

Socioeconomic Status and Increasing Mid-life Mortality

Planning Meeting

June 16, 2017

Keck Center of the National Academies
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Meeting Summary

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Executive Summary

On June 16, 2017, the National Academies of Sciences, Engineering, and Medicine Committee on Population (CPOP) and the National Institute on Aging (NIA) of the National Institutes of Health (NIH) convened a group of experts to discuss evidence regarding the role of widening socioeconomic status (SES) differences in life expectancy, especially during middle age, and to identify gaps in the research needed both to understand and to reverse this trend. Attendees discussed these trends in relation to their own research and explored trends in mortality across time and by characteristics including birth cohort, age, gender, race/ethnicity, socioeconomic status, and geographic location. They were also asked to provide advice and recommendations to CPOP and NIA on ways that these organizations could address these disparities.

The first session, *Overview of Trends and Differentials*, featured speakers who presented information about mortality trends over the past few decades, with a focus on growing inequalities in mid-life. While Case and Deaton's recent high-profile work finds increases in deaths due to "despair" (i.e., drug use, alcoholism, and suicide), relatively little evidence was identified that supports this explanation. Nonetheless, growing use and abuse of legal and illegal opioids were, by themselves, found to be significant contributors to increasing mortality in mid-life. The recent flattening out of mortality associated with metabolic diseases—obesity, diabetes, hypertension, and cardiovascular disease—was also found to be an important contributor to why mid-life mortality is no longer decreasing. Significant disparities by educational attainment and income were documented, with less-educated and poorer people faring worse. Growing geographic variations at the state and county levels and by metropolitan status (urban, rural, micropolitan) were also explored. Discussion highlighted the importance of careful consideration of gender and the changing composition of socioeconomic groups over time as well as a continued focus on racial/ethnic inequalities and smoking as a behavioral risk factor. The explanations of differences in levels of mortality need not be the same as explanations of *changes in levels* of mortality.

The second session, *The Role of the Health System and SES in Access and Constraints to Health and Health Care*, explored the ways that the U.S. health care system both ameliorates and contributes to mid-life mortality. The U.S. health care system is generally better at treating than preventing disease, which may be due to structural aspects of how care is delivered. Government policies also play an important role in determining health outcomes.

The third session, *Life-course Perspectives on Mid-life Mortality*, discussed the factors in early- and mid-life that can influence the risk of morbidity and mortality. Today's young adults are the first birth cohort to have come of age during the obesity epidemic and have experienced higher levels of metabolic syndrome and cardiometabolic risk than their earlier-born counterparts. Because the health consequences of obesity, including increased mortality, are often latent for years or decades, the full implications of the effects of growing up in an obesogenic environment may not yet be fully realized and remain cause for concern. Compared to previous cohorts of young adults, today's adults are heavier and show higher incidence of metabolic diseases such as hypertension and diabetes. Long-term, longitudinal data on these populations

will be key to understanding the consequences of past and current experiences related to family, employment, and health for young adults as they enter middle age.

The fourth session, *Behavioral Factors*, explored the ways that social structures and behavioral norms influence health. Because humans are fundamentally social animals, much of our behavior occurs in a social context. The normative expectations for this behavior—and how certain behaviors are encouraged or discouraged—can have an important impact on health outcomes. The relationship between behavior and health may differ by birth cohort (i.e., when one was born) or by period (i.e., the historical time during which one experiences a certain environment). It is likely that both cohort and period effects shape trends in health and health behaviors and even the relative importance of genetics versus environment.

Successful approaches to reducing mid-life mortality will need to consider not only biologic factors such as genetics but also environmental and social factors. The challenge is to use our understanding of how behaviors and environmental factors relate to mortality to generate more rigorous theoretical models and to develop hypotheses that can be tested to advance our understanding of the factors at play and the ways in which they interact. Ultimately, a better understanding of causal processes could help to identify effective interventions to mitigate rising mid-life mortality rates in certain populations.

Meeting participants suggested several concrete actions that the CPOP and the NIH could take to advance study in this field:

- Commission research papers or host another workshop to identify specific areas for additional research based on the knowledge gaps discussed during this meeting.
- Support a consensus study to identify research priorities over the next 5 to 10 years.
- Encourage research that accounts for variability in age at death and compares trends across subgroups (gender, geography, nativity, socioeconomic status, race/ethnicity).
- Support efforts to identify potentially effective policy or behavioral interventions.

Meeting Summary

Introduction

On June 16, 2017, the National Academies of Sciences, Engineering, and Medicine's Committee on Population (CPOP) and the National Institute on Aging (NIA) of the National Institutes of Health (NIH) convened a group of experts to discuss evidence regarding the role that widening differences in socioeconomic status (SES) may play in life expectancy disparities, especially during middle age, and to identify gaps in the research needed to understand and reverse the trend. The impetus for the meeting was the widely reported study by Anne Case and Angus Deaton that described rising morbidity and mortality in mid-life among non-Hispanic white Americans in recent years.^{1, 2} This increase was largely attributed to increases in "deaths of despair" (e.g., drug use, alcohol abuse, and suicide) among this population.

Dr. Kathleen Mullan Harris, University of North Carolina, Chapel Hill, opened the meeting by inviting attendees to discuss these findings in relation to their own research and to explore trends in mortality across time and populations. She also asked them to provide advice and recommendations to CPOP and NIA on ways that the organizations could address these disparities.

In his opening remarks, Dr. John Haaga, Division of Behavioral and Social Research, NIA, described NIA's long-standing interest in maintaining health across the lifespan and its active research program on mid-life mortality. NIA has issued a Request for Applications (RFA) to study "Socioeconomic Disparities in Health and Mortality at Older Ages."³ The purpose of this research solicitation is to support studies that identify mechanisms, explanations, and modifiable risk factors underlying recent trends of growing inequalities in morbidity and mortality by income, education, and geographic location at older ages in the United States. There are many explanations for why there is an education and income gap in health, but few about why the gap should be larger in 2010 than it was in 1970.

NIA currently supports studies funded under an RFA for "Mid-life Reversibility of Early-established Biobehavioral Risk Factors."⁴ These studies explore the potential for mid-life interventions to improve health outcomes for people affected by various early-life disadvantages and seek to identify the most effective interventions and their optimal timing.

This meeting report captures the key themes of the presentations and discussions that occurred during the 1-day meeting. This planning meeting focused on important questions, such as the appropriate definition of mid-life and whether changes in mid-life mortality trends

¹ A. Case and A. Deaton, Rising morbidity and mortality in midlife among white non-Hispanic Americans in the 21st century, *Proceedings of the National Academy of Sciences USA* 112:15078-15083, 2015.

² A. Case and A. Deaton, *Mortality and Morbidity in the 21st Century*, 2017, available at https://www.brookings.edu/wp-content/uploads/2017/03/casedeaton_sp17_finaldraft.pdf.

³ See <https://grants.nih.gov/grants/guide/rfa-files/RFA-AG-18-011.html>.

⁴ See <https://grants.nih.gov/grants/guide/rfa-files/RFA-AG-14-006.html>.

are driven by changes in behavioral or other factors. The full agenda is provided in Appendix 1, and the participants list is provided in Appendix 2.

Overview of Trends and Differentials

Trends by Age/Period/Cohort, Sex, and Race/Ethnicity

Ryan Masters, University of Colorado-Boulder

Case and Deaton's 2015 study on rising mid-life mortality in non-Hispanic whites and their assertion that this increase was caused by increases in suicide, drug use, and liver disease has been described as indicative of a loss of hope in this age cohort, reflecting psychosocial factors such as despair, diminished expectations, and pain (mis-)management. However, if this explanation were true, one would expect to see similarities in the rate and timing of these increases in despair-related causes of mortality. In addition, these causes of death should be rising faster than other causes of death and should have similar rates in white men and women, and these trends should be especially large in middle-age cohorts.

Instead, Dr. Masters and colleagues believe that three explanations contribute to the observed increase in mid-life (defined as ages 45-54) mortality in this cohort:

1. A period-based increase in drug-related deaths due to the increasing use of legal and illegal opioids
2. An increase in deaths related to increased obesity rates and related health complications
3. A cohort-based increase in deaths related to populations still disproportionately affected by the HIV/AIDS epidemic

If these hypotheses are correct, we would predict increasing rates of drug-related deaths (because the opioid epidemic is still increasing), relatively steady rates of obesity-related metabolic disease deaths (because obesity levels seem to have stabilized), and variable rates of death from HIV-positive individuals depending on their ability to access anti-retroviral therapy.

Decomposing all-cause mortality data into cause-specific mortality data reveals striking differences. Drug-related deaths were higher than those related to alcohol, suicide, or extrinsic causes and steadily increased from 1999 to 2013 in both men and women. During the same time period, deaths from suicide, alcohol, and extrinsic causes were flat in women and, in some cases, decreased in men.

Opioid-related death rates are steadily increasing in black and white men and women. This trend represents a period-based increase that coincides with the U.S. Food and Drug Administration approval of opioid-based painkillers for the treatment of chronic pain in the mid-1990s.

In terms of mortality from metabolic diseases (i.e., obesity, diabetes, and cardiovascular disease), death rates stabilized in the early 1990s. However, there are variations in metabolic

disease mortality that are related to when people were born, with people who spent more years in the modern obesogenic environment having higher morbidity and mortality rates. This effect is seen in both sexes and in both white and black people.

Looking back to 1980 reveals a large residual effect from the HIV/AIDS epidemic—antiretroviral therapy saved thousands of lives in the 1990s, but an increase in HIV-related deaths between 1998 and 2005 suggests that some of these individuals are succumbing to long-term complications of the disease.

There is little evidence for an increase in death rates based on the grouping of drugs, alcohol, and suicide as deaths of despair. Within this trio, drug overdoses explain all of the increase in mortality. The evidence overwhelmingly suggests that recent increases in drug-related mortality are period-based phenomena. Results from age-period-cohort analyses reveal that

1. drug-related mortality rates among baby boom cohorts are neither significantly nor substantively larger than other cohorts.
2. period-based increases in drug-related mortality coincide with the timing of FDA approval of opioid-based painkillers and the subsequent increase in prescription of opioid-based painkillers.
3. drug-related mortality rates have increased for black and white men and women at younger ages as well.

Finally, the influence of the obesogenic environment and its impact on chronic metabolic disease should not be overlooked. Mortality rates from causes of death related to metabolic diseases have increased substantially over successive birth cohorts.

Trends in Mid-life Mortality by SES: Overview and Subgroup Cautions

Jennifer Dowd, King's College London

Overall, life expectancy in the United States has been increasing in most years.⁵ However, when this trend is disaggregated based on race and sex, stark differences emerge, with life expectancy at birth ranging from 83.6 years for Hispanic females to 71.7 years for non-Hispanic black males. Mortality rates also vary significantly by income and educational attainment.

There is consistent evidence that relative and absolute differences in mortality are related to differences in educational attainment. Preston and Elo⁶ reported that men who completed high school in 1960 lived on average 2 years longer than men who did not. This finding was extended in a 2008 study that showed that, when combining people whose education consisted of at least some college, the gains in life expectancy between the 1980s and 2000 were realized

⁵ D.W. Schanzenbach, R. Nunn, and L. Bauer, 2016, *The Changing Landscape of American Life Expectancy*. Brookings Institution, The Hamilton Project. Available <https://www.brookings.edu/wp-content/uploads/2016/07/Full-Paper-2.pdf>.

⁶ S.H. Preston and I.T. Elo, Are educational differentials in adult mortality increasing in the United States? *Journal of Aging Health* 7(4):476-496, 1995.

almost exclusively by the most highly educated groups.⁷ Olshansky and colleagues reported in 2012 that differences in educational attainment resulted in significant widening of differences in life expectancy since 1990.⁸

Evidence suggests that mortality rates are also related to household income. In 2016, Chetty and colleagues reported that the expected age at death for 40-year-old individuals differed by 15 years for those in the bottom 1 percent and top 1 percent of household income.⁹ When disaggregated by income quartile and adjusted for race and ethnicity, the highest income group showed the greatest life expectancy gains. This and other studies suggest that there are widening differences in mortality at middle and older age and that they are related to income.

It is important to ask whether these differences reflect real deteriorations in health among the less educated and lower-earning populations or more rapidly improving health among the better educated and higher-earning populations. In the recent past, there have been dramatic increases in educational attainment and income inequality among individuals. To interpret the data correctly, we need to understand the composition of the group of people who are “well-educated” or “high-earning” to draw meaningful conclusions.

The U.S. high school graduation rate rose from 10 percent in 1900 to nearly 80 percent by 1970. This dramatic change in educational attainment across birth cohorts could artificially generate trends, which makes interpreting trends more difficult. Mortality happens later in life, but selection into education occurs early in life. This observation leads to the possibility of lagged selection bias. For example, the health differentials between adolescents who do and do not complete high school may take decades to become apparent. During the intervening years, evolving social norms may cause the composition of the groups of students who did or did not complete high school to change. As a result, differences may appear to be related to changes across historical periods in factors that influence health but in fact are related to the changing composition of the groups being compared. Indeed, researchers have reported that some estimates of worsening mortality rates among non-Hispanic whites with low SES may be highly sensitive to the way that educational attainment is classified.¹⁰

These findings underscore the importance of selecting subgroups carefully when trying to understand overall effects. Ideally, researchers should strive to select subgroups whose membership is stable over time, although this is not always possible in a complex social environment.

⁷ E.R. Meara, S. Richards, and D.M. Cutler, The gap gets bigger: Changes in mortality and life expectancy, by education, 1981-2000. *Health Affairs* 27(2), 350-360, 2008.

⁸ S.J. Olshansky et al., Differences in life expectancy due to race and educational differences are widening, and many may not catch up, *Health Affairs* 31(8):1803-1813, 2012.

⁹ R. Chetty et al., The association between income and life expectancy in the United States, 2001-2014, *JAMA* 315(16):1750-1766, 2016.

¹⁰ J. Bound et al., Measuring recent apparent declines in longevity: the role of increasing educational attainment, *Health Affairs* 34(12): 2167-2173, 2015.

State-level Trends and Decomposition

Laura Dwyer-Lindgren, University of Washington

The National Vital Statistics System database includes count-level data on nearly all deaths in the United States from 1980 to 2014. In addition to mortality, this dataset includes variables related to age, sex, county of residence, and underlying cause of death. Inclusion of covariates from other datasets, for example on education, income, race/ethnicity, Native American reservations, and population density, enables estimation of all-cause and cause-specific mortality rates using small-area estimation models to generate mortality rates by county, age group, sex, and year.

Dwyer-Lindgren and colleagues have found significant differences in life expectancy at birth at the county level with a 20-year gap in life expectancy between the highest and lowest life expectancy areas. Comparing counties at the extremes of this distribution, the life expectancies of individuals living in counties in the 99th and 1st percentiles have increasingly diverged over time. However, these changes are not consistent across all age groups—inequality in mortality risk has declined in the two youngest groups (0-5 and 5-25) and has increased in the three oldest (25-45, 45-65, and 65-85).

Between 1980 and 2014, the risk of death declined in nearly every county for most age groups; however, there was a significant minority of counties where the risk of death increased among those in the 25-45 age group. From 2000 to 2014, a similar increase in a significant minority of counties was seen for both the 25-45 and 45-65 age groups. In the 25-45 age group, the increase in mortality rates between 2000 and 2014 was seen largely in Appalachia, parts of the South, the southern plains, and the Southwest. Among the 45-65 age group, the distribution was somewhat more limited, with most of the increases occurring in Appalachia and the southern plains.

Mortality due to mental and substance use disorders rose during this period in all age groups and regions. The overall rates were highest in Appalachia and the Southwest, but the areas that saw the largest increases in mortality rates were primarily in the upper Midwest, also known as the Rust Belt. Mortality due to self-harm or interpersonal violence was highest in the West and Alaska. Death due to cirrhosis was highest in the West and Rocky Mountain regions, although there were significant increases in rates in Appalachia, the Southwest, and the Pacific Northwest.

One concern in analyzing data by county is that the results may be influenced by in- and out-migration. However, earlier analyses by others suggest that migration is not likely to be the sole driving force behind these results.

Landscapes of Distress: Spatial Distribution of Risk Factors for Mid-life Mortality

Shannon Monnat, Penn State University

Dr. Monnat has utilized multiple cause of death data from the Centers for Disease Control and Prevention to obtain information about county-level mortality in individuals ages 25 to 64, with

a focus on deaths of despair (i.e., drugs, alcohol, and suicide) and deaths from metabolic diseases (i.e., heart disease, hypertension, diabetes, and obesity). She has used county-level data on socioeconomic, demographic, labor market, social capital, and health care supply to produce linear regression models to examine the relationship between these variables.

Dr. Monnat found that, outside of large metropolitan areas, the risk of mortality for people between ages 45 to 54 has been flat or rising since 1999; only in large metropolitan areas has it been falling. Similar trends are seen across age groups from 25 to 64. Much of this increased mortality risk is driven by non-Hispanic whites, and within this group most of the increase is attributable to increased deaths from drug use. Mortality due to metabolic diseases shows variation based on geographic region and urban status but resides within a narrow range.

The highest number of deaths per 100,000 people ages 25 to 64 were in the South and Western desert states. The highest rates of mortality due to metabolic diseases were in the South. The highest rates of mortality due to drugs, alcohol, and suicide were in Appalachia and the West (including the Rocky Mountain region), with Oklahoma and parts of New England also showing higher levels.

Economic changes seem to be significantly affecting health outcomes in certain geographic areas. These regions may be suffering from a perfect storm that creates a landscape of despair where global economic trends, feelings of distress and despair, and community breakdown lead to a deterioration of social determinants of overall health. These ill health effects manifest themselves not only in deaths of despair but also in deaths due to metabolic diseases. These effects are seen across all non-urban centers, that is, small urban, micropolitan, and rural communities. Most, but not all, of these differences can be explained by differences in population characteristics and levels of economic distress among these communities.

The largest contributing factors to county-level variation in mortality rates from drugs, alcohol, and suicide for individuals ages 25 to 64 were living in a mining-dependent county, the percentage of individuals who were separated or divorced, the percentage of single-parent families, and economic distress (e.g., public assistance receipt, poverty, unemployed/not in labor force). The largest contributing factor to county-level variation in mortality rates from metabolic diseases in the same age group was economic distress, especially the percentage with less than a 4-year college degree and the poverty rate.

Much of the increase in overall mid-life mortality was driven by deaths of non-Hispanic whites outside of large metropolitan areas. The increase was largest for those ages 44 to 55, but was also apparent in those ages 25 to 44. Differences in mortality rates between large urban areas and other areas was largely due to differences in population composition and economic distress. For deaths from drugs, alcohol, and suicide, population composition (i.e., race and age composition) was the largest contributor; for deaths from metabolic disease, socioeconomic distress was the most significant contributors. Dr. Monnat also demonstrated that the presence of social capital-promoting institutions (e.g., churches, sports and recreation facilities, social organizations) is a non-trivial contributor to lower mortality rates.

Discussion

Maxine Weinstein, Georgetown University

Although the focus of this meeting is on mid-life mortality, the inescapable reality is that it is not only middle-age, non-Hispanic whites who are performing poorly. Except for those ages 80 and older, many people living in the United States are faring poorly compared to their international peers. Infant mortality in the United States is relatively higher, and survival to age 50 has been flat for the past 20 years. Life expectancy at birth for non-Hispanic black males is particularly poor.

Dr. Andrew Gelman has noted that, once the Case and Deaton death rates are adjusted for age, much, but not all, of the increase in difference in mortality rates disappears.¹¹ This may reflect a larger issue that arises when datasets are disaggregated into ever-smaller subgroups—the number of individuals from which one tries to draw conclusions becomes smaller and smaller. There are also issues related to lagged selections bias, as Dr. Dowd discussed. That is, people may have sorted themselves into groups years ago, and the consequences of this sorting may only now be emerging. Do these data reflect a cohort effect (i.e., are the members of the group inherently different) or a period effect (i.e., did these people come of age at different times and under different circumstances)?

Zhang and colleagues have found that mortality rates for all adults ages 45-54 increased during the 1990s but leveled off in the 2000s among non-Hispanic white females and declined slightly in non-Hispanic white males.¹² Among non-Hispanic white men and women, deaths from drugs have increased significantly while deaths from alcohol and suicide have remained steady. It is possible that this trend reflects the substitution of one substance for another (e.g., alcohol for opioids) that leads to earlier death among the same vulnerable population.

The causes of these changes may involve shifts in the relationship between work and family. Increasing numbers of single parents—most commonly, employed single women with children at home—may increase vulnerability to conflicts between work and family obligations. Such conflicts may have especially acute effects on single mothers and people with lower SES.^{13,14} Indeed, two of the strongest economic and family stress predictors of mortality are being separated or divorced and single parenthood, with reliance on public assistance being a close third.

Drug-related deaths are clearly rising among non-Hispanic white Americans. However, focus on the larger issue of overall declining health in middle-aged Americans should not be lost.

¹¹ See <http://andrewgelman.com/2015/11/06/age-adjustment-mortality-update/>.

¹² E. Zang et al., Recent Trends in U.S. Adult Mortality: Is It a “Lost Generation” or Lost Generations? An Age-Period-Cohort Perspective. PowerPoint slides presented at symposium in November 2017.

¹³ J.K. Montez et al., Work-family context and the longevity disadvantage of US women, *Social Forces* 93(4):1567-97, 2015

¹⁴ E.L. Sabbath et al., Use of life course work-family profiles to predict mortality risk among US women, *American Journal of Public Health* 105(4):e96-e102, 2015.

Robert Hummer, University of North Carolina, Chapel Hill

This session highlighted several specific patterns and trends that are of great concern, building on the work of Case and Deaton and adding important information and context. Overall, life expectancy in the United States has either been stagnant or declining. Much of the increasing mid-life mortality among whites seems to be associated with increased drug use. Based on Dr. Masters' presentation, an epidemic of drug use and overdose (largely opioid and heroin abuse) is clearly evident, but there is little data to support an equally important increase in deaths due to alcohol abuse or suicide. At the same time, a second major and largely unappreciated epidemic may be unfolding that is associated with dramatic increases in obesity, which, in turn, causes changes in cardiovascular-related mortality. Given the prevalence of obesity in the United States, this trend could have ominous implications for the future. Thus, future work on mid-life mortality should clearly extend its scope beyond the deaths of despair to obesity-related causes of death.

Dr. Dowd described some of the difficulties in understanding trends in mortality based on SES, including the concept of lagged selection bias, which can sometimes lead to apples-to-oranges comparisons if the characteristics of the members of a given group change over time. Analysis of SES as a factor in mortality trends can also be difficult to sort out because it often co-associates with race/ethnicity, immigration status, geographic location, and other variables. Thus, more focus should be given to how SES operates within population subgroups and within birth cohorts to examine its influence in more homogeneous groups of people. All told, though, it is clear that U.S. mortality trends in young adulthood and mid-life for those in the lowest educational and income groups are troubling, no matter which groups are examined and how SES is measured.

Ms. Dwyer-Lindgren described tremendous variation in U.S. life expectancy at the county level. Inequality in mortality seems to be decreasing for people ages 25 and younger but increasing for older age groups. These differences among age groups are quite striking. The data also reveal that the causes of mortality vary greatly by county.

Dr. Monnat's presentation described increases in mortality among young and middle-aged adults living outside of large metropolitan areas. Low SES was strongly associated with death from metabolic diseases. This finding mirrors Dr. Hummer's recent findings on substantially higher cardiovascular disease risk among young adults living outside of the largest metropolitan places. Like that of Ms. Dwyer-Lindgren, this study mines county-level demographic, social, economic, and behavioral correlates of mortality rates and provides important information about social and geographic factors that contribute to mid-life mortality risk.

The impact of personal behavior stands out in several of the causes of death discussed: drug use, interpersonal violence, and alcohol use. How do these factors relate to SES or geographic location? One way to answer this question would be to study two counties with similar demographics, SES, and other dimensions but with very different mortality rates. The results of such a study might yield insights that could be informative for the broader population.

In sum, the observed increase in mid-life mortality—which is often portrayed as a function of increasing drug, alcohol, and distress issues that are concentrated among low-educated whites in rural areas—is deeper, broader, more epidemiologically complex, more diverse, and more institutionally influenced than it seems. Solving this problem is likely to be very difficult. Descriptive studies on mortality patterns and trends have been and will continue to be very important. The search for life course–based mechanisms that connect socioeconomic, behavioral, and health factors to increasing risk of mortality among individuals in mid-life, using population-based data, will also be critical. Finally, it will be important to conduct research on the larger social and economic institutional forces—such as how pharmaceutical companies, the medical establishment, and governmental bureaucracies all may have strongly influenced the opioid epidemic—to best understand increasing mid-life mortality in the United States and what could be done to reverse this trend.

General Discussion

No matter how one examines mortality trends, individuals with fewer years of education or lower SES have poorer outcomes. One challenge of comparing quartiles of data is that today's bottom quartile may be very different from the corresponding cohort 30 years ago, which is an example of lagged selection bias. Another challenge in analyzing education data is simply finding reliable data to analyze and compare.

What is the role of self-selection versus causation of differences in educational attainment on differences in health outcomes and mortality? It may be that, in today's world, students who choose not to complete high school find themselves in a much less welcoming world than the same students a generation ago, leaving them less likely to find a good job, get married, and establish a social support network. It is important to note, however, that trends in educational attainment and SES do not address mechanisms regarding how these factors impact mortality rates.

The Role of the Health System and SES in Access and Constraints to Health and Health Care

The Medical System and Mid-life Mortality

David Cutler, Harvard University

Although economic, social, and psychological factors may be the dominant determinants of mid-life mortality rates, the medical system may also explain some of the variation and likely could do more to address the issue.

Case and Deaton have shown that U.S. whites have an increasing rate of mid-life mortality compared to the populations of several other Western countries. These mortality rates in individuals ages 50 to 54 are highest among people with a high school degree or less. In addition, U.S. whites show a slowing rate of decline in death from heart disease compared to similar populations in other Western countries. What is the role of the medical system in addressing these issues?

First, the medical system may have played a causative role in the increase in deaths of despair. Available data suggest that the striking increase in the use of opioid drugs to manage pain, and the attendant increase in the use of illicit drugs such as heroin, can be traced to a supply-side imbalance of health care providers prescribing pain-relieving medication that has a high risk for addiction. Still, the question remains: Are people in more pain—perhaps due to economic issues, stress, or obesity-related health problems—and therefore require more pain medicine than past generations?

Second, although the medical system has many tools to address cardiovascular disease, the availability of prevention and treatment options is often curtailed in low SES populations. For example, health care providers have known for 50 years about the importance of controlling blood pressure in their patients, yet only about one-third of people with hypertension control it effectively, and there are clear differences based on race and SES within this group.

To provide more effective care, the medical system should focus on reaching out to patients rather than relying on them to seek care. Prevention is much more effective than treatment, in terms of both long-term health outcomes and cost. Health care also needs to be affordable; data show quite clearly that an increase in out-of-pocket costs leads to a decrease in health care utilization.

Geographic areas with a low number of uninsured people do not show significantly better outcomes; life expectancy at age 40 is not strongly related to insurance status for individuals at the bottom quartile of income. Although increasing the availability of health insurance may improve overall health, its impact on survival seems more complex. This leads to the conclusion that, although the health system has an important role to play in improving health outcomes, it is just one factor in a complex ecosystem of health care.

SES and Mid-life Health/Mortality: The (Underappreciated) Role of U.S. States

Jennifer Karas Montez, Syracuse University

To affect change in mid-life health and mortality, it may be necessary to change the way we think about the relationship between education and health and to recognize the role that policies adopted by state governments contribute to the variations across geographic regions.

The central questions are: Why does education shape health and mortality? Why has this association grown stronger over time? Why has the mortality of low-educated adults increased over time?

Conventionally, education in the United States is thought of as a personal resource, with the individual *pursuing* a healthy lifestyle, *seeking* medical knowledge or care, *avoiding* financial hardship, and *developing* social ties, among other positive traits. However, these behaviors occur within a social context.

The Surgeon General's report in 1964 on the dangers of smoking was likely received quite differently by high- and low-educated adults. High-educated adults were more likely to have

known about the report and to have read about the risks of smoking, likely leading to a significant fraction of them choosing to stop smoking. In comparison, low-educated adults were less likely to be aware of the report. Since the 1990s, the range of cigarette taxes has expanded greatly among the states, with some adopting very high taxes and others adopting very low taxes. This provides a natural experiment to compare smoking rates among high- and low-SES individuals in states with higher and lower cigarette taxes.

Dr. Montez and colleagues have found much greater cross-state variation in disability prevalence among low-educated individuals than among high-educated individuals. Therefore, the size of the educational disparity in disability in a state is largely determined by the prevalence of disability among low-educated adults in a state. Disability prevalence of high-educated adults varies very little across states. Therefore, while education level plays a role in an individual's likelihood of disability, so do the policies of the individual's home state.

Similar results are seen in measures of activities of daily living, mobility, vision, hearing, and cognition. Indeed, the probability of being in fair or poor health varies dramatically across states for low-educated adults but varies little for high-educated adults.

To understand the relationship between educational attainment and health outcomes, we must examine the social environments that create and sustain it. Dr. Montez and colleagues found that the policies adopted by U.S. state governments may help to explain the expanding chasm between health outcomes for high- and low-educated residents.

Discussion

Jere Behrman, University of Pennsylvania

Dr. Montez suggested that part of the benefit of educational attainment might be that it allows people to adapt to change, as was shown in the response of the highly educated population to the Surgeon General's report about smoking. However, it is not clear that the relationship between education and health outcomes is causal. This illustrates the difficulty of answering "why" questions in this context. Factors other than cigarette taxes might influence residents' health, including federal and state health care programs such as Medicaid or the generosity of disability insurance. It is difficult to tease out effects of any single policy because none of them acts in isolation.

Dr. Cutler's presentation explored the contribution of the medical system to recent changes in mortality rates in the United States, relative to other countries. For individuals in the bottom quartile of income distribution, access to health insurance does not seem to have a strong correlation with life expectancy.

However, looking at data by income quartiles may mask important contributions of age, race, migrant status, and other factors. In addition, looking only at mortality may mask important differences in overall health and quality of life. The current dataset may not fully incorporate the lagging impact of decades of obesity on a subset of the population. The key to improving mortality rates may lie in changing behavior, not in improving access to health care.

For example, death rates from cardiovascular diseases are declining in the United States but at a slower rate than in other countries. Is this related to a higher level of despair in this country? Is it because the U.S. health system is good at treating but not preventing heart disease? Is it because the United States has had an aggressive blood pressure control campaign for 50 years, effectively already realizing most of the gains from this program? Or is it because the obesity epidemic appeared in the United States years before it appeared in Europe and therefore the European data reflect a lagged selection bias effect?

These questions illustrate the complexity that exists within the data on mortality. More research is needed to clarify the contributors to the observed outcomes and to better characterize their causes and effects.

General Discussion

What is the role of interstate migration in influencing differences in mortality rates among states? It seems that migration is a minor contributor to the overall outcomes. In fact, if one looks only at individuals who have spent their whole lives in one state, the disparities are even greater. It is not clear whether this is an exposure effect (i.e., the longer one lives in a given environment, the more likely it is to impact his or her outcomes) or a selection effect (i.e., the people who are most open to new ideas and experiences are the ones most likely to leave).

Life-course Perspectives on Mid-life Mortality

Health Risks for Mid-life Mortality Arising in Earlier Stages of the Life Course

Kathleen Mullan Harris, University of North Carolina, Chapel Hill

There is a large and growing literature on the importance of early-life conditions for the development of adult health and mortality risk. Early-life circumstances, including the uterine environment and early childhood exposures, are both directly and indirectly associated with health outcomes that occur decades later, as well as human capital and labor force outcomes in adulthood.

Adolescence and early adulthood are important developmental stages because they represent periods during which one spends less time with family and has more control over behavioral choices. Additionally, physical, neurological, psychological, and hormonal changes interact with the adolescent's environment to have long-lasting effects on adult behavior.

The National Longitudinal Study of Adolescent to Adult Health is a longitudinal study of a sample of U.S. adolescents that began when they were students in grades 7 through 12 during the 1994-1995 school year. The study has continued to follow this cohort as they transition into adulthood. The current generation is at the forefront of the obesity and metabolic disease epidemic, which has potentially dire implications for its future risk of disease, as well as social and economic consequences.

During the transition to adulthood phase, which occurs between ages 18 and 26 in this study, researchers observed an increase in multiple negative health traits and behaviors, including

obesity, lack of physical activity, lack of engagement with the health care system (e.g., no regular physical or dental check-ups), smoking, drug use, and binge drinking. During the subsequent young adulthood period, many of these behaviors stabilized but did not significantly decline, while obesity and the rate of sexually transmitted diseases continued to climb.

This study discovered an alarmingly high prevalence of disease risk during the young adulthood phase, when participants assumed that they were healthy because they felt well. Most were not routinely screened for disease risk; therefore, there seems to be a silent epidemic of hypertension and diabetes in this population. Thirty-seven percent are obese; another 30 percent are overweight. Twenty-seven percent have hypertension; another 49 percent have prehypertension. Six percent have frank diabetes; another 27 percent have pre-diabetes. Alarmingly, 75 percent of those with hypertension are unaware, and 90 percent of those with prehypertension are unaware. Of those with diabetes, 67 percent are unaware; virtually all of the 98 percent with pre-diabetes are unaware of their diagnosis.

Dr. Harris and colleagues analyzed these findings by examining risk factors based on several characteristics, including race/ethnicity, parental education, geographic location, and SES status. They found that cardio-metabolic risk correlated with education: the young adult offspring of more-educated parents as well as more-educated young adults have less risk. Young adult household income showed similar correlations with health risk.

Obesity is also associated with multiple social and economic consequences that may be especially important in early adult life. Obesity in adolescence and during the transition to young adulthood is associated with greater social isolation, depression, and suicidal thoughts or attempts in young adulthood; lower rates of marriage and less education; lower wages, household income, homeownership, and assets; and greater debt and job instability in young adulthood.

Young adults today in their 30s represent an obesity cohort in that they are the first young adult group who experienced the dramatic rise in obesity during adolescence. They were obese earlier in life and have been obese for longer as young adults. They have much higher rates of chronic disease and disease risk than prior young adult cohorts. These findings forebode an explosion of cardiovascular disease and metabolic disorder for this group when they reach their 40s and 50s. The time to intervene is now, to avoid permanent biological damage among young adults and future health care costs to families and society.

Life-course Predictors of Mid-life Mortality

Dana Glej, Georgetown University

A challenge to conducting research on mid-life mortality is that few people die in mid-life (defined as ages 46 to 64). In the Mid-life in the United States (MIDUS) cohort study of individuals ages 25-74 in 1995-1996, only about 16 percent of the participants died within 18 years and only 285 of those (less than 5 percent) died between the ages of 46 and 64. An

extremely large sample would be required to study mortality during the mid-life period at the individual level.

The existing literature points to several life-course factors that predict mid-life mortality. One of the earliest is early childhood SES. Although early childhood SES is associated with mortality (and may be even more strongly associated with mid-life mortality although there are very few studies that investigate mid-life mortality specifically), most of the effect is mediated by SES in adulthood, and the effect of adult SES appears to be stronger. However, childhood SES is a fundamental cause; it is likely to be linked with a cluster of exposures that may influence mortality later in life.

Prior studies also suggest that employment and career trajectory are linked to mortality rates. Specific individual-level factors related to mortality risk in men include job complexity, total family income, and net assets. There is evidence at the county level that areas with better economic opportunity and greater economic mobility have better health outcomes. Additionally, both men and women who were consistently married showed lower mid-life mortality.

Using data from MIDUS, Dr. Gleib and colleagues asked what variables were the best prognostic predictors of mortality, during mid-life (40-64) and later-life (65-92). The best and perhaps most obvious predictor is age: the older you are, the higher your risk of mortality, although age is a much better predictor of mortality in later life than in mid-life. Sex and race/ethnicity made a small incremental contribution (net of age), as did childhood SES, educational attainment, and marriage and parenthood, especially in mid-life. Subjective measures of a person's work and financial situation (including expectations about future work) also predict mortality rates above and beyond the contribution of current social and economic status, but again the contribution is much greater for mid-life than for later-life mortality. Net of age, self-assessed overall health, which likely incorporates measures of both physical and mental well-being, is the single best predictor of mortality (in both mid-life and later life).

In prior studies based on data from the National Health and Nutrition Examination Survey (NHANES), Dr. Gleib's team examined whether several commonly collected biomarkers could predict mid-life mortality and found few strong relationships. The only biomarkers with notable predictive ability were C-reactive protein (a marker for generalized inflammation) and homocysteine (a marker for inflammation in blood vessels). Many of the standard clinical markers, such as obesity, blood pressure, and lipid levels, were poor predictors of mortality even in later life. Of course, these markers could still influence overall health and well-being, even if they are not the best predictors of mortality.

MIDUS has extensive information about childhood adversity, psychological health, and personality, as well as more than 18 years of follow-up data. However, it enrolled few minorities and has sparse information about labor force entry and career development. The National Longitudinal Study of Youth 1979 could be another important source of information. It is a nationally representative sample of more than 12,000 young men and women who were between the ages of 14 and 22 when they were first surveyed in 1979. These individuals were

interviewed annually through 1994 and are currently interviewed on a biennial basis. Currently, however, NLSY79 does not include mortality follow-up. Another potential data source is the National Longitudinal Mortality Study administered by the U.S. Census Bureau, which contains approximately 3.8 million records with more than 550,000 identified mortality cases as well as data related to SES that may not be available in other databases, but it contains limited information regarding life history.

Discussion

Mark Hayward, University of Texas-Austin

A life-course approach is fundamentally important in understanding current population and mid-life mortality trends, yet it does have pitfalls. Although researchers have a good understanding of the life-course origins of adult mortality, it can be difficult to discern early- and mid-life signals that predispose some individuals to increased risk of mid-life mortality compared to late-life mortality.

The data presented show that Americans increasingly reside in “multiple Americas,” with different life-course trajectories that are influenced by educational, economic, and geographic factors. There seems to be a clear relationship between educational attainment and life expectancy at age 25 in both men and women. These trends are more pronounced across increasing ages. Comparing the leading causes of death in individuals between the ages of 45 and 64 in 2014 versus 1980 reveals an increase in unintentional injuries, chronic liver disease, pulmonary disease, diabetes, and suicide. The increases in deaths from these causes point toward behavioral factors as an underlying cause of increased all-cause mortality.

Childhood and adulthood are both characterized by a set of risks, rewards, and behaviors that together may influence mid-life mortality. Some of these are difficult to modify at the individual level (e.g., parents’ SES), while others may be more feasible to change (e.g., smoking, exercise, or nutrition). The question for researchers is whether specific points during the lifespan are especially amenable to intervention to effect lasting changes.

It is worth discriminating between lifetime exposure risks and behavioral risks. It is not possible to change an individual’s upbringing, but it may be possible to influence his or her behavior as a young adult. Are there strategies to help people cope with early-life adversity so that its impact in mid- and later-life is diminished? How might the increase in childhood obesity intersect with new influences in adulthood, such as the loss of working-class employment and instability in marriage and family relationships? A challenge to addressing these issues is that the composition of any cohort is changing over time and is subject to lagged selection bias.

Behavioral Factors

Determinants of Mid-life Health Behavior

Jason Boardman, University of Colorado-Boulder

In 1995, Link and Phelan developed a theory of fundamental cause of disease.¹⁵ Instead of focusing on individual risk factors for major diseases such as diet, cholesterol level, and exercise, they argued for the importance of identifying and addressing the fundamental causes of those risk factors. In this model, factors such as SES and social support are likely fundamental causes of good or bad health outcomes. For example, people of higher SES are more likely to exhibit positive health behaviors, whereas people of lower SES are more likely to exhibit detrimental health behaviors. Understanding the reasons behind this disparity in health behavior might allow for the development of novel interventions that could produce substantial health benefits.

It is important to consider the social environment in which individuals make behavioral choices that affect their individual health outcomes and, by extension, the collective health of populations. The current mid-life cohort is experiencing a new social context as it ages and undergoes demographic transformation. The cohort's composition is modified not only by status changes of the individuals who comprise it but also by selective changes of its membership.

Is there a genetic component to these social behaviors? By comparing the correlation of traits between identical and fraternal twins it is possible to infer the proportion of social variation that is due to genetic influence. For example, smoking among adolescents in the United States is highly heritable but can be modified by environmental factors such as higher cigarette taxes. The ability of environmental factors to alter genetic predisposition to behavior suggests the possibility that cohorts themselves could be environmental moderators of genetic influences.

The heritability of regular smoking is between 40 and 50 percent, and it has been increasing across birth cohorts. The heritability of educational attainment has remained at approximately 40 percent across birth cohorts. The negative correlation between education and smoking has increased across birth cohorts over time.

Dr. Boardman and colleagues asked whether it was possible that the genes related to educational attainment were also related to smoking behavior. They also examined whether the genetic correlation between education and smoking has changed across birth cohorts. They found that the relationship between education and smoking has increased significantly over recent cohorts. This association is primarily among college-educated individuals who are reducing smoking at a far greater rate than other individuals.

¹⁵ B.G. Link and J. Phelan, Social conditions as fundamental causes of disease, *Journal of Health and Social Behavior* 80-94, 1995.

These genetic and social factors may be sorting people into different schools, jobs, and neighborhoods, leading to increasing correlation between education and smoking. These traits may also be self-reinforcing, further widening disparities. Theories of SES and health need to consider this selection pattern and its implications for the perpetuation of inequality. The non-random selection of genetically similar people into education and health lifestyle patterns will complicate the role of cohort as a purely environmental effect.

Behavioral Contributions to Mid-life Morbidity and Mortality

Neil Mehta, University of Michigan

It is important to differentiate between overall mid-life mortality rates and how these rates change over time. Mortality rates among non-Hispanic white men due to coronary heart disease and smoking-related and other cancers have either remained flat or have declined between the 1990s and the 2000s, while those from drug overdoses have increased. Similar results are seen in mortality rates of non-Hispanic white females.

The incidence of diabetes by birth cohort has risen steadily over time, with a marked increase beginning with those born around 1960. Since the mid-1970s, the proportion of people who report having ever smoked or having ever been obese has held steady at around 75 to 80 percent. The decrease in individuals who report smoking has been almost exactly offset by individuals who have been, or are, obese.

Longitudinal data suggest that older cohorts are more likely to smoke but are less likely to be obese. However, this overall difference masks important trends based on educational attainment: those with a college degree are half as likely to be obese as those without. Individuals born during the 1940s and 1950s show low levels of obesity at age 25 but higher levels during mid-life. This increase in obesity is consistent with a period effect, beginning in the mid-1980s. People born in the 1960s and 1970s will experience greater exposure to an obesogenic environment.

Members of different SES groups exhibit different mortality risks, and the rate at which these risks change over time also varies by SES status. Comparing individuals with and without diabetes, and comparing annual income of more than or less than \$20,000, the poorest people with diabetes showed the least improvement in survival.

Turning from mortality to disability, the most important risk factors for disability among those ages 55 to 74 were obesity, hypertension, and smoking, which with other factors accounted for approximately 70 percent of the risk. This is notable because there have been significant efforts to promote control of hypertension, yet its contribution to disability remains stubbornly high.

Slowdowns in the rate of decline of heart disease and cancers have contributed to the stagnation of mortality rates. Obesity and diabetes are the most likely culprits for the leveling off of these mortality trends, but the evidence is not conclusive. All SES groups are benefiting from advances in the management of cardiovascular disease, and related risk factors remain an open question.

Discussion

Hillard Kaplan, University of New Mexico

Dr. Kaplan provided deeper evolutionary context with respect to diet, physical activity, sociology, and cardiovascular disease. The Tsimane are an indigenous people in Bolivia who lead a hunter/gatherer lifestyle. They consume large numbers of calories but also expend energy through walking and other physical activities. Their society is organized around multi-family bands that cooperate in food acquisition and sharing. The men and women in this population show astonishingly low rates of coronary heart disease, which is likely due to a combination of both dietary and social factors.

If placed in a modern setting with access to calorie-rich, nutrient-poor food, and fewer opportunities to engage in physical activity, the finely tuned evolutionary equilibrium of the Tsimane would be upset. The example of hunter-gatherer economies such as that of the Tsimane can inform development of testable hypotheses and a multi-level causal framework for dynamically evolving and divergent socioeconomic-behavioral-health equilibria.

There are many potential explanations for differences in mid-life mortality rates: behavioral factors such as obesity, hypertension, and smoking as well as social factors such as early-life adversity, class distinctions, and lack of access to information. Some of these factors have a genetic component, although there is evidence that these can be modified by environmental influences. Approaches to address this problem will therefore need to consider not only biologic factors such as genetics but also environmental and social factors.

Challenges for future research include addressing why genetic heritability of important traits is so high; incorporating biology more fully into social demography; explaining complementarity and competing theoretical constructs; organizing causal hypotheses in multi-level path models; developing equation systems for incorporating the effects of context on outcomes; and understanding how networks of beliefs and practices evolve over time in the context of dynamically changing equilibria.

General Discussion

Dr. Behrman noted a tension in developing experimental model systems between trying to be all-inclusive versus focusing on a subset of factors that could be interpreted in a more meaningful way. There may be a trade-off between a simple theory that models a few things well versus a complex theory that is too opaque and unwieldy. Dr. Kaplan thought that it might be better to try a complex model first and then use parts of it to conduct reductionist studies if the overall framework proves unwieldy. This should not be thought of as an “either-or” question, but as “both-and” question.

Summary, General Discussion, and Needs for Further Research

Dr. Haaga encouraged the attendees to consider critical gaps in knowledge and how those could be addressed through efforts supported by the National Academies and NIH. Support could include additional workshops or meetings, career development activities, or research initiatives. Many of the ideas discussed at this workshop could be developed into NIH

initiatives. He also encouraged the attendees to continue to think about the issues discussed at this meeting in their future research. This workshop represents the beginning of a conversation about this very important topic.

Dr. Harris suggested that the National Academies could commission research papers and host another workshop to identify specific areas for additional research based on the knowledge gaps that were discussed at this meeting. This effort could result in a consensus study to identify research priorities over the next 5 to 10 years. The issues facing individuals in mid-life are complex and interconnected and will require serious thought and study to address.

Dr. Kaplan noted that some of the data presented, such as those on the impact of cigarette taxes on health and mortality, suggest that some straightforward interventions could have a large impact. Efforts to identify these strategies should be supported. Dr. Wilmoth reminded the attendees that the overall goal of any research effort should be to improve the lives of all individuals. While it is tempting to focus on average data, it is important to ensure that no one is left behind.

Appendix 1: Agenda

- 9:00 a.m. **Welcome and Discussion of Agenda**
Kathleen Mullan Harris, University of North Carolina, Chapel Hill
- 9:10 **National Institute of Aging Mid-Life Mortality Research Program**
John Haaga, Office of Behavioral and Social Research, NIA
- 9:20 **Overview of Trends and Differentials**
- Trends by Age/Period/Cohort, Sex, and Race/Ethnicity**
Ryan Masters, University of Colorado-Boulder
- Trends in Mid-life Mortality by SES: Overview and Subgroup Cautions**
Jennifer Dowd, King's College London
- State-level Trends and Decomposition**
Laura Dwyer-Lindgren, University of Washington
- Landscapes of Distress: Spatial Distribution of Risk Factors for Mid-life Mortality**
Shannon Monnat, Penn State University
- Discussion**
Maxine Weinstein, Georgetown University
Robert Hummer, University of North Carolina, Chapel Hill
- 11:45 **LUNCH**
- 12:30 p.m. **The Role of the Health System and SES in Access and Constraints to Health and Health Care**
- The Medical System and Mid-life Mortality**
David Cutler, Harvard University
- SES and Mid-life Health/Mortality: The (Underappreciated) Role of U.S. States**
Jennifer Karas Montez, Syracuse University
- Discussion**
Jere Behrman, University of Pennsylvania

- 1:45 **Life-course Perspectives on Mid-life Mortality**
- Health Risks for Mid-life Mortality Arising in Earlier Stages of the Life Course**
Kathleen Mullan Harris, University of North Carolina, Chapel Hill
- Life-course Predictors of Mid-life Mortality**
Dana Gleib, Georgetown University
- Discussion**
Mark Hayward, University of Texas-Austin
- 3:00 **BREAK**
- 3:15 **Behavioral Factors (Substance Use, Addiction, Diet, Physical Activity, etc.)**
- Determinants of Mid-life Health Behavior**
Jason Boardman, University of Colorado-Boulder
- Behavioral Contributions to Mid-life Morbidity and Mortality**
Neil Mehta, University of Michigan
- Discussion**
Hillard Kaplan, University of New Mexico
- 4:30 **Summary, General Discussion, and Needs for Further Research**
- 5:00 **Adjourn**

Appendix 2: List of Participants

Jere Behrman, PhD, University of Pennsylvania
Jason Boardman, PhD, University of Colorado, Boulder
David Cutler, PhD, Harvard University (via Webex)
Jennifer Dowd, PhD, King's College London
Laura Dwyer-Lindgren, MPH, University of Washington
Dana Gleib, PhD, Georgetown University
Kathleen Mullan Harris, PhD, University of North Carolina, Chapel Hill
Mark Hayward, PhD, University of Texas, Austin
Robert Hummer, PhD, University of North Carolina, Chapel Hill
Hillard Kaplan, PhD, Chapman University
Ryan Masters, PhD, University of Colorado, Boulder
Neil Mehta, PhD, University of Michigan
Shannon Monnat, PhD, Penn State University
Jennifer Karas Montez, PhD, Syracuse University
Maxine Weinstein, PhD, Georgetown University
John Wilmoth, PhD, United Nations Population Division

National Institutes of Health

Gregory Bloss, MA, Economist, Division of Epidemiology and Prevention Research, National Institute on Alcohol Abuse and Alcoholism
Nancy Breen, PhD, Health Economist, Applied Research Program, Division of Cancer Control & Population Sciences, National Cancer Institute
Regina Bures, PhD, Extramural Program Staff, National Institute of Child Health and Human Development (NICHD)
Rebecca Clark, PhD, Chief, Population Dynamics Branch, NICHD
Sarah Duffy, PhD, Associate Director for Economics Research, Services Research Branch, Division of Epidemiology, Services and Prevention Research, National Institute on Drug Abuse
Jovier Evans, PhD, Chief, Geriatric Translational Neuroscience Program, National Institute of Mental Health
Lawrence Fine, MD, Chief, Clinical Applications and Prevention Branch (CAPB), Division of Cardiovascular Sciences (DCVS), National Heart, Lung, and Blood Institute (NHLBI)
John Haaga, PhD, Director, Division of Behavioral and Social Research (BSR), National Institute on Aging (NIA)
Amelia Karraker, PhD, Health Scientist Administrator, BSR, NIA
Evelyn Neil, Program Analyst, BSR, NIA
Lisbeth Nielsen, PhD, Chief, Individual Behavioral Processes Branch, BSR, NIA
Georgeanne Patmios, MA, Acting Chief, Population and Social Processes Branch, BSR, NIA
Nicole Redmond, MD, PhD, MPH, Medical Officer, CAPB, Program in Prevention and Population Sciences, DCVS, NHLBI

Michael Spittel, PhD, Health Scientist Administrator, Office of Behavioral and Social Sciences Research

Division of Behavioral and Social Sciences and Education (DBASSE), National Academies

Peter Donaldson, PhD, Interim Board Director, Committee on Population

Monica Feit, PhD, Deputy Executive Director

Mary Ghitelman, Senior Program Assistant, Board on Environmental Change and Society, the Committee on Population, and the Board on Children, Youth, and Families

Leticia Garcilazo Green, Senior Program Assistant, Committee on Law and Justice

Malay Majmundar, PhD, Associate Director, Committee on Law and Justice