

Annual Review of Sociology Social Inequality and the Future of US Life Expectancy

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Annu. Rev. Sociol. 2021. 47:501-20

First published as a Review in Advance on March 10, 2021

The *Annual Review of Sociology* is online at soc.annualreviews.org

https://doi.org/10.1146/annurev-soc-072320-100249

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Keywords

social inequality, life expectancy, biodemography, diffusion of innovations, extrinsic causes of death

Abstract

Despite decades of progress, the future of life expectancy in the United States is uncertain due to widening socioeconomic disparities in mortality, continued disparities in mortality across racial/ethnic groups, and an increase in extrinsic causes of death. These trends prompt us to scrutinize life expectancy in a high-income but enormously unequal society like the United States, where social factors determine who is most able to maximize their biological lifespan. After reviewing evidence for biodemographic perspectives on life expectancy, the uneven diffusion of health-enhancing innovations throughout the population, and the changing nature of threats to population health, we argue that sociology is optimally positioned to lead discourse on the future of life expectancy. Given recent trends, sociologists should emphasize the importance of the social determinants of life expectancy, redirecting research focus away from extending extreme longevity and toward research on social inequality with the goal of improving population health for all.

INTRODUCTION

The future of life expectancy in the United States—centered around issues of what is biologically possible and/or socially attainable in years to come—has long been a focal topic of interest in population research. The study of life expectancy over the past 20 years is best-described by a growing set of research questions intent on deciphering mortality patterns and trends and their many determinants. "How long will humans live?" serves as one of the fundamental motivating questions (Bongaarts 2006), continuing to spur debates about the biological limits of the human lifespan (Barbi et al. 2018, de Beer et al. 2017, Dong et al. 2016, Lenart & Vaupel 2017, Newman 2018). Yet more nuanced inquiries are pursued as well; formal demographers and researchers on aging consider, "What are the biological processes that define aging, senescence, and maximal lifespan?" (Baudisch & Vaupel 2012, Christensen et al. 2006, Hjelmborg et al. 2006, Vaupel 2004, Vaupel 2010), extending their research to question, "Who lives longest?" and "How much variation is observed?" (Carnes & Olshansky 2007, Carnes et al. 2013, Oeppen & Vaupel 2002, Olshansky et al. 2002, Rau et al. 2008, Shkolnikov et al. 2011b). A separate but closely related branch of research runs in parallel, with sociologists and social demographers asking, "How is longevity socially patterned?", "What are its social determinants?", "How are these determinants allocated?", and "What are the mechanisms by which these determinants augment or reduce longevity?" (Elo 2009, Fenelon & Boudreaux 2019, Firebaugh et al. 2014, Hayward et al. 2015, Lariscy et al. 2016, Masters et al. 2014, Miech et al. 2011, Montez et al. 2011, Montez & Zajacova 2013, Rogers et al. 2013, Sasson & Hayward 2019).

Consequently, the scientific insight gained regarding life expectancy is matched only by the degree of continued uncertainty surrounding its biological and social determinants. Perhaps never has understanding these biological and social inputs been so paramount: As innovations in science, technology, and medicine portend the onset of improved health and lower mortality, we simultaneously observe steeper socioeconomic disparities in US mortality (Hayward et al. 2015, Masters et al. 2012, Miech et al. 2011, Montez & Zajacova 2013, Sasson 2016b, Sasson & Hayward 2019), continued racial/ethnic disparities in mortality (Elo et al. 2014, Hummer & Chinn 2011, Hummer & Gutin 2018, Gennuso et al. 2019, NCHS 2019, Williams 2012), and emerging and unexpected threats to population health that are reversing decades of progress (Alexander et al. 2018, Case & Deaton 2015, Glei & Preston 2020, Ho 2020, Masters et al. 2018, Sasson 2016a). These disturbing trends reflect the complexity of understanding and forecasting the biological and social realities of life expectancy amid a context of high and rising social inequality. This complexity is increasingly borne out in trends within the contemporary United States, where stark inequalities in life expectancy highlight the diverging health prospects between the most and least socially advantaged subgroups of the population.

Forecasting life expectancy is a matter of national significance, integral to anticipating the social, economic, and health needs of future generations. These projections are vital for understanding the burden on welfare programs and health care infrastructure, as well as the changing composition of families and the labor force, to name just some of the many social institutions affected (Bongaarts 2006, Olshansky 2013, Olshansky et al. 2009a, Rae et al. 2010). Understanding the future of life expectancy in the United States—as simultaneously bounded by the biological limits of human lifespan; the potential for scientific, technological, and medical innovations to allow us to reach or extend these limits; and the societal forces defining individuals' abilities to capitalize upon these innovations—is an important endeavor, requiring the integration of multiple perspectives on the determinants of life expectancy.

The purpose of this review is to synthesize recent biodemographic and social demographic research in order to highlight how prognoses of future life expectancy are inextricably linked to

social inequality in the United States. We begin by summarizing what research in biodemography suggests about the scientific and social conditions needed to stimulate gains in life expectancy. We then consider how these expectations interact with research documenting how social inequality structures individuals' access to the innovations and opportunities necessary to promote longevity. Finally, we examine what emerging evidence on socioeconomic and racial/ethnic disparities in US mortality, largely driven by the rise in extrinsic causes of death, suggests about future trends. As the forces shaping life expectancy are principally social rather than biological, we argue that sociologists should be at the forefront of this discourse, shifting population health research away from a focus on extending longevity and toward efforts to better understand inequalities in longevity, with the goal of healthier and longer lives for all rather than for just a small subset of the population.

BIODEMOGRAPHIC PERSPECTIVES ON LIFE EXPECTANCY

The emergence of biodemography—an interdisciplinary area of study concerned with integrating biological concepts into demographic approaches to better understand population problems and processes (Carey & Vaupel 2005)—has been integral in advancing research on life expectancy. Given that humans are unique in their degree of control over their environments and are not confined to a biologically predetermined option set of aging and mortality patterns (Baudisch & Vaupel 2012), biodemography affords a powerful theoretical and analytical framework for studying longevity as a biosocial phenomenon.

Biodemographers are careful to note the inherent complexity of isolating any single factor as a determinant of longevity, as well as the limitations of translating biological knowledge into biomedical applications. Vaupel (2004) concedes that the original goal in studying human lifespans was to arrive at a core set of keys to longevity, such as specific genetic and/or behavioral and environmental factors. However, most researchers agree that lifespan is a function of thousands of genetic and nongenetic influences (Christensen et al. 2006) and that lifelong socioenvironmental exposures are more consequential than biological predisposition (Hjelmborg et al. 2006). Indeed, evidence is mixed regarding the different combinations of environmental factors, behaviors, psychosocial influences, and health profiles that serve as pathways to exceptional longevity (Christensen et al. 2006), consistent with the view of aging as a remarkably plastic process (Vaupel 2004).

Consequently, extending longevity is an ambitious, multi-pronged operation contingent upon future success in maintaining population prosperity, improved health among younger people, public health efforts to maintain salubrious lifestyles, high-quality health care, and development of knowledge regarding continued scientific innovation in reducing morbidity and mortality (Vaupel 2010). The relative importance of these factors is subject to debate, as is the broader question of whether humans have a maximum lifespan (Barbi et al. 2018, Dong et al. 2016, Oeppen & Vaupel 2002, Olshansky et al. 2002). Thus, rather than provide a comprehensive review of extant evidence, we emphasize the core biodemographic perspectives that are most salient to the future of life expectancy.

Broken and Breaking Limits to Life Expectancy

The hopeful outlook on the future of life expectancy reflects a world in which new knowledge is readily available and advances in biotechnology, preventive and curative medicine, and drug treatment will continue to be effective in reducing mortality (Bongaarts 2006). This standpoint on aging as a malleable process underlies the optimistic perspective on future life expectancy. Vaupel, Oeppen, and other optimists promote a view of seemingly unbounded gains to life expectancy, as

evidenced by increasing trends in life expectancy throughout human history—repeatedly breaking past the limits set in prior research and slowing mortality at older ages (Barbi et al. 2018, Burger et al. 2012, de Beer et al. 2017, Oeppen & Vaupel 2002, Lenart & Vaupel 2017, Rau et al. 2008, Shkolnikov et al. 2011b, Vallin & Meslé 2009).

A core assumption of this optimistic outlook is that scientific innovations, both existing and hypothesized, will delay senescence and increase late-life survival. Though past and current gains in life expectancy are attributable to multiple factors—e.g., improvements in educational attainment, public health, nutrition, sanitation, and housing (Burger et al. 2012, Cutler & Miller 2005, Vallin & Meslé 2009, Vaupel 2010)—innovations in science, technology, and medicine are consistently singled out as vital to life extension in recent decades (Rae et al. 2010, Rau et al. 2008). Bongaarts (2006) contends that pessimistic forecasts of life expectancy are misguided on account of their inability to anticipate continued improvements; Vallin & Meslé (2009) note the possibility of future advances that extend maximum human lifespan. Consequently, the only way to truly forestall senescent mortality is through highly targeted interventions that target the bio-genetic roots of aging itself (Vaupel 2010).

Indeed, this drive to isolate the biological causes of poor health and premature death—and, in turn, develop individualized treatments—is at the forefront of US government-funded initiatives to improve population health. Perhaps no future innovation has received as much attention and funding as precision medicine, in using biological and genetic data to develop targeted, patient-specific solutions for leading causes of morbidity and mortality. The National Institutes of Health-funded All of Us initiative seeks to recruit over one million participants in the coming decade to advance these bio-genetic data collection efforts (Precis. Med. Initiat. Work. Group 2015). While some have questioned the efficacy of seeking out biomedical solutions for health issues rooted in social inequality (Weiss 2017), it is clear that many individuals and institutions continue to promote a fundamentally optimistic view of the role of innovation in increasing US life expectancy.

The Realist View on Longevity

Arguing that demographic, biological, and biomedical constraints will stymy continued gains in life expectancy, Carnes & Olshansky (2007) contend that the labeling of their view as pessimistic is inaccurate with respect to their expectations for future reductions in mortality. They instead propose realist as a more accurate descriptor of their more conservative position, emphasizing the value of projecting life expectancy based on present-day conditions rather than potential advances. Contra optimists, realists argue that the existence of some limit—perhaps higher than current estimates (Carnes et al. 2013, Olshansky & Carnes 1997, Olshansky et al. 2002) and liable to be broken as well (Carnes & Olshansky 2007)—is a biological certainty (Dong et al. 2016, Newman 2018). While realists do not challenge the ability of scientific advances to expand knowledge of health and reduce mortality (Carnes & Olshansky 2007, Olshansky 2013, Olshansky et al. 2009b), disagreement persists over the extent to which these innovations will enable sustained population-level increases in life expectancy.

Core demographic principles stipulate that entropy in the life table (Olshansky et al. 1990, 2001)—i.e., increases or decreases in life expectancy at birth being less sensitive to changes in death rates at older ages than at younger ages—requires more than double the level of mortality reductions to achieve a one-year increase in life expectancy today as compared with the early twentieth century (Olshansky et al. 2001). Beyond the challenge of attaining such remarkable mortality reductions, future innovations would have to target causes of death that are increasingly difficult to treat because contemporary mortality is defined by diseases of aging (Carnes et al. 2013). While scientific innovations have made and will continue to make progress in reducing

senescent mortality, the complexity of aging and death as a cumulative and idiosyncratic process makes it incredibly difficult to modify, and we should not conflate our ability to address aging-related morbidities with curing aging itself (Carnes et al. 2013). As there is no reliable centenarian lifestyle or consistent set of keys to longevity (Vaupel 2004), targeted innovations are unlikely to bring about the large-scale increases in life expectancy projected by some, or hoped for by proponents of All of Us and precision medicine. Indeed, future projections of US life expectancy are not particularly optimistic, emphasizing the potential for complex and cumulative health conditions like obesity and related metabolic disorders to offset the gains associated with eliminating key risk factors like smoking (Preston et al. 2014, 2018), leading to shorter life expectancies among future generations (Olshansky et al. 2005).

However, realists do not entirely reject the promise of innovation; the so-called artificial extension of lifespan via manufactured biomedical mechanisms may permit survival not only up to, but beyond, one's biological potential (Olshansky & Carnes 1997). Olshansky & Carnes (1997) concede that the lifespan of a population may exceed its biological limits when enough of its members benefit from these innovations—a sentiment not unlike that expressed by optimists. This alternative concession and/or consensus on the part of both biodemographic camps suggests that social rather than biological constraints are the more fundamental issue in preventing enough members of an unequal society like the United States from surviving up to (and beyond) their biological potential.

WHO BENEFITS FIRST AND MOST?

Shifting from the biodemographic question of what is possible concerning future life expectancy, we instead turn to sociological and social demographic research on who in the population is most able to realize these possibilities and thus benefits the first and most from innovations. In this vein, Link and Phelan's now-canonical fundamental cause theory (FCT) is noteworthy not only for identifying the central irony facing US population health—the continued growth of health inequalities despite our increased ability to protect health—but also for "replac[ing] the ironic connection with a causal one" in arguing these disparities have increased "in significant part *because* of remarkable advances in our ability to *prevent*, *diagnose*, *and treat disease*" (emphasis ours; Freese & Lutfey 2011, p. 68). FCT is predicated on the understanding of how social inequality creates the circumstances placing individuals at risk of risks. The flexibility built into FCT permits the respecification of the definition and mechanisms of action through which social inequality operates over time, while highlighting how individuals' social status remains unyielding in its effect on health and mortality (Clouston & Link 2021, Link & Phelan 1995, Phelan & Link 2015, Phelan et al. 2004, Phelan et al. 2010).

This simultaneous flexibility and time invariance are critical for anticipating changes to life expectancy at the intersection of human biology, innovations, and inequality. Carpiano et al. (2008) lay out the process by which the socioeconomic status (SES)-health association persisted throughout the nineteenth, twentieth, and twenty-first centuries, as infectious diseases were replaced by chronic diseases as the leading causes of death, and technological developments led to the detection and treatment of these diseases, further reducing mortality. Though life expectancy increased for all groups, socioeconomic disparities persisted. Carpiano et al. (2008) stress that this is consistent with FCT's prediction that disparities emerge when we gain control over health conditions, as the benefits of these innovations are concentrated among the most advantaged.

Critically, social (dis)advantage is not exclusively a function of SES; individuals' race—as experienced through the historically engrained and systemic racism that shapes their day-to-day lives—is a key fundamental cause of US health disparities. In their comprehensive review of the

mechanisms connecting racism to health, Phelan & Link (2015) note the significance of innovation as a key pathway through which racial disparities in health and longevity are perpetuated over time. As new mechanisms emerge to replace old ones due to social and scientific changes, such as new knowledge about the prevention or treatment of a disease, White adults observe a mortality advantage over their Black counterparts, regardless of SES.

Clouston et al. (2016) elaborate on this process in identifying the growth of cause-specific mortality disparities as an important stage in population health, occurring shortly after a disease has taken hold and initial knowledge has been obtained toward its treatment or mitigation. While this knowledge eventually disseminates throughout the population, this process of diffusion is highly inefficient, leading to long-term inequalities in mortality risk (Clouston et al. 2016). Indeed, empirical tests of FCT demonstrate that the relationship between social status and mortality is especially pronounced when the changing preventability of causes of death is taken into consideration. Masters et al. (2015) contend that socioeconomic gradients in mortality are larger for causes of death under greater human control, as personal resources can be used to attain health-relevant knowledge, services, and interventions. Likewise, Hayward et al. (2015) assert that only very highly educated individuals have truly been able to capitalize on the technological advances that contribute to health and longevity gains in recent decades.

Consequently, researchers consistently find that the association between SES, race/ethnicity, and mortality risk is stronger for more preventable causes of death than less preventable causes (Elo et al. 2014, Hummer & Lariscy 2011, Macinko & Elo 2009, Masters et al. 2012, Phelan et al. 2004, Rubin et al. 2014, Tehranifar et al. 2009, Warren & Hernandez 2007). Given these inefficiencies and the consistent re-creation of disparities regardless of cause of death, Freese & Lutfey (2011) shrewdly note that for sociologists studying the interplay between inequality and innovation, technological advancement should raise concerns about the social conditions that lead to more opportunities for those at the top rather than better prospects for those at the bottom.

Diffusion of Innovations

More broadly, empirical assessments of FCT invoke differential access to and benefit from innovations as a primary pathway through which amenable and preventable causes of death are particularly sensitive to individuals' social status, given that groups who are richer in resources benefit most from advancements in controlling disease (Phelan & Link 2005). This inequitable social diffusion of innovations—or "the process by which a novel development is communicated over time among the members of a social system" (Korda et al. 2011, p. 224)—accounts for much of the socioeconomically and racially graded relationship between innovations and health. Assuming an innovation is effective, it initially reaches only a few, select members of society prior to more rapid uptake among the broader population; however, this rate of diffusion varies among different subgroups, such that new innovations increase disparities by initially affecting more advantaged individuals until greater diffusion is attained (Korda et al. 2011).

Glied & Lleras-Muney (2008) find strong empirical support of SES gradation in technological diffusion across a number of causes of death and different health innovations over time. Speculating as to the mechanisms by which innovations mediate the relationship between education and health, the authors posit that more-educated adults have greater access to information about, and thus place greater value on, the benefit of innovations, which contributes to their status as early adopters. They are likely to have access to higher-quality providers using newer technologies and greater propensity to seek out specialist care. Of particular salience to preventable mortality, the advantage more-educated individuals have in access to new technologies outside of a medical context is likely to enhance their ability to follow complex protocols and tolerate side effects.

Research by Goldman & Lakdawalla (2005) substantiates this latter explanation, finding that a more complicated treatment regimen like antiretroviral HIV therapy was disproportionately beneficial to well-educated patients. Treatment success was highly dependent on individuals' ability to comply with the difficult medication protocol; higher-SES adults' social advantages allow for greater compliance relative to their lower-SES counterparts. Rubin et al. (2010), also studying social gradients in HIV/AIDS mortality, expand this line of reasoning to include a variety of mechanisms underlying observed disparities, such as knowing about or living near treatment, initially receiving correct and actionable medical advice, and having support and being treated equitably and respectfully throughout the treatment process. Critically, their work—along with similar findings from Elo et al. (2014) and Levine et al. (2007)—emphasizes how both SES and race are subject to inequalities in these mechanisms, thereby creating disparities in mortality.

This basic framework for understanding the relationship between social status, innovation, and health can be observed across a variety of medical advances in recent years. Chang & Lauderdale (2009) show how the introduction of cholesterol-lowering statins contributed to a reversal in the income gradient, such that more advantaged adults transitioned from having higher cholesterol than their lower-SES counterparts to having far lower rates. Even though statins improved population health overall, they created new links between social factors and disease (Chang & Lauderdale 2009). Cancer screening is among the most critical of these new links. For instance, Saldana-Ruiz et al. (2013) document a reversal in the association between SES and colorectal cancer mortality with the advent of better screening, noting how a disease once associated with affluence became concentrated among poorer adults (Saldana-Ruiz et al. 2013, p. 102). Link et al. (1998, p. 396) also find that the development of improved cancer screening technologies and protocols led to the emergence of "new SES-linked protective factors" and thus new gradients in mortality.

Unsurprisingly, studies have documented pronounced and widening SES and racial/ethnic disparities in mortality across many types of cancer that have become more preventable due to innovations in cancer detection and care (Rubin et al. 2014; Tehranifar et al. 2009, 2016; Wang et al. 2012), as well as for precursors to future cancer risk like human papillomavirus vaccination (Polonijo & Carpiano 2013). Given that systematic racism structures individuals' interactions with the health care system, neighborhood characteristics, and access to social and economic resources, Black adults have lower access to and worse quality of cancer screenings and treatment (Hunt et al. 2014; Levine et al. 2008, 2010; Soneji et al. 2010; Tehranifar et al. 2009, 2016), and are less likely to have critical preventative knowledge (Polonijo & Carpiano 2013). While most studies focus on Black-White disparities, there is evidence to suggest that minority racial/ethnic groups like American Indians/Native Americans and Hispanics also fare much worse in preventable cancer mortality relative to their White counterparts (Tehranifar et al. 2009, Tehranifar et al. 2016). More broadly, the work of Elo and colleagues clearly shows that much of the life expectancy gap between Black and White adults is attributable to causes of death directly amenable to medical care (Elo et al. 2014, Macinko & Elo 2009), such that reducing disparities in access to treatment would lead to a two-year reduction in this gap.

Given these well-documented disparities in mortality, FCT is justifiably cautious with respect to innovation as a singular positive force for improving population health. However, this does not preclude such innovations from eventually improving life expectancy. Link et al. (1998) acknowledge that medical advances often improve health, and inequalities in the benefit of an innovation are unintentional. More pointedly, Schnittker & Karandinos (2010) argue that too much focus in sociology on the upstream causes of mortality has contributed to the unfortunate conclusion that more proximate mechanisms like medical innovations are insignificant. Given that pharmaceutical innovations account for a substantial reduction of deaths in the latter half of the twentieth century,

the authors conclude that technological innovations will continue to improve outcomes, especially in high-demand areas like chronic disease. Similarly, Cutler et al. (2006) are almost indistinguishable from optimist biodemographers in identifying continued gains in knowledge, science, and technologies as the keys to declining mortality. While acknowledging that the changing nature of these innovations creates temporary health disparities, they maintain that a silver lining to social gradients in innovations is that "help is on the way, not only for those who receive it first, but eventually for everyone" (Cutler et al. 2006, p. 117).

Nevertheless, an outstanding concern is that this period of convergence when the rest of the population benefits from innovations is only temporary, at which point a new innovation appears and divergence in access and health inevitably reoccurs (Vallin & Meslé 2004). Indeed, the evidence reviewed above suggests that innovations will continue having a disproportionate impact on reducing mortality among the most advantaged members of society, thus maintaining or widening the SES-mortality gradient throughout the twenty-first century (Hayward et al. 2015, Miech et al. 2011, Warren & Hernandez 2007) and contributing to the perpetuation of racial/ethnic-mortality gradients (Phelan & Link 2015). Previous gains in life expectancy were largely made possible by the successful translation of innovations into population-level interventions (Cutler et al. 2006, Cutler & Miller 2005, Vallin & Meslé 2004, Omran 1971). As this translational process is delayed and/or diminished, the importance of individuals' access to continuous medical interventions for delaying mortality in the twenty-first century will only increase further. In turn, the growing concern is not the ability of new innovations to extend life, but the extent to which continued reliance on these innovations deepens existing inequalities and mitigates changes in population life expectancy.

LIFE EXPECTANCY TRAJECTORIES IN THE TWENTY-FIRST-CENTURY UNITED STATES

Though continued medical innovations and increase in life expectancy anticipated by optimists remains possible, sociological and demographic research challenges this narrative. Recent increases in US adult mortality have been concentrated among the least advantaged members of society, who are least able to capitalize on such innovations. While Case & Deaton's (2015, 2020) work on increasing mortality among middle-aged White adults highlighted the surprising increase in mortality among lower-educated adults—leading to decreases in overall US life expectancy—social demographic research has consistently documented the widening of socioeconomic inequalities in mortality during the last two decades of the twentieth century and the first two decades of the twenty-first century (Crimmins & Zhang 2019).

Notably, Elo's (2009) review of the many mechanisms connecting social class to mortality concludes that the widening of educational differentials toward the close of the twentieth century is best explained by the importance of education in increasing one's ability to take advantage of new innovations and to change behavior in response to public health messaging. Income exacerbates inequality further, as overall income inequality in a society augments existing status differences in health by strengthening the mechanisms through which social class affects individuals' lives (Pickett & Wilson 2015). Widening socioeconomic inequality in the United States from the 1970s onward—with high income and wealth, in particular, increasingly concentrated among a smaller proportion of the population (Piketty & Saez 2014, Reardon & Bischoff 2011)—has foreshadowed a parallel trajectory of widening inequality in longevity between the most and least advantaged members of society. Socioeconomic disparities in health and longevity have always existed, but the growth of these disparities over the past 50 years has been especially pronounced

(Zajacova & Lawrence 2018), leading many to conclude that the link between increasing inequality and decreasing gains in US life expectancy is likely causal rather than correlational (NRC 2011, NRC 2013).

The increasing divergence of life expectancy within the United States in the early twenty-first century is especially dramatic, with numerous studies documenting stratification along socio-economic lines, further intersecting with individuals' demographic characteristics and geographic contexts (Chetty et al. 2016; Dwyer-Lindgren et al. 2017; Hayward et al. 2015; Masters et al. 2012; Miech et al. 2011; Montez & Zajacova 2013; Montez et al. 2011, Montez et al. 2019; Sasson 2016b; Sasson & Hayward 2019). Collectively, these studies suggest that the recent lack of gains in US life expectancy was less unforeseen and more of an inevitability amid growing social inequality.

The absolute magnitude of disparities in US life expectancy is immense. Sasson (2016b) finds that the gap in life expectancy at age 25 between low- and college-educated Whites nearly doubled for men and tripled for women between 1990 and 2010, reaching 11.9 and 9.3 years, respectively; similar patterns are true among Black Americans, for whom the educational gap is more than 8.6 years for men and 4.7 years for women. This divergence is consistent for income; Chetty et al. (2016) document a 10-year gap in life expectancy at age 40 between women in the top and bottom 1% of the income distribution, and a nearly 15-year gap among men. Recent research also documents significant declines and/or stagnation in life expectancy among various subgroups in the United States. Montez & Zajacova (2013) and Sasson (2016b) both find declines in life expectancy among White adults, by as much as 3.1 years among low-educated women, and 0.6 years for men. Likewise, Chetty et al. (2016) find continued increases in longevity for individuals in the top 5% of the income distribution (2.34 and 2.91 years for men and women, respectively), as compared with virtually no change among individuals in the bottom 5% (0.32 and 0.04 years for men and women, respectively).

Larger Losses at Younger Ages

Perhaps most troublingly, recent research on US mortality emphasizes the widening disparities among younger Americans as the driving force behind diverging life expectancies. Innovations may reduce biologically rooted mortality at older ages, but mortality at younger ages is almost exclusively a product of social determinants (Braudt et al. 2019, Gillespie et al. 2014, Rogers et al. 2020). Years of data and evidence definitively show how social inequality—in the form of both socioeconomic and racial/ethnic stratification—leads to countless lost years of life at younger ages in the United States, accounting for much of its lower life expectancy relative to other wealthy nations (NRC 2013).

Many recent studies have reached a similar conclusion concerning the disproportionate impact of early deaths on US life expectancy (Ho & Preston 2010, Ho & Hendi 2018, Vaupel et al. 2011). Most strikingly, Rogers et al. (2020) find that compared with its peer countries, the United States has 60% higher age-specific mortality among adults in their 20s, and it is the only high-income nation where 1% of deaths occur before age 20 and 10% of deaths occur before age 60. While the United States is similar to its peers in reducing chronic disease mortality among older adults, Shkolnikov et al. (2011a) single out higher US mortality at younger ages from causes of death linked to inequality—such as homicides, drug overdoses, accidents, and communicable diseases (NRC 2013)—as the leading explanation for the life expectancy gap between the United States and its peers.

Because causes of death at young ages are primarily attributable to socially determined behavioral and lifestyle factors, researchers emphasize how harmful, if not outright dangerous, aspects

of US culture and policy contribute to its laggard status in early life mortality. The popularity and availability of firearms, greater reliance on personal transportation, and engrained social and political values that prioritize private enterprise and individual choice (e.g., fewer regulations on workplace safety and seat belt use) are just some of the likely explanations (NRC 2013). Higher socioeconomic inequality in the United States concentrates these preventable deaths among poorer and less highly educated adults, as well as children and adolescents in their families (Braudt et al. 2019, Gillespie et al. 2014, Miech et al. 2011, van Raalte et al. 2014); the racial dynamics of these deaths are more complicated given that White youth and adults tend to have higher drug-related and suicide mortality, while their Black and Hispanic counterparts are overrepresented in homicide and other violent causes (Heron 2019). Nevertheless, higher mortality for all young people—regardless of cause of death or which group is more affected—leads to lower life expectancy in the country as a whole.

Re-Emerging and Extrinsic Causes of Death

The sensitivity of life expectancy to inequality and mortality at younger ages is particularly worrisome in light of the rise in extrinsic causes of death among US young and middle-aged adults. Even if improvements among leading intrinsic causes of death continue at a steady pace—to the extent that they may one day be fully preventable—recent mortality trends suggest that a halcyon era of unrestricted growth in life expectancy remains out of reach. Despite sustained progress in reducing chronic disease mortality (Ma et al. 2015), the rise in deaths from drug overdoses and other extrinsic causes—such as emerging pandemics—among disadvantaged groups represents a new pathway through which social inequality will challenge improvements to US life expectancy in coming decades (Natl. Acad. Sci. Eng. Med. 2021).

In general, rising causes of death are concentrated among lower-SES adults (Miech et al. 2011), as is consistent with FCT (Clouston & Link 2021). Preceding Case and Deaton's (2015) study on increases in midlife mortality among low-educated Whites, Miech et al. (2011) singled out accidental poisonings as most emblematic of these patterns. Increases in prescription opioid availability and use led to a substantial increase in accidental poisoning risk in recent years; in turn, lower-educated adults were most vulnerable to this emerging risk factor, leading to accidental poisonings exhibiting a very wide educational gradient in adult mortality. Although Miech et al. (2011) were among the first to sound the alarm, the visibility of Case & Deaton's (2015) paper was instrumental in providing empirical evidence for the massive toll of rising external mortality among low-educated White adults over the past 20 years.

The rise in drug overdoses—i.e., the opioid epidemic—has had an outsized effect on life expectancy among socially disadvantaged adults and in the United States as a whole (Glei & Preston 2020). Studying the role of drug overdoses in widening educational gradients in life expectancy between 1992 and 2011, Ho (2017) finds that overdoses account for roughly 16% to 18% of the difference in life expectancy between middle-aged White high school and college graduates by 2011. Masters et al. (2018) similarly emphasize the importance of drug overdoses for rising mortality, particularly the increasing availability, overprescription, and abuse of opioid-based painkillers, combined with increases in heroin use and its disproportionate impact on vulnerable groups. More broadly, Sasson (2016a) notes that, between 1990 and 2010, external and residual causes of death have accounted for an increasing number of years of life lost among all education groups except college-educated adults. Unsurprisingly, the lowest-educated adults saw the largest absolute and relative loss of years of life, offsetting any of their minor gains in life expectancy attributable to declines in cardiovascular disease and cancer mortality and further widening the educational gap in life expectancy (Sasson 2016a).

Critically, while much of this work initially centered on the impact of drug overdoses on White women and men, recent evidence finds similar patterns among Black adults (Alexander et al. 2018, Sasson & Hayward 2019). Research by Woolf et al. (2018) further shows that virtually all major race/ethnic groups have seen increased midlife mortality from alcohol-related liver disease, suicides, and drug overdoses in the past 5–10 years. Nevertheless, the question of why race and ethnic differences in these deaths are observed remains of interest. A full examination of how race and inequality interact to shape health and social outcomes is beyond the scope of this review; however, recent work by Malat et al. (2018) and Metzl (2019) provides a much-needed framework for understanding how decades of policies and behaviors designed to maintain White supremacy in the United States actively undermine the health of lower-SES White adults. Namely, resistance to social welfare policies that would benefit all members of society, regardless of race, and embedded narratives of White victimhood appear to be at play in the recent trend of declining physical and mental health that underlies many of these despair-associated deaths.

The opioid epidemic is particularly notable, as it is emblematic of the unforeseen threats to future gains in life expectancy discussed by both optimists and realists (Carnes & Olshansky 2007, Vaupel 2010). Perhaps no threat was less foreseen than the ongoing coronavirus disease 2019 (COVID-19) pandemic, which had accounted for more than 500,000 US deaths as of March 2021; its impact on life expectancy is yet unknown, but early estimates suggest a reduction in US life expectancy at birth of 1.13 years, to 77.48 years, which is lower than any year since 2003 (Andrasfay & Goldman 2021). Moreover, hard-hit areas like New York City may experience as much as a five-year decrease (Prevent Epidemics 2020). The emergence of COVID-19 is truly unexpected from an epidemiologic transition perspective, given the historical shift toward noncommunicable diseases as leading causes of death (Omran 1971). Yet there is nothing unprecedented about the social patterning of its impact on US mortality. While one message surrounding this catastrophe is that it is impacting everyone, an egalitarian crisis is a myth in a fundamentally unequal society. Evidence on the socioeconomic and racial/ethnic disparities associated with COVID-19 is still nascent, but extant sociological knowledge suggests that the disproportionate impact on lower-SES and racial/ethnic minority adults is inevitable (Clouston & Link 2021). Their limited opportunities and higher risk—with respect to exposure in both leaving home for work and living in more dense areas (Reeves & Rothwell 2020), as well as underlying vulnerability in terms of having preexisting conditions (Elo 2009, Williams 2012)—emphasize the luxury inherent to physical distancing and saving safer at home.

Indeed, early data on the racial and ethnic inequality in COVID-19 highlight the mounting toll on the Black population and other minority groups. Rates of infection and mortality are disproportionately higher in Black communities and other communities of color (Hooper et al. 2020, Laurencin & McClinton 2020, Yancy 2020), to the extent that the CDC (2020) has designated non-White adults as a group who needs extra precautions on account of "long-standing systemic health and social inequities [that] have put some members of racial and ethnic minority groups at increased risk of getting COVID-19 or experiencing severe illness." Key social risk factors like household composition and employment are especially pertinent with respect to contracting COVID-19: Black adults at high risk of illness are 1.6 times as likely as Whites to live in households with health-sector workers, while almost two-thirds of high-risk Hispanic adults live in households with at least one worker unable to work at home, compared with less than half of White adults (Selden & Berdahl 2020). Likewise, social mechanisms influence the likelihood of experiencing severe illness and/or mortality: Decades of research underscore the role of both intrapersonal and institutional discrimination and marginalization in eliciting stress processes that contribute to premature aging and elevated chronic disease risk among non-White adults (Geronimus et al. 2006, Levine & Crimmins 2014, Williams 2012). Given the totality of these racially and ethnically stratified exposures and vulnerabilities to COVID-19, emerging research suggests that the pandemic will reverse years of progress in reducing the Black-White mortality gap, which is estimated to increase by 40%, from 3.6 years to over 5 years (Andrasfay & Goldman 2021). This study also estimates a decrease in the Hispanic life expectancy advantage over White adults, down from 3 years to less than 1.

Critically, the far-reaching consequences of COVID-19 are still unknown, especially in impacting future life expectancy among those who survive but are left with permanently disabling physical and mental health conditions affecting future health and social outcomes. Nor are these health consequences restricted to those infected; the psychosocial and economic ramifications of increased morbidity and mortality among family, friends, and members of one's community are likely to have a disproportionate effect on minority and low-SES groups for years to come (Donnelly et al. 2020, Umberson 2017, Umberson et al. 2017), exacerbating the systemic flaws that make a novel risk like COVID-19 map so neatly onto existing inequalities.

Scientific innovation will hopefully eliminate the immediate threat posed by COVID-19, but numerous other risks contribute to diverging trajectories of US life expectancy. Homicides, suicides, violence, and accidents continue to be overrepresented among low-SES adults at young ages (Bijwaard et al. 2017, Braudt et al. 2019, Dollar et al. 2020, Miech et al. 2011, van Raalte et al. 2014), imposing a social limit on the magnitude of possible gains to US life expectancy. Sweden serves as a case study of how extrinsic causes of death continue to shape SES disparities in life expectancy, even in a wealthy, technologically advanced society with far lower social inequality than the United States. Namely, Bijwaard et al. (2017) find that reducing differences in educational attainment among Swedish men would result in a 22% reduction in person-years lost to death between the ages of 18 and 64. While reductions in cancer, cardiovascular disease, and other disease-specific mortality account for a large proportion of this reduction, the greatest proportion (28%) is attributable to reduced mortality from extrinsic causes. Essentially, in a near best-case scenario that innovations permit reductions in US disease mortality such that it resembles a more egalitarian society like Sweden, substantial inequality in life expectancy is likely to remain.

Indeed, many researchers envision such a best-case world in which biophysiological senescence and intrinsic mortality are eradicated, and largely background mortality remains (e.g., accidents, violence, infectious disease [Bongaarts 2006]). However, this counterfactual reality presents its own challenges to increasing life expectancy. The elimination of one form of risk for mortality inevitably precedes its substitution via another (Miech et al. 2011, Vallin & Meslé 2004)—many of which are increasingly unexpected. Yet the inequitable impact of these risks is entirely predictable in perpetuating existing disparities in life expectancy. Thus, rather than hypothesizing this disease-free ideal, one need only examine contemporary mortality trends in the United States to understand how extrinsic causes of death—largely outside the realm of scientific or medical innovation—lead to declines in life expectancy among the most disadvantaged, and sometimes even the population as a whole.

CONCLUSION

Research on longevity is often premised in the study of outliers, from whom we seek the keys to unlocking long life (Vaupel 2004) that will advance knowledge and engender innovation. Yet, this research is inadequate without understanding who the recipients and beneficiaries of this knowledge and innovation are in an increasingly stratified society like the United States. Beyond questions of "What is possible?" and "How do we make it possible?", sociological theory and evidence necessitate the critical addendum of "For whom?"

The past 20 years of research have consistently documented the widening of socioeconomic gradients in mortality during the first two decades of the twenty-first century, including for causes of death among which rates had been on a persistent decline over the course of the twentieth century owing to scientific and medical advances, such as heart disease, cancer, and diabetes (Chetty et al. 2016, Clouston et al. 2016, Hayward et al. 2015, Masters et al. 2012, Miech et al. 2011, Phelan et al. 2010). Trends in life expectancy among the population as a whole do not necessarily reflect the health and mortality experiences of key sociodemographic groups, some of which have seen declines far earlier than the national-level decrease observed in recent years (Montez & Zajacova 2013). Perhaps most worrisome, increased variability in life expectancy—while receiving less attention than declines—has been extensively documented by social demographers as evidence of a growing rift in the longevity prospects for different segments of the US population (Brown et al. 2012, Crimmins & Zhang 2019, Gillespie et al. 2014, Rogers et al. 2020, Sasson 2016b, Shkolnikov et al. 2011a).

The more optimistic research on the future of life expectancy nevertheless invokes science, medicine, and technology as the primary means by which we delay the onset of aging, biological senescence, and, ultimately, death (Rae et al. 2010, Vaupel 2004, Vaupel 2010)—assuming the rapid pace of continuous innovation that has improved health in the past will continue unabated in years ahead (Bongaarts 2006, Burger et al. 2012, Rau et al. 2008, Shkolnikov et al. 2011b, Vallin & Meslé 2009). However, sociological research forewarns that the impact of innovations is far from egalitarian and unlikely to address existing disparities in life expectancy. FCT demonstrates how the diffusion of innovations exacerbates mortality disparities, proving robust to the dynamic nature of both innovations and causes of poor health over time (Freese & Lutfey 2011, Glied & Lleras-Muney 2008, Korda et al. 2011, Link et al. 1998, Masters et al. 2015, Phelan & Link 2015, Timmermans & Kaufman 2020). Recent trends in adult mortality further emphasize this point, indicative of how future projections of US life expectancy may come to reflect the amplified significance of non-age-related, premature causes of death—most notably, drug overdoses and infectious diseases—stratified along socioeconomic and racial/ethnic lines. In such a scenario, it is reasonable to assume that innovations contribute little to extending longevity, as the etiology of mortality becomes increasingly less amenable to biomedical intervention. If past is prologue—as is the argument in favor of innovations as a source of future gains to life expectancy—then observed social gradients in the uptake and benefit of innovations point to persistent, if not increasing, inequality in life expectancy among future generations.

Taken together, emerging biodemographic and social demographic research reveals a paradoxical state of affairs regarding the future of US life expectancy. As we continue to advance understanding of how much progress is attainable on the basis of our biological potential for longevity—and the ability for innovations to help realize this potential—progress in increasing life expectancy in the short term appears to have stalled entirely due to social factors and the increasing bifurcation of social opportunities and health in our society. This intersection of biological and social forces cannot be overlooked because it clearly emphasizes the extent to which social inequality imposes, and will continue to impose, limits to US life expectancy above and beyond the gains attributable to progress in the realms of science, technology, and medicine.

Ultimately, this review points toward the emergence of competing narratives concerning the allocation of gains to life expectancy in coming decades, largely preempted by these early twenty-first-century trends. A select portion of the US adult population—namely, White adults with high educational attainment, the skill set necessary to participate in an increasingly knowledge-based economy, and adequate financial resources—will continue to be the immediate and perpetual beneficiaries of humanity's near-complete control over environmental and health risks, reaping the immense benefits of continued innovations. Currently pushing up against the boundaries of

human longevity, they are most likely to capitalize upon the much-heralded antiaging and life-extending innovations of the future, potentially attaining a near-perfect biological state (Kurzweil & Grossman 2010, Mykytyn 2010, Olshansky 2016). The hope is that these gains in longevity will eventually diffuse into the broader population, and all may benefit from a world in which aging is all but cured. Indeed, elements of this narrative recur through much of social history; innovations in science and technology first impact the beliefs and behaviors of an elite few before diffusing to the population at-large, as evidenced by the mortality and fertility changes observed during the demographic transition (NRC 2001). Yet, a less utopian view of future life expectancy provides a starker account. Instead, the disadvantaged members of society—perpetually disenfranchised on the basis of their SES and/or race/ethnicity—enter a new stage of the epidemiologic transition in which the immense benefits attributable to scientific, technological, and medical advances in the twentieth century taper off and epidemics of chronic pain, morbidity, and infectious disease are increasingly concentrated at the lower end of the social hierarchy. In reality, these are not necessarily competing narratives; rather, they are parallel narratives reflecting how opportunity, innovation, and health already interact in the contemporary United States.

Though we, along with many researchers, stress the need for fundamental social reforms in addressing current and anticipated disparities in life expectancy, the scientific pursuit of better understanding of the biological foundations of human longevity should not and cannot be ignored. Better knowledge of human aging, senescence, and mortality allows for improved models of population change and development. Better models beget better forecasts, which are vital in anticipating the social, economic, and health needs of present and future cohorts. By the same token, this core biodemographic knowledge paves the way for science, technological, and medical innovations that can reduce the burden of aging, prevent and/or eliminate diseases, and improve population health as a whole, as has been the case throughout much of the past two centuries of human history.

However, decades of social scientific research confirm the basic empirical fact that human longevity is not context free. Much of our knowledge about mechanisms of longevity is derived from nonhuman species that are studied in highly restricted and controlled scientific environments. Yet the social environment in which humans reside is complex and highly stratified. Although the biological potential for a long life may be stochastically distributed throughout the population, individuals' opportunities to capitalize upon this potential are not; health and wellbeing are highly correlated with one's position in the social hierarchy. Consequently, high-status individuals endowed with an unfavorable biological predisposition for early mortality may leverage their many resources to extend their lives for many years beyond their predestined lifespan. Conversely, cumulative disadvantage throughout the life course may contribute to the premature death of low-status individuals with the potential to live well into their centenarian years. The many stakeholders in the life expectancy crusade—researchers, corporations, and policy-makers alike—should avoid a narrow-minded focus on either the biological or social determinants of a long, healthy life. As decades of research show, the value of our increasing biophysiological knowledge—and the extent to which science, technology, and medicine help to make this knowledge actionable—can only be meaningful and beneficial when it reaches those who need it most in a highly unequal society.

DISCLOSURE STATEMENT

The authors are not aware of any affiliations, memberships, funding, or financial holdings that might be perceived as affecting the objectivity of this review.

ACKNOWLEDGMENTS

This research was supported by the Eugene Kennedy Shriver National Institute of Child Health and Human Development of the National Institutes of Health by training grant T32 HD007168 and infrastructure grant P2C HD050924, both awarded to the Carolina Population Center at The University of North Carolina at Chapel Hill. This research was also supported by the National Institute on Aging of the National Institutes of Health by infrastructure grant P30 AG066615, awarded to the Carolina Population Center, and by network grant R24 AG045061. We would also like to thank Kathleen M. Harris, an anonymous reviewer, and the Co-Editors for their helpful feedback.

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