Disorders								
Nervous System	2.61	1.45	2.72	2.24	5.07	6.78	10.02	10.61
Genitourinary System	1.21	1.94	1.04	1.23	9.23	11.90	12.37	11.80
Respiratory System	2.91	1.27	3.44	2.72	19.80	16.32	16.89	19.89
Digestive System*	2.90	2.00	2.54	3.53	25.71	21.34	24.39	26.75
Other Causes of Death								
Homicides	7.18	3.68	2.64	2.96	2.59	4.08	1.51	1.94
Transport Injuries	16.18	15.72	11.98	12.41	14.16	15.21	13.45	11.31
Other External Causes	2.44	2.73	3.18	2.50	5.44	7.52	5.65	6.28
All Other Causes of Death	5.64	5.26	5.93	7.07	15.23	15.59	15.17	16.45

*Changes in the International Classification of Diseases (ICD)-10 code for diseases of the digestive system in 2006 affected the comparability of both digestive system deaths and alcohol-induced deaths before and after that year. More information is provided in Chapter 5.

NOTE: The table shows the all-cause and cause-specific mortality rates among Hispanic working-age females in nonmetropolitan areas by age group (25–44 and 45–64). These causes of death are exhaustive of all underlying cause-of-death codes and are based on the International Classification of Diseases (ICD)-9 (1990–1998) and ICD-10 (1999–2017) codes. Mortality rates within each age group are age-adjusted by single-year of age groups to match the age distribution of the U.S. population in 2000 in order to improve comparability over time. More information about the classification of causes of death can be found in Chapter 5.

SOURCE: Data from https://www.cdc.gov/nchs/nvss/nvss-restricted-data.htm.

Appendix B

Meeting Agendas

Committee on Rising Midlife Mortality Rates and Socioeconomic Disparities
Meeting #1
February 11-12, 2019

Keck Center, E Street Conference Room
500 Fifth Street, NW
Washington, DC 20001

DAY 1 – MONDAY, FEBRUARY 11, 2019

9:00 – 9:30 am **Welcome, Introductions, Overview of Agenda**

Mary Ellen O'Connell, Executive Director, Division of Behavioral and Social Sciences and Education (DBASSE)

Kathleen Mullan Harris (Committee Chair), University of North Carolina at Chapel Hill

Malay Majmundar, Study Director

9:30 – 10:00 am	**Overview of the National Academies Study Process – What Lies Ahead** *Mary Ellen O'Connell*, Executive Director, DBASSE
10:00 – 11:00 am	**Sponsor Interests and Perspectives; Discussion of Statement of Task** *Amelia Karraker*, National Institute on Aging *Kerry Anne McGeary*, Senior Program Officer, Research-Evaluation-Learning, Robert Wood Johnson Foundation
11:00 – 11:15 am	BREAK
11:15 am– 12:00 pm	**Mid-Life Mortality: Overview of Trends and Differentials** *Ryan Masters*, University of Colorado Boulder
12:00 – 12:40 pm	LUNCH (**discussion continues**)
12:40 – 2:00 pm	**Behavioral Factors, Life-Course Perspectives, and the Role of SES and the Health System: Highlights from the June 2017 Planning Meeting**[1] *Kathleen Mullan Harris* (Committee Chair), University of North Carolina at Chapel Hill
2:00 – 5:15 pm	**CLOSED SESSION (committee and staff only)**
5:15 pm	Adjournment

[1]See http://sites.nationalacademies.org/DBASSE/CPOP/DBASSE_180012.

DAY 2 – TUESDAY, FEBRUARY 12, 2019

9:00 am – 3:00 pm	**CLOSED SESSION (committee and staff only)**
3:00 pm	Adjournment

Committee on Rising Midlife Mortality Rates and Socioeconomic Disparities
Meeting #2
April 30 – May 1, 2019

Keck Center, Room 206
500 Fifth Street, NW
Washington, DC 20001

DAY 1 – TUESDAY, APRIL 30

8:30 – 10:30 am	**CLOSED SESSION (committee and staff only)**
10:30 – 11:15 am	**Understanding the Role of Economic Changes and Macroeconomic Shocks** *Christopher Ruhm,* University of Virginia (15 minute presentation followed by Q&A)
11:15 am – 12: 00 pm	**Understanding Trends in Optimism/Wellness/ Despair** *Carol Graham,* Brookings Institution (15 minute presentation followed by Q&A)
12:00 – 12:45 pm	LUNCH
12:45 – 1:30 pm	**Understanding Family Structure and the Life Course** *Andrew Cherlin,* Johns Hopkins University (15 minute presentation followed by Q&A)

1:30 – 2:00 pm	**General Discussion**
2:00 – 2:15 pm	BREAK
2:15 – 5:00 pm	**CLOSED SESSION (committee and staff only)**
5:00 pm	Adjournment

DAY 2 – WEDNESDAY, MAY 1

9:00 am – 3:00 pm	**CLOSED SESSION (committee and staff only)**
3:00 pm	Adjournment

Committee on Rising Midlife Mortality Rates and Socioeconomic Disparities
Meeting #3
July 18-19, 2019

Keck Center, Room 208
500 Fifth Street, NW
Washington, DC 20001

DAY 1 – THURDAY, JULY 18

8:30 – 8:45 am	**Welcome and Overview of Agenda**
8:45 – 9:45 am	**Anne Case (Princeton University)** Will discuss updates to her research on midlife mortality
9:45 – 10:00 am	BREAK
10:00 – 11:00 am	**Erika Blacksher (University of Washington)** Will discuss concepts of "whiteness" and the potential relevance of race/racism as it relates to midlife mortality
11:00 – 11:30 am	**General Discussion**

11:30 am – 12:30 pm	LUNCH
12:30 – 5:00 pm	**CLOSED SESSION (committee and staff only)**
5:00 pm	Adjournment

DAY 2 – FRIDAY, JULY 19

9:00 am – 3:00 pm	**CLOSED SESSION (committee and staff only)**
3:00 pm	Adjournment

Committee on Rising Midlife Mortality Rates and Socioeconomic Disparities
Meeting #4
October 21-22, 2019

Keck Center, Room 101
500 Fifth Street, NW
Washington, DC 20001

DAY 1 – MONDAY, OCTOBER 21

8:30 – 8:45 am	**Welcome and Overview of Agenda**
8:45 – 9:45 am	**Jennifer Silva (Indiana University Bloomington)** Will discuss qualitative/ethnographic work relating to mid-life mortality, including on sentiments on "hopelessness" and "despair" and how they affect health
9:45 – 10:00 am	BREAK
10:00 – 11:00 am	**Kathleen Frydl** Will provide an overview of the role played by opioids and other drugs, as well as historical/political/regulatory context for supply- and demand-side forces

11:00 am – 12:00 pm	**General Discussion**
12:00 – 1:00 pm	LUNCH
1:00 – 5:30 pm	**CLOSED SESSION (committee and staff only)**
5:30 pm	Adjournment

DAY 2 – TUESDAY, OCTOBER 22

9:00 am – 3:30 pm	**CLOSED SESSION (committee and staff only)**
3:30 pm	Adjournment

Appendix C

Biographical Sketches

KATHLEEN MULLAN HARRIS (*Chair*) is James E. Haar distinguished professor of sociology, adjunct professor of public policy, and Carolina Population Center faculty fellow at the University of North Carolina at Chapel Hill. Her research focuses on social inequality and health with particular interests in health disparities, biodemography, sociogenomics, and life-course processes. Harris has served as director and principal investigator of the National Longitudinal Study of Adolescent to Adult Health (Add Health) since 2004. She developed the integrative design in Add Health that links social, behavioral, and biological sciences for the study of developmental and health trajectories across the early life course. Harris leads the research team of scholars from sociology, epidemiology, nutrition, economics, cardiology, genetics, and survey methodology in analysis of the multidisciplinary, multilevel Add Health data. Her work has been funded by the National Institutes of Health for the past 30 years, and she has published more than 150 articles in more than 80 different disciplinary journals. She was awarded the Golden Goose Award from the U.S. Congress in 2016 for major breakthroughs in medicine, social behavior, and technological research. Harris is past president of the Population Association of America and an elected member of the National Academy of Sciences and of the American Academy of Arts and Sciences. She holds a Ph.D. in demography from the University of Pennsylvania.

TARA BECKER (*Program Officer*) is a program officer for the Committees on National Statistics and Population in the Division of Behavioral and Social Sciences and Education at the National Academies of Sciences,

Engineering, and Medicine. In addition to this study, she serves as the study director for a study examining the older workforce and employment at older ages and another focused on developing guidelines for the collection of sex, gender identity, and sexual orientation data. Before joining the National Academies, she was a senior public administration analyst and senior statistician for the California Health Interview Survey at the Center for Health Policy Research at the University of California, Los Angeles, where she conducted research on disparities in health insurance coverage and access to health care, as well as on survey data quality and methodology. Prior to this, she was a postdoctoral fellow in the Department of Health Policy and Management at the University of California, Los Angeles and a biostatistician at the University of Wisconsin, Madison Department of Biostatistics and Medical Informatics. She has a B.A. in sociology and mathematics, an M.S. in sociology, an M.S. in statistics, and a Ph.D. in sociology from the University of Wisconsin, Madison.

MICHAEL E. CHERNEW is Leonard D. Schaeffer professor of health care policy and director of the Healthcare Markets and Regulation Lab in the Department of Health Care Policy at Harvard Medical School. His research interests are focused on innovations in payment reform and benefit design. Chernew is a member of the Congressional Budget Office's Panel of Health Advisors and a member of the Committee on National Statistics at the National Academies of Sciences, Engineering, and Medicine. He also serves as vice chair of the Massachusetts Health Connector Board of Directors, research associate of the National Bureau of Economic Research, coeditor of the *American Journal of Managed Care,* and editor of the *Journal of Health Economics*. In 2010, he was elected to the National Academy of Medicine. Chernew holds a Ph.D. in economics from Stanford University.

DAVID M. CUTLER is Otto Eckstein professor of applied economics in the Department of Economics at Harvard University. He examines how population health is changing over time; the importance of medical and nonmedical factors in improved health; and the value of increased medical spending, arguing that medical care is more productive than current statistics indicate and that the medical care cost problem is overstated. Cutler is also interested in the economics of health insurance and the effects of managed care on the medical system. In 2001, he was elected to the National Academy of Medicine, and he serves as a research associate at the National Bureau of Economic Research. Cutler has authored two books, several chapters in edited books, and many published papers on health care and other public policy topics. He holds a Ph.D. in economics from the Massachusetts Institute of Technology.

ANA V. DIEZ ROUX is dean and distinguished university professor of epidemiology in the Dornsife School of Public Health at Drexel University. Her research is focused on the social determinants of population health and the study of how neighborhoods, particularly urban neighborhoods, affect health; her work has been highly influential in the policy debate on population health and its determinants. With funding from the National Institutes of Health and foundations, Diez Roux has led large research and training programs in the United States and in collaboration with international partners. Most recently, she convened the Network for Urban Health in Latin America and the Caribbean, which focuses on promoting research, training, and policies to promote urban health throughout the region. Diez Roux was awarded the Wade Hampton Frost Award by the American Public Health Association for her contributions to public health. She is an elected member of the American Epidemiological Society, the Academy of Behavioral Medicine Research, and the National Academy of Medicine. Dr. Diez Roux holds an M.D. from the University of Buenos Aires, an M.P.H. from Johns Hopkins University, and a Ph.D. from the Johns Hopkins University School of Hygiene and Public Health.

IRMA T. ELO is chair of the Department of Sociology at the University of Pennsylvania and is research associate of the university's Population Aging Research Center and Population Studies Center. She has served as a member and/or chair of several national and international committees, including the Board of Scientific Counselors of the National Center for Health Statistics, the U.S. Census Bureau's Scientific Advisory Committee, the sociology section on population for the American Sociological Association, the Population Association of America's (PAA's) board of directors, PAA's Committee on Population Statistics, and the International Advisory Board of the Swedish Initiative for Research on Microdata in the Social and Medical Sciences. Elo's main research interests are focused on socioeconomic and racial/ethnic disparities in health and mortality across the life course and demographic estimation of mortality, including health and mortality among racial/ethnic immigrant subgroups. She is currently principal investigator of the study, funded by the National Institute on Aging, titled Causes of Geographic Divergence in American Mortality Between 1990 and 2015: Health Behaviors, Health Care Access and Migration. Elo holds a Ph.D. in public affairs and demography from Princeton University.

DARRELL J. GASKIN is William C. and Nancy F. Richardson professor in health policy and director of the Hopkins Center for Health Disparities Solutions at the Johns Hopkins Bloomberg School of Public Health. He is a health services researcher and health economist with a focus on health and health care disparities. Gaskin has published in the leading health services

and public health research journals, including the *American Journal of Public Health*, *Health Services Research*, *Health Affairs*, *Inquiry*, *Medical Care*, *Medical Care Research and Review*, and *Social Science & Medicine*. He is a member the National Advisory Committee of the Robert Wood Johnson Foundation's Systems for Action program and serves on the board of directors of the American Society of Health Economists. Gaskin has also served as chair of the board of directors of AcademyHealth and has served on the Board of Scientific Counselors of the National Center for Health Statistics. He is a 2019 recipient of the Presidential Early Career Award for Scientists and Engineers. Gaskin holds an M.A. in economics from the Massachusetts Institute of Technology and a Ph.D. in health economics from Johns Hopkins University.

ROBERT A. HUMMER is Howard W. Odum distinguished professor of sociology and fellow of the Carolina Population Center at the University of North Carolina at Chapel Hill. He is president of the Population Association of America (PAA) and director of Wave VI of the National Longitudinal Study of Adolescent to Adult Health. Hummer's research is focused on the accurate description and more complete understanding of population health and mortality patterns and trends in the United States. His most recent book is *Population Health in America* (with Erin R. Hamilton, published by the University of California Press, 2019). Hummer's work has been funded by the National Institute of Child Health and Human Development, the National Institute on Aging, and the National Science Foundation. In 2010, he was presented with the Clifford Clogg Award for Early Career Achievement by PAA and in 2019 was presented with the Mentoring Award by the Interdisciplinary Association for Population Health Science. He holds a Ph.D. in sociology from Florida State University.

MALAY K. MAJMUNDAR (*Study Director*) directs the Committee on Population (CPOP). In addition to serving as study director for this report, he is currently overseeing CPOP activities on family planning and women's empowerment, climate change and human health, forced migration and refugee movements, the workplace and aging, and sexual and gender diverse populations. He is also developing a future research portfolio for CPOP. While at the National Academies, he has worked on studies on demography, criminal justice, immigration enforcement and statistics, and the federal budget. He has a B.A. in political science from Duke University, a J.D. from Yale University, and a Ph.D. in public policy from the University of Chicago.

RYAN K. MASTERS is assistant professor of sociology and faculty associate of the Population Program and the Health and Society Program at the

Institute of Behavioral Science at the University of Colorado Boulder. His interests include the examination of long-term trends in U.S. morbidity; chronic diseases; and mortality rates, including the health consequences of the U.S. obesity epidemic, especially as it relates to premature mortality among the U.S. adult population. Masters has been involved in advancing and testing new methodological approaches to studying period-based factors, such as health-promoting policies and new medical technologies, and cohort-based factors, such as early-life disease exposure, related to adult health. He holds a Ph.D. in sociology and demography from the University of Texas at Austin.

SHANNON M. MONNAT is Lerner chair for public health promotion; associate professor of sociology; and codirector of the Policy, Place, and Population Health Lab in the Maxwell School of Citizenship and Public Affairs at Syracuse University. Her research is focused on trends and geographic differences in health and mortality, with a special interest in rural health. Monnat is a national expert on social, structural, and spatial determinants of opioid and other drug use and overdose. Her most recent research has focused on understanding why overdose rates are higher in some places than others. Monnat has consulted about causes and solutions for addressing the overdose crisis with several national and international organizations, including the National Institutes of Health (NIH), the U.S. Department of Agriculture (USDA), the U.S. Office of National Drug Control Policy, and the United Nations Office on Drugs and Crime. She has been the principal or coprincipal investigator on several large federal agency and foundation grants, including through NIH, USDA, the U.S. Department of Justice, the Robert Wood Johnson Foundation, and the Institute for New Economic Thinking. Monnat has published more than 70 peer-reviewed academic journal articles, book chapters, research briefs, and reports, and has presented her research to numerous public, academic, and policy audiences. She holds a Ph.D. in sociology from the State University of New York at Albany.

BHRAMAR MUKHERJEE is John D. Kalbfleisch collegiate professor and chair of biostatistics, professor of epidemiology, and professor of global public health at the University of Michigan (UM). She is also founding director of UM's Big Data Summer Institute, research professor at the Michigan Institute of Data Science, and associate director of cancer control and population sciences at the UM Rogel Cancer Center. Mukherjee's research interests include statistical methods for analysis of electronic health records, studies of gene–environment interaction, Bayesian methods, shrinkage estimation, and analysis of multiple pollutants. She has coauthored more than 260 publications in statistics, biostatistics, medicine, and public health, and

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