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Research Article

Socio-behavioral factors contributing to recent mortality trends in the United States

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Samuel Preston¹ Yana Vierboom² Mikko Myrskylä³

Abstract

METHODS

We investigate the contribution of socio-behavioral factors to changes in US adult mortality over the period 1997–2019 using National Health Interview Surveys for the years 1997–2018 linked to death records through 2019. The variables studied include alcohol consumption, cigarette smoking, health insurance coverage, educational attainment, mental distress, obesity, and race/ethnicity. We evaluate the contribution of each socio-behavioral variable to mortality change by estimating the mortality risks associated with each variable in a hazards model and applying the risks to changes in the variable's distribution.

RESULTS

When all variables are included in the model, we find that reductions in cigarette smoking and increases in educational attainment are the largest contributors to recent mortality improvements, accounting between them for 66% of mortality improvements. The contribution of educational attainment nearly doubles when variables that may be considered downstream to it are omitted from the analysis. In a secondary analysis, we compare two subperiods to investigate whether the variables can account for a widely observed slowdown in the rate of mortality reduction that occurred *within* the period of study. Rising levels of psychological distress, combined with very high risks associated

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with distress, contributed substantially to the slowdown. However, most of the slowdown remains unaccounted for.

CONTRIBUTION

We develop and apply a rigorous method to identify the role of many socio-behavioral factors in recent mortality change in the United States. Results highlight the role of a behavioral change (declines in smoking) and a broad social advance (educational expansion).

1. Introduction

Hundreds of factors affect levels of mortality in every population. Changes in the prevalence or fatality of these factors, and in how they combine, produce changes in a population's level of mortality. These trends in mortality have important implications for quality of life, social and family relationships, and the fiscal viability of age-based programs of economic transfer (Trustees 2022).

There are several approaches to identifying the various factors that influence mortality trends in large populations. One straight-forward approach uses cause-of-death assignments on death certificates (e.g., Woolf and Shoomaker 2019). Since some underlying causes of death are the product of well-recognized causal processes – such as motor vehicle accidents, death in childbirth, and lung cancer – changes in these causes of death contain a causal attribution that may satisfy the standards of the investigator. This approach is less informative for causes of death that are multifactorial and are the outcome of many, often overlapping causal processes. The majority of deaths fall into this category, including deaths from most cancers, cardiovascular diseases, and dementias. Nevertheless, cause-of-death assignments in these cases are still useful in limiting the range of search for causal factors.

A second approach measures the distributions of various mortality-influencing factors at two points in time. By using information on the mortality risks associated with each factor, the approach can estimate the contribution of changes in the distribution of that factor to mortality change. An analysis may focus on a behavioral risk factor such as smoking or obesity (e.g., Stewart and Cutler 2015) or elements of medical services and medical technology, such as patterns of pharmaceutical use (e.g., Buxbaum et al. 2020). Estimates of prevalence and relative risks are typically made independently and are sometimes based on different data sources, which may introduce biases when different classification systems or sampling strategies are used in the different sources (Flegal, Panagioutou, and Graubard 2014). Some studies have been able to derive estimates of mortality trends, prevalence of risk factors, and relative risks from a single data source (Deeg et al. 2013; Preston, Vierboom, and Stokes 2018).

In this paper, we use data from a single source, the National Health Interview Survey (NHIS), to identify factors contributing to mortality trends in the United States at ages 25–84. The NHIS is the largest national data source on individuals and their health behaviors and circumstances. Our principal goal is to identify the effects of various sociobehavioral factors on trends in mortality during the period 1997–2019. In pursuit of this goal, we identify (1) the *mortality risks* associated with a socio-behavioral factor; (2) changes in the distribution of that factor; and (3) the mortality trend produced by the risk combined with the distributional changes.

The period 1997–2019 begins with the availability of important variables in the NHIS and ends in the last year for which the surveys were linked to death reports. Coincidentally, the period ends just before the outbreak of the COVID-19 epidemic in 2020 and the tumultuous mortality changes that it introduced. The period 1997–2010 is one of relatively steady improvements in mortality in the United States (Kochanek et al. 2019: Figure 1), while the rate of mortality improvement during 2010–2019 was considerably slower than during the earlier period (Xu et al. 2021). Official estimates of life expectancy at birth for the nation show a value of 76.5 years in 1997 and 78.7 years in 2010, rising minimally to 78.8 years in 2019 (Aries and Xu 2022). A secondary goal of the paper is to identify whether the factors considered in this paper can account for reductions in the rate of mortality decline *during* the period under study.

2. Data and methods

2.1 Data

We use annual waves of the NHIS for years the 1997–2018, made available by IPUMS (Blewett et al. 2017). The NHIS is a cross-sectional survey that is nationally representative of non-institutionalized civilians of all ages living in the United States. The National Center for Health Statistics has linked respondents from surveys from 1997–2018 to death records through 2019, allowing for longitudinal mortality follow-up. For more information, see the survey's website (CDC 2022).

2.2 Analytic approach

Our goal is to assess how population-level changes in socio-behavioral factors contributed to mortality trends between 1997 and 2019. To achieve this goal, we apply a partial regression decomposition framework that combines information on the changing prevalence of a factor with the relative mortality risks associated with that factor.

Annual contribution of
change in variable to
$$= \frac{\sum_{i} (\Delta x_{i} * \beta_{i})}{\Delta_{t}}$$
(1)
change in mortality

where x_i is the proportion of the population in category *i* of the variable in the national distribution, Δx_i is change in this proportion during the period studied, β_i is the regression coefficient for category *i* from a Cox proportional hazards model identifying the relationship between a variable and mortality, and Δ_t is the mean duration between observations.

In our Cox models, we specify time since initial participation in the survey as the exposure time. Individuals remain exposed to the risk of death until they are censored by death, by reaching the end of survey linkage to death records on January 1, 2020, or by reaching age 85 (N = 565,092). To reduce imprecision resulting from changes in individual characteristics between survey and period of exposure to the risk of death – for example, some may quit smoking – we censor all individuals after five full years since initial observation. In a sensitivity analysis, we explore the use of an alternative period of censoring.

To identify distributional changes, we divide the sample into three periods. T1 includes individuals sampled during 1997–2004. T2 includes those sampled during 2005–2011, and T3 refers to those sampled during 2012–2018. We focus on distributional changes between T1 and T3 to supply the values of Δx_i in the above formula. The hazard model used to supply values of B_i in the above formula is based on the entire set of observations from 1997 to 2019.

We estimate three models. The distinction among them recognizes that the value of some variables is established in a manner that predates that of other variables. In particular, the staging of the life course produces temporal relations among variables and creates natural distinctions between upstream and downstream variables, also referred to as proximate and distal variables. We recognize three stages. Model 1 includes attributes established at birth: sex, age, and race/ethnicity. Model 2 adds to the variables in Model 1 educational attainment, a variable whose value is normally established by early adulthood. Model 3 adds to variables in Model 2 all other variables considered. These include biobehavioral risk factors of smoking, alcohol consumption, and body mass index (BMI); a measure of access to health care; and a measure of psychological distress.

The sample is limited to adults ages 25–84 who were: (1) eligible for mortality follow-up and (2) selected by the survey to answer additional health-related questions. We do not exclude individuals based on missingness on key variables. Rather, when a variable has missing values, we add a level for "unknown" to that variable. We use mortality weights for the sample population, called *mortwtsa* in IPUMS. The complete sample includes 565,092 adults and 3,047,232 person-years of follow-up.

Analyses were performed using Stata 17 (StataCorp 2021). Our Stata code is publicly available at https://github.com/yvboom/SocBehFac.

2.3 Variables

We include variables whose associations with mortality have been well established and whose distribution is sufficiently heterogeneous that the variables produce a substantial population attributable risk of death. Variables are constructed in the following way:

Calendar Year. A calendar year variable is created to identify trends in mortality that are not captured by other variables in the model. After initial assignment to a particular year at survey, the value of the year variable increases by one year for each year of follow-up. Since the Cox model predicts the natural log of the death rate, the coefficient of the year variable is the estimated annual rate of mortality decline during the period under study. When a model includes only age and year of observation, the coefficient of year is treated as the age-controlled rate of mortality change.

Age and Sex. Age measures age at last birthday and, like calendar year, increases by one year for each additional year of observation. In the NHIS, sex is treated as constant and binary.

Race/Ethnicity. Mortality differentials by race/ethnicity have been widely documented (e.g., Martin and Soldo 1997; Curtin and Arias 2019; Hooper, Napoles, and Perez-Stable 2020). Some of the differentials are a product of differences in the distribution of socioeconomic and behavioral variables among the groups, while others are a product of specific features of group membership, such as discrimination against Black people (Jackson et al. 2011) and processes of migrant selectivity among Hispanics (Markides and Eschbach 2011).

We use a conventional categorization of race/ethnicity: non-Hispanic White ("White"); non-Hispanic Black ("Black"); Hispanic; and other races.

Educational Attainment. Educational attainment has become the most prominent variable representing an individual's socioeconomic status in studies of health and mortality (Elo 2009). One advantage of the variable is that data on educational attainment are typically available for all adults, unlike data on occupation or income. A second advantage is that individuals typically complete their education in young adulthood, so the variable remains stable afterward. As a result, it is not highly vulnerable to problems of reverse causation during adulthood. Other socioeconomic variables, such as income and occupational status, can change as a result of one's health status, creating problems of statistical inference. Survey reporting of educational attainment is more reliable and complete than that of other socioeconomic variables (Hummer and Lariscy 2011).

Educational attainment reflects the stock of human capital established relatively early in life that is available to individuals throughout their life course (Elo 2009). That capital may include health-related knowledge, resources, and skills. In their summary of relevant literature, Hummer and Lariscy (2011) and Hummer and Hernandez (2013) identify powerful mortality effects associated with educational attainment.

We use what has become a standard set of categories for educational attainment: less than high school completion, high school diploma, some college, and college completion.

Smoking Status. Following a thorough account of smoking/mortality relations in the NHIS (Lariscy, Hummer, and Rogers 2018), we use the following categories of smoking status: Current smokers report smoking 100 or more cigarettes in their lifetime and now smoke every day or some days. Current smokers are further disaggregated by the number of cigarettes smoked per day (< 10, 10–19, 20–39, and 40+ cigarettes). Former smokers report smoking 100 or more cigarettes in their lifetime but none currently. Never smokers report smoking fewer than 100 cigarettes in their entire life. The NHIS question we use refers only to cigarette smoking, meaning that smokers who now use e-cigarettes are coded as former smokers in our analysis. The NHIS has collected information about e-cigarettes only since 2014, so including an e-cigarette smoking category would produce incomparability in the smoking variable over time.

Body Mass Index. For use in analysis of mortality, an individual's lifetime maximum BMI is preferred to BMI measured at survey because it is less affected by reverse causation resulting from disease-initiated weight loss (Stokes and Preston 2016a, 2016b). However, maximum BMI is not available in NHIS.

Instead we use a continuous measure of an individual's BMI units above 25.0, the beginning of the overweight range. Using continuous measures of BMI produces results that are much less sensitive to the common errors of self-reports than are those attained using a set of categorical variables (Preston, Fishman, and Stokes 2015).

Alcohol Consumption. Mortality attributable to excessive alcohol consumption has been rising (Vierboom 2020). We follow the Behavioral Risk Factors Surveillance System definition of heavy drinking, which combines measures of drinking frequency and volume (CDC 2015). Lifetime abstainers reported drinking fewer than 12 drinks in their lifetime, while former drinkers reported more than 12 drinks in their lifetime but none in the last year. Men who drank, on average, fewer than 15 drinks per week and women who drank fewer than 8 drinks per week were classified as light/moderate drinkers. Men who drank 15+ drinks per week and women who drank 8+ drinks per week were classified as heavy drinkers.

Psychological Distress. Several studies have linked psychological distress to subsequent mortality (e.g., Keyes and Simoes 2012; Gilman et al. 2017). A prominent explanation of rising mortality among middle-aged White people uses cause-of-death assignments to identify rising mortality from "deaths of despair," a category that includes deaths from suicide, drug poisoning, and alcoholic liver disease (Case and Deaton 2017, 2020, 2022). Despair is not a well-recognized psychological construct, but it is related to depression and anxiety, for which widely validated scales exist and which have been included in the NHIS since 1997. We use the K-6 scale combining depression and anxiety

to capture "non-specific psychological distress" (Kessler et al. 2002; Lace et al. 2020). Three of the six K-6 questions tap into a layperson's concept of despair: "During the past 30 days, how often did you feel (1) so sad that nothing could cheer you up? (2) hopeless? Or (3) worthless?" With six items, each of which is scored from 0 to 4, the additive K-6 variable takes on scores of 0-24. If a respondent is missing the answer to just one of the six questions, we replace the missing value with the mean of the available five. We then use a conventional scale that trichotomizes the variable: a score of 0-4 indicates little to no psychological distress, 5-12 indicates moderate psychological distress, and 13+ indicates severe psychological distress (Tomitaka et al. 2019).

Access to Health Care. Most of the variables in the NHIS that relate to the availability of medical care combine elements of availability with elements of the respondent's health history, as illustrated by the question, "In the last 12 months have you needed but couldn't afford medical care?" We seek a measure of health care availability that does not depend on a respondent's illnesses, which we would expect to correlate with subsequent survival for reasons unrelated to availability. For this purpose, we use a dummy variable indicating whether an individual has health insurance coverage. Wilper et al. (2009) use hazard models to estimate that 45,000 annual deaths are associated with a lack of health insurance.

3. Results

Table 1 shows the distribution of population characteristics for adults interviewed in the years 1997–2004 (T1), 2005–2011 (T2), and 2012–2018 (T3). The distributions are based on respondent characteristics at baseline and are weighted to be nationally representative (using *mortwtsa* in IPUMS).

Mean age in years (std dev) 48.0 (0.1) 48.8 (0.1) 49.9 (0.1) Male 47.9 (0.1) 48.3 (0.2) 48.2 (0.2) Race NH White 74.9 (0.3) 70.3 (0.3) 66.5 (0.4) NH Black 10.9 (0.2) 11.3 (0.2) 11.7 (0.2) Hispanic 10.3 (0.2) 13.0 (0.2) 11.4 (0.3) Other 3.9 (0.1) 5.4 (0.1) 6.9 (0.2) Educational attainment < High school 14.6 (0.2) 12.7 (0.2) 10.3 (0.2) High school 32.2 (0.2) 29.9 (0.2) 26.5 (0.2) < 4 years college 27.4 (0.1) 28.0 (0.2) 29.1 (0.2)	Characteristic	1997–2004 (T1)	2005–2011 (T2)	2012–2018 (T3)
Male 47.9 (0.1) 48.3 (0.2) 48.2 (0.2) Race	Mean age in years (std dev)	48.0 (0.1)	48.8 (0.1)	49.9 (0.1)
Bace 74.9 (0.3) 70.3 (0.3) 66.5 (0.4) NH White 74.9 (0.2) 11.3 (0.2) 11.7 (0.2) Hispanic 10.3 (0.2) 13.0 (0.2) 14.8 (0.3) Other 3.9 (0.1) 5.4 (0.1) 6.9 (0.2) Educational attainment	Male	47.9 (0.1)	48.3 (0.2)	48.2 (0.2)
Kace 74.9 (0.3) 70.3 (0.3) 66.5 (0.4) NH White 74.9 (0.2) 11.3 (0.2) 11.7 (0.2) Hispanic 10.3 (0.2) 13.0 (0.2) 14.8 (0.3) Other 3.9 (0.1) 5.4 (0.1) 6.9 (0.2) Educational attainment < High school	2			
Hr Black 14.5 (0.5) 10.3 (0.5) 00.3 (0.4) NH Black 10.9 (0.2) 11.3 (0.2) 11.7 (0.2) Hispanic 10.3 (0.2) 13.0 (0.2) 14.8 (0.3) Other 3.9 (0.1) 5.4 (0.1) 6.9 (0.2) Educational attainment 14.6 (0.2) 12.7 (0.2) 10.3 (0.2) High school 32.2 (0.2) 29.9 (0.2) 26.5 (0.2) < 4 years college	Race	74.0 (0.2)	70.3 (0.3)	66 E (0 4)
Hispatic 10.3 (0.2) 11.3 (0.2) 11.3 (0.2) Hispanic 10.3 (0.2) 13.0 (0.2) 14.8 (0.3) Other 3.9 (0.1) 5.4 (0.1) 6.9 (0.2) Educational attainment 12.7 (0.2) 10.3 (0.2) 10.3 (0.2) High school 32.2 (0.2) 29.9 (0.2) 26.5 (0.2) 4 years college 27.4 (0.1) 28.0 (0.2) 29.1 (0.2)		10.0 (0.2)	11.2 (0.2)	11 7 (0.2)
Comparing Tots (0.2) Tots (0.2) Tots (0.2) Other 3.9 (0.1) 5.4 (0.1) 6.9 (0.2) Educational attainment	Hispanic	10.3 (0.2)	13.0 (0.2)	11.7 (0.2)
Educational attainment Educational attainment < High school	Other	39(01)	5.4 (0.1)	69(02)
Educational attainment 14.6 (0.2) 12.7 (0.2) 10.3 (0.2) High school 32.2 (0.2) 29.9 (0.2) 26.5 (0.2) 4 years college 27.4 (0.1) 28.0 (0.2) 29.1 (0.2)	Calor	0.0 (0.1)	0.4 (0.1)	0.0 (0.2)
< High school 14.6 (0.2) 12.7 (0.2) 10.3 (0.2) High school 32.2 (0.2) 29.9 (0.2) 26.5 (0.2) < years college	Educational attainment			
High school 32.2 (0.2) 29.9 (0.2) 26.5 (0.2) < 4 years college	< High school	14.6 (0.2)	12.7 (0.2)	10.3 (0.2)
< 4 years college 27.4 (0.1) 28.0 (0.2) 29.1 (0.2)	High school	32.2 (0.2)	29.9 (0.2)	26.5 (0.2)
	< 4 years college	27.4 (0.1)	28.0 (0.2)	29.1 (0.2)
≥ 4 years college 25.8 (0.3) 29.4 (0.3) 34.2 (0.3)	≥ 4 years college	25.8 (0.3)	29.4 (0.3)	34.2 (0.3)
Cigarette smoking	Cigarette smoking			
Never 52.1 (0.2) 55.3 (0.2) 59.1 (0.2)	Never	52.1 (0.2)	55.3 (0.2)	59.1 (0.2)
Former 24.6 (0.1) 23.7 (0.2) 24.1 (0.2)	Former	24.6 (0.1)	23.7 (0.2)	24.1 (0.2)
Current (<10/day) 6.4 (0.1) 6.9 (0.1) 6.7 (0.1)	Current (< 10/day)	6.4 (0.1)	6.9 (0.1)	6.7 (0.1)
Current (10–19/day) 5.7 (0.1) 5.8 (0.1) 5 (0.1)	Current (10–19/day)	5.7 (0.1)	5.8 (0.1)	5 (0.1)
Current (20–39/day) 9.0 (0.1) 6.7 (0.1) 4.4 (0.1)	Current (20–39/day)	9.0 (0.1)	6.7 (0.1)	4.4 (0.1)
Current (40+/day) 1.6 (0.0) 0.9 (0.0) 0.4 (0.0)	Current (40+/day)	1.6 (0.0)	0.9 (0.0)	0.4 (0.0)
Unknown 0.6 (0.0) 0.8 (0.0) 0.5 (0.0)	Unknown	0.6 (0.0)	0.8 (0.0)	0.5 (0.0)
Alcohol consumption	Alcohol consumption			
Accord consumption $20.2/0.2$ $10.4/0.2$ $17.4/0.2$	Lifetime obstainer	20.2 (0.2)	10.1 (0.2)	17.1 (0.2)
Enterine diskaner $20.2(0.2)$ 19.1(0.2) 17.1(0.2)	Enermor drinkor	20.2 (0.2)	15.5 (0.2)	14.8 (0.1)
Light/moderate drinker $55.7(0.2)$ $56.6(0.2)$ $14.0(0.1)$	l ight/moderate drinker	55.7 (0.2)	56.5 (0.2)	59.6 (0.2)
Hence drinker $68(0.1)$ $71(0.1)$ $71(0.1)$	Heavy drinker	6.8 (0.1)	7 1 (0 1)	7 1 (0 1)
$\begin{array}{cccccccccccccccccccccccccccccccccccc$	Linknown	1 3 (0 1)	19(01)	14(0.0)
	Ginalowii	1.0 (0.1)	1.0 (0.1)	1.4 (0.0)
Mean BMI above 25 (std dev) 2.8 (0.0) 3.4 (0.0) 3.7 (0.0)	Mean BMI above 25 (std dev)	2.8 (0.0)	3.4 (0.0)	3.7 (0.0)
Health insurance coverage	Health insurance coverage			
Yes 86.4 (0.1) 84.1 (0.2) 87.7 (0.2)	Yes	86.4 (0.1)	84.1 (0.2)	87.7 (0.2)
No 13.4 (0.1) 15.7 (0.2) 11.9 (0.1)	No	13.4 (0.1)	15.7 (0.2)	11.9 (0.1)
Unknown 0.3 (0.0) 0.2 (0.0) 0.4 (0.0)	Unknown	0.3 (0.0)	0.2 (0.0)	0.4 (0.0)
Mental distress	Mental distress			
None (K-6: 0-4) 81.1 (0.2) 80.8 (0.2) 77.7 (0.2)	None (K-6: 0–4)	81.1 (0.2)	80.8 (0.2)	77.7 (0.2)
Moderate (K-6: 5–12) 14.6 (0.1) 15.0 (0.1) 16.0 (0.1)	Moderate (K-6: 5–12)	14.6 (0.1)	15.0 (0.1)	16.0 (0.1)
Severe (K-6: ≥ 13) 3.1 (0.1) 3.3 (0.1) 3.5 (0.1)	Severe (K-6: ≥ 13)	3.1 (0.1)	3.3 (0.1)	3.5 (0.1)
Unknown 1.1 (0.0) 0.9 (0.1) 2.8 (0.1)	Unknown	1.1 (0.0)	0.9 (0.1)	2.8 (0.1)
Mean calendar year follow-up (std dev) 2001.5 (0.004) 2008.1 (0.01) 2015.5 (0.01)	Mean calendar year follow-up (std dev)	2001.5 (0.004)	2008.1 (0.01)	2015.5 (0.01)
N 212.281 159.097 193.714	N	212.281	159.097	193.714

Table 1: Characteristics of the sample, by period of interview

Notes: The sample, which includes adults ages 25–84 at follow-up, is weighted to be nationally representative using *mortwtsa* in IPUMS. Percent distributions and standard errors are in parentheses unless otherwise noted. Source: NHIS.

The population underwent significant educational upgrading between T1 and T3, with the proportion having a bachelor's degree rising from 25.8% to 34.2%. At the same time, the proportion of people who had ever smoked decreased from 47.9% to 40.9%, alongside a reduction in the proportion of smokers who smoke heavily. A growing proportion of people reported Hispanic ethnicity. The percentage reporting that they had

health insurance coverage rose by 1.4 percentage points. The largest change in alcohol consumption patterns was an increase of 3.9 percentage points in the proportion who currently drink but are not heavy drinkers. Mean units of BMI above 25 rose by 0.84 kg/m² and mental distress increased.

Table 2 shows the coefficients of the three hazard models that recognize different phases of the life course. In all models, variables are related to mortality in the expected direction. Mortality falls with rising educational attainment and rises with smoking intensity, increased psychological distress, absence of health insurance coverage (insignificantly), and higher BMI. Coefficients of alcohol consumption are more complex. Relative to light/moderate drinkers, former drinkers and current heavy drinkers had higher mortality but so did lifetime abstainers. A J-shaped relationship is commonly found, with the mortality nadir among light drinkers and higher mortality among nondrinkers and moderate to heavy drinkers (Rogers et al. 2013).

The coefficient for calendar year can be interpreted as the unaccounted-for annual trend that remains after controlling for changes in the included covariates. With all variables in the model, the coefficient of calendar year for the full period is -0.0046 - implying an annual mortality change of -0.46%. With only calendar year and age in the model (to control only the effects of changes in the age distribution), the coefficient of calendar year is -0.0112 (Table 2, notes). In other words, age-standardized mortality on average decreased by 1.12% each year of the period. When sex and race/ethnicity are added in Model 1, mortality decreased at a rate of -0.0113 per year, nearly the same pace as the age-standardized rate. Comparing the rate of decline of the age-standardized rate to the rate of decline when all variables are present in Model 3 implies that (0.0046/0.0112 = 0.41) 41% of the trend remains unaccounted for by variables included in the model.

Table 2 shows how the coefficients of variables change as different stages of the life cycle are introduced. Coefficients of age and sex change relatively little across the three models. Hispanics have lower mortality than Whites in Model 1, reflecting the well-known Hispanic paradox. Their advantage grows markedly when account is taken of their lower average educational attainment in Model 2. The decline in the Hispanic coefficient between Models 2 and 3 shows that the variables introduced in Model 3 account for a substantial amount of the Hispanic advantage. Among these, lighter cigarette smoking among Hispanics appears to play a central role (Fenelon 2013).

Relative to those who didn't graduate from high school, those who attained higher levels of education lose about half of their mortality advantage when variables reflecting later stages of adult life are introduced.

	Model 1		Model 2		Model 3	
	Coefficient	CI	Coefficient	CI	Coefficient	CI
Calendar year	-0.0113	(-0.0141, -0.0085)	-0.0042	(-0.0070, -0.0013)	-0.0046	(-0.0074, -0.0018)
Age	0.084	(0.082, 0.085)	0.079	(0.078, 0.081)	0.083	(0.082, 0.085)
Male	0.406	(0.377, 0.435)	0.439	(0.410, 0.469)	0.388	(0.355, 0.421)
Race/Ethnicity						
Non-Hispanic White	ref	ref	ref	ref	ref	ref
Non-Hispanic Black	0 383	(0 341 0 426)	0 255	(0.212, 0.298)	0.259	(0.217_0.302)
Hispanic	-0 179	(-0.239 - 0.118)	-0.410	(-0.473 - 0.347)	-0.269	(-0.333 -0.205)
Other	_0.245	(_0.349, _0.141)	_0.232	(_0.336, _0.129)	_0 181	(_0.285, _0.077)
Other	-0.243	(-0.343, -0.141)	-0.252	(-0.000, -0.120)	-0.101	(-0.203, -0.077)
Educational attainment						
< High school			ref	ref	ref	ref
High school			-0.318	(-0.357, -0.278)	-0.153	(-0.193, -0.113)
< 4 vears college			-0.465	(-0.512, -0.419)	-0.227	(-0.273, -0.181)
≥ 4 years college			-0.929	(-0.982, -0.875)	-0.492	(-0.546, -0.438)
· •						
Cigarette smoking						
Never					ref	ref
Former					0.405	(0.364, 0.446)
Current (< 10/day)					0.842	(0.777, 0.906)
Current (10–19/day)					0.843	(0.775, 0.911)
Current (20–39/day)					0.964	(0.906, 1.023)
Current (40+/day)					1.148	(1.036, 1.260)
Unknown					0.490	(0.299, 0.682)
Alcohol use						
Never					0.391	(0.347, 0.434)
Former					0.448	(0.410, 0.487)
Light/moderate					ref	ref
Heavy					0.201	(0.133, 0.269)
Unknown					0.180	(0.032, 0.329)
Mental distress						
None (K-6: 0-4)					ref	ref
Moderate (K-6: 5-12)					0.476	(0.437, 0.515)
Severe (K-6: ≥ 13)					0.771	(0.710, 0.832)
Unknown					0.501	(0.394, 0.608)
Body Mass Index > 25					0.008	(0.004, 0.013)
Llootth incurrence course						
Treaturi insurance coverag	e				rof	rof
tes					rer	
					0.010	(-0.060, 0.079)
Unknown					0.010	(-0.304, 0.324)

Table 2: Coefficients from Cox proportional hazard models predicting mortality within six years of interview

Notes: CI: 95% confidence interval. Not shown are results of a model including only calendar year (coefficient: -0.0112; CI: -0.0140--0.0084) and age (coefficient: 0.083; CI: 0.081--0.084). The sample, which includes adults ages 25–84 at follow-up, is weighted to be nationally representative using *mortwtsa* in IPUMS. *Source*: NHIS.

3.1 Accounting for mortality change

Table 3 shows changes in the annual rate of mortality change associated with each variable of interest using Model 3 in conjunction with the regression decomposition (Equation 1). Values below zero indicate that changes in the given variable pulled mortality downward, while positive values suggest that changes increased mortality. Results are presented in graphical form in Figure 1. By far the largest driver of declining mortality over this period was evolving smoking patterns. Changes in smoking were responsible for an average annual decline in mortality of 0.0047. This value can be compared to the annual rate of mortality decline of 0.0112 in the regression on year controlling only age. Changes in the distribution of smoking behaviors therefore account for 42% (0.0047/0.0112) of the age-controlled decline in mortality over this period.

Variable	Model 1	Model 2	Model 3
Calendar Year	-0.0113	-0.0042	-0.0046
Age	0.0116	0.0110	0.0116
Male	0.0001	0.0001	0.0001
Race/Ethnicity	-0.0009	-0.0017	-0.0011
Educational attainment		-0.0048	-0.0026
Cigarette smoking			-0.0047
Alcohol use			-0.0012
Mental distress			0.0013
Health insurance coverage			0.0000
Body mass index			0.0005

Table 3:Contribution of change in variable to change in the annual death
rate, 1997–2019

Source: NHIS.

The variable with the second-largest effect on the rate of mortality decline over the entire period was improvements in educational attainment, which contributed an annualized amount of 0.0026 to the decline. This represents 24% of the age-controlled decline of 0.0112 over the period.

An increase in the prevalence of light/moderate drinking, together with its low risk, accounts for 11% (0.0012/0.0111) of the mortality decline. Rapid increases in the proportion of the population classified as Hispanic or other races (Table 1), combined with large mortality advantages for these groups relative to Whites (Table 2), were responsible for 10% (0.0011/0.0111) of the mortality decline. Changes in health care coverage, as measured here, had a negligible effect.

Figure 1: The contribution of changes in sociodemographic variables to mortality change, by period



Notes: Insurance coverage is abbreviated as Ins. Results are based on Model 3.

Two factors contributed to rising mortality during the full period. Increasing levels of psychological distress raised annual mortality by 0.0013 per year, while rising BMI increased mortality by 0.0005 per year.

The variable whose results are most sensitive to the inclusion of other variables in a model is educational attainment. In Model 2, this variable accounts for an annual mortality decline of 0.0048, about double its contribution in Model 3 (0.0026). If this reduction in its contribution is thought to be an indicator of the extent to which educational attainment is "acting through" other variables whose values it helps determine rather than simply being correlated with them, then its contribution in Model 2 is the proper estimate.

3.2 Accounting for a slowdown in mortality improvements

As noted earlier, data from vital statistics showed that the rate of mortality decline slowed during the period. To investigate whether the variables employed in this paper may have contributed to the slowdown, we repeat the regression decomposition to investigate mortality change twice, once between T1 and T2 and a second time between T2 and T3.

Based on official life tables for the United States (Arias and Xu 2022: Table 19), the mean level of life expectancy at birth in T1 was 76.96 years. In T2 it was 78.23 years, and in T3 it was 78.75 years. So the slowdown is clearly captured using these periods, with mean life expectancy gaining 1.27 years between T1 and T2 and only 0.52 years between T2 and T3. Observations in T1 are separated from those in T2 by a mean of 7.5 years, identical to the separation between T2 and T3. We use the Model 3 hazard model estimated for the full period so that the only difference in results between T1/T2 and T2/T3 must arise from differences between them in rates of distributional change of variables during these periods.

Results for these subperiods are shown in Table 4 and Figure 1. Declines in smoking are the largest drivers of mortality reduction in both subperiods, while educational upgrading is the next largest. For both variables, the contribution is larger in the second subperiod than in the first, implying that these variables were exerting greater downward pressure on mortality during the second period and were therefore not responsible for the slowdown in the rate of mortality decline.

Variable	T1 vs T2	T2 vs T3
Year	-0.0046	-0.0046
Age	0.0111	0.0120
Male	0.0002	-0.0001
Race	-0.0013	-0.0009
Educational attainment	-0.0024	-0.0028
Cigarette smoking	-0.0043	-0.0050
Alcohol consumption	-0.0008	-0.0016
Body mass index	0.0007	0.0003
	0.0000	0.0000
Health Insurance coverage	0.0000	0.0000
Mental distress	0.0003	0.0022

Table 4:	Contribution of change in variable to change in the annual death rate
	for subperiods

Notes: T1 = 1997–2004; T2 = 2005–2011; T3 = 2012–2019. Results are based on Model 3. Source: NHIS.

Notably, the only variable driving mortality upward more sharply in the second period than the first is mental distress. In the first subperiod, changes in mental distress increased mortality by 0.0003, while changes in the second subperiod pulled mortality upward by 0.0022. This is the largest contribution of any variable to differences in rates of mortality change between subperiods T1/T2 and T2/T3. The result reflects the rapidly growing prevalence of moderate and severe distress during T2/T3 (Table 1) combined with the very high mortality penalty associated with these conditions (Table 2). When the

average rate of mortality decline is -0.0112, a deflection in the rate of change of 0.0019 (0.0022-0.0003) is impactful.

Summing the contributions of the variables in our model, exclusive of age, shows that they account for a decline in annual mortality of 0.0076 between T1 and T2 and a decline of 0.0079 between T2 and T3. Thus a slower improvement in the distribution of variables during the second subperiod relative to the first is not a promising explanation of any slowdown in the rate of mortality improvement during 1997–2019, although rapidly rising mental distress stands out as a partial exception.

3.3 Robustness check

We performed a sensitivity analysis using a death follow-up period of three years since survey rather than five years. Results were minimally affected by shortening the followup period. (See Figure A-1 in the appendix.)

4. Discussion

We begin by comparing the mortality trend implicit in the NHIS to that in national vital statistics. Over the full period 1997–2019, the age-controlled rate of mortality change in the NHIS was –0.0112 (Table 2). Using data from the official vital statistics published for 1997 and 2019 (Xu et al. 2021), we find that the annualized rate of change in the age-standardized death rate among people aged 25–84 between 1997 and 2019 was –1.09%.⁴ Thus there is excellent agreement in the estimated pace of adult mortality change over the period between NHIS surveys linked to mortality and official vital statistics. There is also good agreement with the Social Security Administration's annual estimates of age/sex standardized death rates. Based on data in the Trustees (2022) annual report, the average annual rate of change in mortality between 1997 and 2019 was –1.14%.

In Model 3, the variable making the largest contribution to mortality decline over the full period as well as both subperiods is cigarette smoking. Smoking is a major risk factor for death, while large declines in proportions of the population who smoke make this a very active variable on the mortality landscape. Its role should come as no surprise, as demographers and epidemiologists have described its impact on population mortality levels for many years (Preston 1976; Peto et al. 1992; National Research Council 2011).

The second most powerful variable contributing to mortality decline in Model 3 is educational attainment. And its contribution is nearly twice as great in Model 2, where variables through which it may be plausibly imagined to be working are excluded. This

⁴ This calculation uses the age distribution of the US Census in 2000 to age-standardize death rates in five-yearwide age intervals in 1997 and 2019.

result is consistent with the very strong relationship between educational attainment and mortality (Hummer and Lariscy 2011; Hummer and Hernandez 2013; Sasson and Hayward 2019; Case and Deaton 2022) and the rapid improvement of the educational distribution of Americans (Table 1). Nevertheless, the result may seem surprising because the variable, often studied as a major factor in cross-sectional mortality differentials, is seldom featured in discussions of mortality trends. An important exception is the work of Wolfgang Lutz, who has used international cross sections and time series to argue that the dominant factor in global improvement in mortality over the past century is an increase in adult educational attainment (e.g., Lutz and Kebede 2018; Lutz and Skirbekk 2014).

Luy et al. (2019) also found that rising educational attainment contributed to improvements in mortality. They decomposed changes in life expectancy at age 30 in three countries between 1990 and 2010 into effects associated with changes in the educational distribution and effects of mortality change at a given level of educational attainment. In the United States, 19% of the gain in life expectancy was associated with educational upgrading. In Italy and Denmark, respectively, 20% and 24% of improvements in life expectancy were attributed to educational upgrading.⁵

In view of the close association between educational attainment and health/mortality, a number of studies have investigated the extent to which educational attainment causally affects health. The leading alternative explanation of the association is that both education and health are heavily influenced by third variables, such as childhood family circumstances or personality features such as conscientiousness. The principal research design used to investigate causal pathways is to study the effect of compulsory schooling laws, treated as an exogenous variable, on the adult health of affected cohorts. Results in the United States and elsewhere are inconclusive, with some positive results scattered among many negative results. (For summaries, see Zajacova and Lawrence [2018] and Moorthy, Figinski, and Lloro [2022].) This research design is not ideal because compulsory schooling laws affect only a non-representative minority who would not have attended school in the absence of compulsion.

What is not in doubt is that people with more schooling are more likely to exhibit healthy behaviors with respect to such factors as smoking, obesity, vegetable consumption, and seat belt use. They also have a lower prevalence of heart disease, stroke, and cancer (Cutler and Lleras-Muney 2006). And even if educational attainment were simply a proxy for an alternative set of socioeconomic circumstances, its powerful role shows that such circumstances deserve a prominent place among the variables accounting for declining mortality.

⁵ It should be noted that we are dealing with death rates directly, whereas Luy et al. (2019) examine changes in life expectancy. With survival patterns similar to those in the United States, the effect of a proportionate change in death rates typically translates into a proportionate effect on life expectancy that is only about one-quarter as large (Keyfitz 1977). So our results are not strictly comparable to theirs.

It should be noted that random measurement error typically biases coefficients toward zero. Such errors would be expected to produce an underestimate of the contribution of variables to mortality change. Errors in measurement extend to variables like smoking and alcohol consumption, the lifetime dynamics of which cannot be fully captured by a single cross-sectional survey. Measurement error is particularly worrisome in the case of obesity. Our results regarding obesity contrast with those of Preston, Vierboom, and Stokes (2018). Using a research design similar to that of the present paper, they conclude that rising obesity reduced the annual rate of mortality decline by 23% over the period 1988–2011. The principal difference between the studies is that different variables were used to represent obesity. The present study uses NHIS data on body mass index at baseline, whereas the earlier study used a much smaller dataset on lifetime maximum BMI from the National Health and Nutrition Examination Survey. Lifetime maximum BMI is associated with much larger estimated mortality risks than is baseline BMI (Stokes and Preston 2016a, 2016b). A main reason for the difference is that people who develop a major chronic disease lose substantial weight on average (Vierboom, Preston, and Stokes 2018). The inflow of sick individuals into lower weight categories creates large reverse causation biases in the relation between BMI and mortality when baseline weight is used. As a result, we believe that the effect of obesity on mortality trends is underestimated in Tables 2 and 3.

Our measure of smoking references conventional cigarette use. Since the study period witnessed the rise of e-cigarettes, smokers who eventually switched from conventional cigarettes to e-cigarettes are coded as former smokers in our analysis. The former smoker category may therefore be associated with additional tobacco-related risk in more recent years than in the past.

Our construct representing alcohol consumption is susceptible to a similar reverse causation bias since ill individuals may forgo alcohol, artificially decreasing the mortality penalty of alcohol consumption for reasons unrelated to alcohol. In addition, alcohol use may be underreported for reasons of social desirability, another reason the analysis may underestimate the true contribution of changing drinking patterns to national mortality trends.

To our knowledge, this study is the first to use individual-level observations to analyze the impact of a changing population burden of mental distress on trends in mortality. We document that increasing mental distress contributed to the stagnation of mortality improvement during the period. Population-level research on mental distress and mortality is only emerging, and it is possible that we are not accurately estimating the impact. The K-6 scale available in the NHIS, categorized into three groups, is not uncommon in research on distress (e.g., Tomitaka et al. 2019). However, there is little research on how this construct captures the variable's association with mortality. The sensitivity and specificity of the K-6 construct in detecting an association with mortality remain uncertain.

Referring to Model 3 in Table 4, we were not able to account for 0.0046 of the original age-controlled decline of 0.0112. Our failure may be attributable to imprecise measurement or conceptual weakness of sociobehavioral variables. It may also reflect the activity of variables that are not represented in our framework or in the NHIS. Buxbaum et al. (2020) conclude that improvements in pharmaceutical practices, especially greater use of statins and blood pressure drugs, accounted for 35% of the US mortality decline over the period 1990–2015. Such a fraction would account for much of the trend left unexplained.⁶

We are unable to account for a slowdown in rates of mortality decline during the period. We show that the socio-behavioral variables were working to reduce mortality during 2009–2016 as forcibly as they were during 2000–2008. But their activity was not sufficient to avert a major slowdown in the rate of mortality improvement. Rising levels of mental distress were partially responsible for the slowdown, consistent with accounts that feature a rise in "deaths of despair" (Case and Deaton 2020, 2022). Such explanations based on a generalized deterioration in mental health have been questioned; proposed explanatory substitutes involve specific illicit drug supply and demand factors (Ruhm 2018; Masters, Tilstra, and Simon 2018). Our results, empirically linking an individual-level indicator of mental health to rising mortality, provide support for including despair or its surrogates in an explanatory framework.

For more than a century, mortality in advanced countries declined steadily, encouraging the idea that future declines could be predicted by extrapolating rates of decline observed in the past (Vaupel, Villavicencio, and Bergeron-Boucher 2021). But the declines were not on automatic pilot; they were a result of human activity in many different spheres, including advances in education and social programs to reduce smoking. Better knowledge about the sources of past mortality changes can inform this important chapter of social history. It can also improve projections of future mortality. For example, levels of educational attainment are readily predictable on a birth cohort basis since attainments change little after age 30 (Lutz and KC 2011). Likewise, the effects of smoking changes on mortality are predictable by observing cohort patterns of lung cancer mortality (Preston et al. 2014). Carefully integrating such factors into mortality projections should help improve their reliability.

⁶ In addition to a longer period, their study focused on life expectancy at birth and includes infancy and childhood, so results are not strictly comparable to ours. Some of the same variables are considered, including smoking, obesity, and health care coverage. They find that improved "public health," including reductions in smoking and improved traffic safety, accounts for 45% of US mortality decline between 1990 and 2015. They do not consider the role of increased educational attainment.

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Appendix

Figure A-1: The contribution of changes in sociodemographic variables to mortality change over full period, by length of mortality follow-up



Notes: Race/ethnicity is abbreviated as R/e; insurance coverage as Ins; and mental distress as MD. Results are based on Model 3. Source: NHIS.