Association Among Socioeconomic Status, Health Behaviors, and All-Cause Mortality in the United States

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Background: Health behaviors may contribute to socioeconomic inequalities in mortality, although the extent of such contribution remains unclear. We assessed the extent to which smoking, alcohol consumption, and physical inactivity have mediated the association between socioeconomic status (SES) and all-cause mortality in a representative sample of US adults.

Methods: Initiated in 1992, the Health and Retirement Study is a longitudinal, biennial survey of a national sample of US adults born between 1931 and 1941. Our analyses are based on a sample of 8037 participants enrolled in 1992 and followed for all-cause mortality from 1998 through 2008. We used exploratory and confirmatory factor analysis to derive a measure of adult SES based on respondents' education, occupation, labor force status, household income, and household wealth. Potential mediators (smoking, alcohol consumption, and physical inactivity) were assessed biennially. We used inverse probability–weighted mediation models to account for time-varying covariates.

Results: During the 10-year mortality follow-up, 859 (10%) participants died. After accounting for age, sex, and baseline confounders, being in the most-disadvantaged quartile of SES compared with the least disadvantaged was associated with a mortality risk ratio of 2.84 (95% confidence interval = 2.25–3.60). Together, smoking, alcohol consumption, and physical inactivity explained 68% (35–104%) of this association, leaving a risk ratio of 1.59 (1.03–2.45) for low SES.

Conclusions: The distribution of health-damaging behaviors may explain a substantial proportion of excess mortality associated with low SES in the United States, suggesting the importance of social inequalities in unhealthy behaviors.

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Differences in adult morbidity and mortality by socioeconomic status (SES), particularly across educational groups, have widened in the United States over the past three decades.[1–3] Growing socioeconomic gradients in the leading behavioral causes of death have coincided with this trend. For instance, disparities in rates of smoking by education have increased for men and women in the United States since the mid-1960s,[4] and disparities in sedentary behavior by education have increased since the mid-1970s.[2] These trends suggest that health behaviors may be among several related mechanisms—including differential exposure to stress and differential access to material resources and medical care quality—linking SES to mortality.[3]

Sparse US studies have estimated the contribution of health behaviors to socioeconomic disparities in mortality, with this work generally inferring that health behaviors—including smoking, alcohol consumption, obesity, and physical inactivity—make only modest contributions to socioeconomic gradients in health.[5–8] However, recent European evidence suggests otherwise.[9–11] For example, health behaviors (including smoking, heavy drinking, unhealthy diet, and physical inactivity) explained the majority of the association between SES and mortality among London-based civil servants.[9]

We investigated the extent to which smoking, alcohol consumption, and physical inactivity mediated the association between adult SES and all-cause mortality in a sample of 8037 US Health and Retirement Study participants. An inverse probability–weighted marginal structural model was used to estimate the "controlled direct effect" of adult SES on mortality not mediated by health behaviors. Unlike conventional methods for effect decomposition,[12] marginal structural models account for time-varying confounding of the direct effect,[13] including the potential for socially graded diseases (e.g., cardiovascular events) to predict subsequent changes in health behaviors and mortality.[14]

METHODS

Study Population

The Health and Retirement Study is a longitudinal, biennial survey of a national sample of US adults born

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between 1931 and 1941 and their spouses. The study was initiated in 1992, based on a multistage area probability sample. The initial response rate was 81%.[15] Biennial follow-up interviews (or proxy for decedents) were conducted between the initial assessments in 1992 (baseline) and 2008 (wave 9), with wave-to-wave retention rates through 2008 of >90%.[15] The University of Michigan Health Sciences Human Subjects Committee approved the study. Further details of the study design and outcomes assessment are available elsewhere.[16–18] Among 9760 age-eligible participants enrolled in 1992, we restricted our sample to 8,037 who were alive in 1998 with nonmissing information on covariates of interest. Follow-up began in 1998 to maximize the number of survey waves with comparable measures on health behaviors of interest and maintain the temporal ordering of potential confounding, mediating, and outcome variables. For consistency with previous studies, we used standard RAND Corporation coding of wealth, income, and other variables, when available.[19]

Outcome

We followed respondents from 1998 through 2008 for all-cause mortality. The study most commonly learns of the death of a respondent when an interviewer attempts to reach the respondent for a biennial follow-up interview. In these instances, the respondent's spouse or another close family member completes a final (exit) interview. The overall response rate for exit interviews conducted through 2008 was 83%.[15] The study attempts linkages using the Social Security Death Index and the National Death Index to confirm the status, timing, and cause of death. We used the reported month and year of participant visits and deaths to assign mortality status to each visit, assuming that visits and deaths occurred at the midpoint of the month.

Independent Variables

Socioeconomic Status

We estimated early-life and adult SES using exploratory and confirmatory factor analyses applied to available measures of life course SES (eTable 1, http://links.lww.com/EDE/A752). Indicators of early-life SES included father's educational attainment (0–7, 8–11, 12, >12 years), mother's educational attainment (0–7, 8–11, 12, >12 years), father's occupation (manual/unskilled service, professional/white collar), birth in the southern United States (yes/no), and rural residence during childhood (yes/no), assessed retrospectively at baseline with the exception of father's occupation, which was assessed in 1998. Early-life SES was dichotomized, with roughly the bottom 20% categorized as "low." Indicators of adult SES included respondent's educational attainment (<high school, high school/General Educational Development (GED), some college, college+), occupation of respondent's longest job (manual/unskilled service, professional/white collar), respondent's labor force status (works full-time/part-time/retired, unemployed/disabled/not in labor force), household income (split into quartiles), and household wealth (split into quartiles), all assessed at enrollment in 1992. Continuous adult SES scores were divided into quartiles for analysis. Further details concerning the measurement of SES are provided in eAppendix A (http://links.lww.com/EDE/A752).

Health Behaviors

Potentially mediating health behaviors included current smoking, alcohol consumption, and physical inactivity (eTable 2, http://links.lww.com/EDE/A752). Behavioral mediators were lagged one wave before the mortality outcome assessment and are therefore drawn from wave 4 in 1998 through wave 8 in 2006. Current smoking at each visit was measured by asking respondents whether they currently smoke cigarettes. Alcohol consumption at each visit was measured by asking respondents to estimate the number of drinks they had on days they consumed alcohol in the last 3 months. Physical inactivity was measured by asking respondents about the frequency of vigorous physical activity, including sports and physical labor on the job.

Covariates

All models were adjusted for age at enrollment and sex. Additional baseline confounders included race and early-life SES. Potential time-varying confounders included self-reported health and self-report of doctor's diagnosis of major medical conditions (i.e., self-report of doctor's diagnosis of cancer, psychiatric disorder, stroke, heart disease, diabetes) since the respondent's last interview. Assessments of these time-varying confounders were lagged one wave before health behaviors and were drawn from wave 3 in 1996 through wave 7 in 2004.

Statistical Analyses

Our analytic objective was to estimate the extent to which the association between adult SES (A_i) and all-cause mortality (Y_{it}, where i indexes the individual and t indexes the wave of mortality assessment) was mediated by observed time-varying health behaviors (i.e., smoking, alcohol consumption, and physical inactivity), represented by M_{it-1}, after accounting for (1) a vector of baseline characteristics, C_i, that included potentially confounding baseline covariates and (2) potentially confounding time-varying covariates, represented by the vector R_{it-2}.

We began by describing the distribution of potential mediators and confounders at the first wave when they were assessed, as well as the bivariate association between these characteristics and mortality between 1998 and 2008 using two-tailed chi-square tests. Second, we estimated on the risk ratio (RR) scale the age-and sex-adjusted association between adult SES and health behaviors by fitting separate log-linear models regressing smoking, alcohol consumption, and physical inactivity on adult SES, age, and sex. Third, we estimated on the RR scale the age-and sex-adjusted association between health behaviors and mortality by fitting three separate log-linear models regressing mortality ($Y_{it} = 1$ if participant i died between waves $t-1$ and t) on health behaviors.

Fourth, we estimated the total effect of adult SES on mortality on the RR scale by regressing mortality ($Y_{it} = 1$ if participant i died between waves $t-1$ and t) on quartiles of adult SES, age, sex, race, and early-life SES (model 1):

$$\log\left[P\left(Y_{it} = 1 \middle| A_i = a, C_i = c\right)\right] = \beta_0 + \beta_1' A_i + \beta_2' C_i. \quad (1)$$

The coefficient β_1 in model 1 estimates the total effect of adult SES on mortality provided that measured baseline covariates suffice to control for confounding between adult SES and mortality.

Fifth, we assessed on the RR scale the controlled direct effect of adult SES on mortality not through mediating health behaviors using a series of log-linear models. To respect the temporal ordering of potential confounders, mediators, and the outcome, health behaviors were lagged one wave before the assessment of mortality and time-varying confounders were lagged one visit before the assessment of mediators, as shown in the eFigure 1 (http://links.lww.com/EDE/A752). In addition, because the controlled direct effect of adult SES on mortality may depend on the value to which health behaviors were set,[20] we tested for interaction in all models estimating the direct effect of adult SES on mortality by including cross-product terms between adult SES and health behaviors. There was no evidence for interaction between adult SES and any of the health behaviors (eTable 3, http://links.lww.com/EDE/A752), beyond what would be expected by chance alone, and so the cross-product terms were omitted from the models we present. In models 2–5, we estimated the effect of adult SES on mortality with regression adjustment for health behaviors modeled individually (models 2–4) and concurrently (model 5):

$$\begin{aligned}\log\left[P\left(Y_{it} = 1 \middle| A_i = a, M_{it-1} = m, C_i = c\right)\right] \\ = \beta_0 + \beta_1' A_i + \beta_2' M_{it-1} + \beta_3' C_i.\end{aligned} \quad (2\text{–}5)$$

The coefficient β_1 in model 5 estimates the direct effect of SES not mediated by measured health behaviors (M_{it-1}) provided that measured baseline covariates (C_i) suffice to control for confounding between (1) adult SES and mortality and (2) health behaviors and mortality. We compared two strategies for handled potential confounding by time-varying health status—the conventional regression adjustment approach (model 6) and a stabilized inverse probability–weighted marginal structural model (model 7). In contrast to the conventional regression adjustment approach, marginal structural models handle potential confounding by time-varying covariates through weighting rather than conditioning on covariates. This allows for identification of the direct effect of adult SES on mortality even in settings in which conventional approaches are biased, including when there is a consequence of adult SES that confounds the association between health behaviors and mortality. We present the methods for models 6 and 7 in eAppendix B (http://links.lww.com/EDE/A752). Finally, we repeated analyses of the total and direct effects of adult SES on mortality (models 1 and 7) on the risk-difference scale.

We assessed the proportion of the total effect of adult SES on mortality (model 1), represented by the RR comparing the most to the least-disadvantaged quartiles of adult SES, that was explained on the excess relative risk scale[21] by smoking, alcohol consumption, and physical inactivity modeled separately (models 2–4), concurrently (model 5), and concurrently after additionally accounting for time-varying confounding using a marginal structural model (model 7). The %BOOT SAS macro (SAS, Cary, NC) was used to generate a bootstrap distribution for each percentage attenuation parameter based on 2500 resamples and to calculate an attendant 95% bootstrap confidence interval (CI). All models included respondent-level sample weights. Sample weights were incorporated into the marginal structural modeling approach using the method described by Brumback and colleagues[22]; the product of the sample and inverse probability weights were truncated at the 99th percentile to improve the precision of estimates.[23] Analyses were conducted using SAS version 9.1.3.

Sensitivity Analyses

We conducted additional analyses to assess the robustness of results. First, SES is a multidimensional construct that cannot be measured by a single indicator. We therefore used a factor-analytic procedure to measure the latent construct SES. However, to facilitate comparison with other studies, we additionally measured adult SES by respondents' educational attainment, one of the most common indicators of adult SES in US research on social determinants of health,[24] and estimated the total and direct effects of education on mortality. These inferences were qualitatively similar (eTable 4, http://links.lww.com/EDE/A752). Second, diet is hypothesized to mediate the association between SES and mortality but is unmeasured in this study. Consistent with prior work that has used body mass index (BMI) as a proxy for nutritional status,[6,7] we assessed the sensitivity of our results by including BMI, based on self-reported height and weight, as a proxy for diet and nutritional status; results were similar to those from models for the controlled direct effect without BMI (eTable 5, http://links.lww.com/EDE/A752). Third, we were concerned about potential selection bias induced by left censoring by death or dropout due to starting follow-up in 1998 and right censoring due to attrition between 1998 and 2008. In eAppendix C (http://links.lww.com/EDE/A752), we consider the direction and magnitude of the potential bias introduced by selective mortality. Fourth, there were several potential sources of measurement error. In eAppendix D (http://links.lww.com/EDE/A752), we discuss the sensitivity of results to (1) changes in the measurement of physical activity in the HRS over time; (2) nondifferential error in the measurement of health behaviors, particularly physical activity; (3) an alternative measure of alcohol consumption; and (4) measuring health behaviors at a unique time point rather than over the life course.

TABLE 1. Distribution of Independent Variables and Mortality, Weighted by Respondent-level Sample Weights, Health, and Retirement Study (n = 8,037)

	Weighted % Total	No. Deaths[a] (Weighted % Mortality)	Difference in Proportions (95% CI)
Total	100	859 (10)	
Age at enrollment in 1992 (years); mean (SE)	55.48 (0.04)		
50–52[b]	23	154 (8)	0.0
53–55	28	206 (8)	0.51 (–1.27 to 2.30)
55–58	26	255 (12)	3.92 (1.93 to 5.90)
59+	22	273 (14)	6.00 (3.82 to 8.19)
Sex			
Men[b]	47	501 (13)	0.0
Women	53	358 (8)	–5.42 (–6.88 to –3.95)
Race			
White/other[b]	91	719 (11)	0.0
Black	9	195 (15)	4.12 (1.90 to 6.34)
Early-life SES[c]			
High[b]	80	595 (10)	0.0
Low	20	264 (14)	4.17 (2.28 to 6.05)
Current smoker[d]			
No[b]	80	523 (8)	0.0
Yes	20	317 (20)	12.40 (10.08 to 14.73)
Alcohol consumption (no. drinks per drinking occasion)			
0[b]	65	619 (11)	0.0
1–2	26	121 (7)	–4.88 (–6.43 to –3.32)
3–4	6	460 (12)	0.49 (–2.80 to 3.77)
5+	2	172 (22)	10.71 (3.75 to 17.67)
Physically inactive[d,e]			
No[b]	49	277 (7)	0.0
Yes	51	563 (14)	6.53 (5.07 to 7.98)
Self-rated health[f]			
Excellent[b]	20	70 (5)	0.0
Very good	34	155 (6)	1.30 (–0.26 to 2.87)
Good	27	260 (12)	7.59 (5.65 to 9.53)
Fair	14	197 (17)	11.96 (9.34 to 14.58)
Poor	5	140 (29)	24.66 (20.02 to 29.31)
Stroke[f]			
No[b]	97	746 (10)	0.0
Yes	3	69 (26)	16.25 (10.40 to 22.11)
Heart disease[f]			
No[b]	84	577 (9)	0.0
Yes	16	234 (19)	9.98 (7.49 to 12.47)
Diabetes[f]			
No[b]	89	619 (9)	0.0
Yes	11	188 (19)	10.37 (7.52 to 13.22)
Emotional or psychiatric problems[f]			
No[b]	86	630 (10)	0.0
Yes	14	172 (15)	5.83 (3.39 to 8.27)
Cancer[f]			
No[b]	3	732 (10)	0.0
Yes	7	86 (16)	6.21 (2.74 to 9.67)

[a]Death during follow-up between 1998 and 2008.
[b]Reference category.
[c]Factor scores from confirmatory factor analysis with maximum likelihood estimation.
[d]Health behaviors measured at visit 4 in 1998.
[e]Between wave 4 in 1998 and wave 6 in 2002, respondents were asked if they engaged in vigorous physical activity three or more times per week for the past 12 months; physical inactivity was defined as engaging in vigorous physical activity less than three times per week. For wave 7 in 2004 and wave 8 in 2006 respondents were asked if they engaged in vigorous physical activity more than once per week, once per week, one to three times per month, or never. For these last two waves, physical inactivity was defined as engaging in vigorous physical activity less than once per week.
[f]Value of prior health status and conditions measured at visit 3 in 1996.
SE indicates standard error.

TABLE 2. Association Between SES and Health Behaviors, Adjusted for Age and Sex, Health, and Retirement Study (n = 8037)[a]

	Current Smoking	Alcohol Consumption (Drinks/Drinking Occasion)			Physical Inactivity
	Smoking vs. Nonsmoking RR (95% CI)	1–2 Drinks vs. 0 Drinks RR (95% CI)	3–4 Drinks vs. 0 Drinks RR (95% CI)	5+ Drinks vs. 0 Drinks RR (95% CI)	Inactive vs. Active RR (95% CI)
SES[b]					
Q1: highest[c]	1.00	1.00	1.00	1.00	1.00
Q2	1.44 (1.23–1.69)	0.66 (0.61–0.71)	0.97 (0.80–1.17)	2.17 (1.40–3.38)	1.10 (1.05–1.15)
Q3	1.88 (1.61–2.18)	0.47 (0.42–0.51)	0.72 (0.59–0.89)	2.64 (1.70–4.09)	1.21 (1.16–1.27)
Q4: lowest	2.35 (2.03–2.72)	0.24 (0.21–0.27)	0.58 (0.46–0.73)	3.00 (1.94–4.66)	1.39 (1.33–1.45)

[a]All models include respondent-level sample weights.
[b]Factor scores from confirmatory factor analysis with maximum likelihood estimation, split into quartiles (Q1–Q4) of adult SES.
[c]Reference category.

TABLE 3. Association Between Individual Health Behaviors and Mortality 1998–2008, Adjusted for Age and Sex, Health, and Retirement Study (n = 8,037)[a]

	RR (95% CI)
Current smoker	
No[b]	1.00
Yes	2.18 (1.88–2.54)
Alcohol consumption (no. drinks per drinking occasion)	
0[b]	1.00
1–2	0.47 (0.38–0.58)
3–4	0.63 (0.45–0.88)
5+	1.22 (0.81–1.84)
Physically inactive[c]	
No[b]	1.00
Yes	3.34 (2.76–4.03)

[a]All models include respondent-level sample weights.
[b]Reference category.
[c]Between waves 4 in 1998 and 6 in 2002, respondents were asked if they engaged in vigorous physical activity three or more times per week for the past 12 months; physical inactivity was defined as engaging in vigorous physical activity less than three times per week. For waves 7 in 2004 and 8 in 2006 respondents were asked if they engaged in vigorous physical activity more than once per week, once per week, one to three times per month, or never. For these last two waves, physical inactivity was defined as engaging in vigorous physical activity less than once per week.

RESULTS

There were 859 (10% of total) deaths between 1998 and 2008 (Table 1). The mean age at enrollment in 1992 was 55.5 years. At the first year of follow-up in 1998, 20% of respondents reported smoking, 52% reported physical inactivity, and 32% reported any alcohol consumption.

Adult SES and Health Behaviors

We found gradients in the prevalence of health behaviors by adult SES (eTable 6, http://links.lww.com/EDE/A752). There was a dose–response association between SES and smoking; compared with those in the least-disadvantaged quartile of SES, those in the second (RR = 1.44 [95% CI = 1.2–1.69]), third (1.88 [1.61–2.18]), and fourth (2.35 [2.03–2.72]) most-disadvantaged quartiles of SES had substantially increased risks of smoking (Table 2). Respondents in the most-disadvantaged quartile of SES were less likely to report 1–2 drinks on days when they drank alcohol versus abstaining, compared with those in the least-disadvantaged quartiles of SES (0.24 [0.21–0.27]). Conversely, those in the most-disadvantaged quartiles of SES were more likely to report five or more drinks on occasions when they drank alcohol versus abstaining, compared with respondents in the least-disadvantaged quartile of SES (3.00 [1.94–4.66]). There was a dose–response association between SES and physical inactivity; compared with those in the least-disadvantaged quartiles of SES, those in the second (1.10 [1.05–1.15]), third (1.21 [1.16–1.27]), and fourth (1.39 [1.33–1.45]) most-disadvantaged quartiles of SES had increased risks of physical inactivity.

Health Behaviors and Mortality

Current smoking was associated with a more than two-fold increased risk of mortality (2.18 [1.88–2.54]) (Table 3). There was a U-shaped relation between alcohol consumption and mortality, with lower risk of mortality among those reporting one to two drinks (0.47 [0.38–0.58]) and three to four drinks (0.63 [0.45–0.88]), relative to none. Physical inactivity was associated with a more than three-fold increased risk of mortality (3.34 [2.76–4.03]).

Adult SES and Mortality

After accounting for potential confounding by race and early-life SES, the total effects model (model 1) indicated elevated mortality among those in the second (1.23 [0.98–1.55]), third (1.75 [1.40–2.19]), and fourth (2.84 [2.25–3.60]) most-disadvantaged quartiles of adult SES, compared with the least-disadvantaged quartile (Table 4). After adjusting for all three health behaviors concurrently (model 5), the second, third, and fourth most-disadvantaged quartiles of SES were associated with 6% (1.06 [0.85–1.34]), 33% (1.33 [1.06–1.66]), and 86% (1.86 [1.46–2.36]) increased risk of mortality, respectively, relative to those in the least-disadvantaged quartile. In addition, adjusting for time-varying covariates using the conventional regression approach attenuated the estimated effects of SES on mortality to nearly null (model 6).

TABLE 4. Role of Time-varying Health Behaviors in Explaining Association Between SES and Mortality, Health, and Retirement Study (n=8037)[a]

	Model 1[b]	Model 2[c]	Model 3[d]	Model 4[e]	Model 5[f]	Model 6[g]	Model 7[h,i,j]
	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)
SES[k]							
Q1: highest[l]	1.00	1.00	1.00	1.00	1.00	1.00	1.00
Q2	1.23 (0.98–1.55)	1.19 (0.94–1.49)	1.13 (0.90–1.43)	1.18 (0.94–1.48)	1.06 (0.85–1.34)	0.93 (0.74–1.17)	1.10 (0.70–1.74)
Q3	1.75 (1.40–2.19)	1.61 (1.30–2.01)	1.53 (1.22–1.93)	1.58 (1.27–1.97)	1.33 (1.06–1.66)	1.03 (0.82–1.29)	1.24 (0.82–1.88)
Q4: lowest	2.84 (2.25–3.60)	2.48 (1.96–3.14)	2.39 (1.88–3.05)	2.37 (1.87–3.00)	1.86 (1.46–2.36)	1.20 (0.93–1.56)	1.59 (1.03–2.45)
Current smoker							
No[l]		1.00			1.00	1.00	1.00
Yes		1.94 (1.66–2.26)			1.81 (1.55–2.12)	1.72 (1.47–2.01)	1.27 (0.87–1.85)
Alcohol (no. drinks per drinking occasion)							
0[l]			1.00		1.00	1.00	1.00
1–2			0.57 (0.46–0.71)		0.60 (0.49–0.74)	0.70 (0.56–0.87)	0.70 (0.47–1.01)
3–4			0.69 (0.49–0.97)		0.63 (0.45–0.87)	0.76 (0.54–1.06)	0.37 (0.20–0.68)
5+			1.16 (0.77–1.73)		0.91 (0.62–1.34)	1.06 (0.71–1.58)	1.91 (0.75–4.88)
Physically inactive[m]							
No[l]				1.00	1.00	1.00	1.00
Yes				3.05 (2.52–3.69)	2.84 (2.35–3.45)	2.14 (1.76–2.60)	1.97 (1.37–2.83)

[a]All models include respondent-level sample weights.
[b]Model 1: SES adjusted for time-fixed covariates (age, sex, race, early-life SES).
[c]Model 2: Model 1 + smoke, lagged one visit.
[d]Model 3: Model 1 + alcohol consumption, lagged one visit.
[e]Model 4: Model 1 +physical inactivity, lagged one visit.
[f]Model 5: Model 1 + smoke + drink + physical inactivity, lagged one visit.
[g]Model 6: Model 5 + time-varying confounders (i.e., cancer, psychiatric disorder, stroke, heart disease, diabetes, self-rated health), lagged two visits.
[h]Model 7: Results from marginal structural model fit with inverse probability weights, where each participant in the model is weighted by the inverse of the probability that he had the mediating behaviors he actually had, given his SES, baseline covariates, and history of behaviors and time-varying covariates.
[i]Combined weights (the product of individual sample weights and inverse probability weights) were truncated at the 99th percentile.
[j]Health behaviors were included as independent variables in weight models and effects of health behaviors on mortality are not interpretable
[k]Factor scores from confirmatory factor analysis with maximum likelihood estimation, split into quartiles (Q1–Q4) of adult SES.
[l]Reference category.
[m]Between waves 4 in 1998 and 6 in 2002, respondents were asked if they engaged in vigorous physical activity three or more times per week for the past 12 months; physical inactivity was defined as engaging in vigorous physical activity less than three times per week. For waves 7 in 2004 and 8 in 2006 respondents were asked if they engaged in vigorous physical activity more than once per week, once per week, one to three times per month, or never. For these last two waves, physical inactivity was defined as engaging in vigorous physical activity less than once per week.

In contrast, estimates of the direct effect from the marginal structural model (model 7), which handled potential confounding by time-varying covariates using stabilized inverse probability weights, showed substantial direct effects of SES on mortality, with estimates similar to those from model 5. Results from risk-difference models for the total effect of SES on mortality and the direct effect of SES on mortality not mediated by health behaviors, as estimated by the marginal structural model (eTable 7, http://links.lww.com/EDE/A752), suggested qualitatively similar conclusions as models on the RR scale. Together, health behaviors explained 68% (35–104%) of the excess relative risk comparing the most-disadvantaged with the least-disadvantaged quartile of SES (Figure).

DISCUSSION

In a study of American adults from a nationally representative survey, there was a dose–response association between adult SES and all-cause mortality, with respondents in the most-disadvantaged quartile of SES having an almost three-fold increased risk of mortality relative to those in the least-disadvantaged quartile. Health behaviors associated with increased risk of mortality, particularly smoking and physical inactivity, were more prevalent among those with lower compared with higher SES. Together, smoking, alcohol consumption, and physical inactivity accounted for about two-thirds of the total effect of SES on mortality comparing the most- to the least-disadvantaged quartile of SES. Estimates were similar after accounting for potential confounding by time-varying health status, using a stabilized inverse probability–weighted marginal structural model.

Other studies with analogous analytic approaches for estimating the contribution of health behaviors to socioeconomic disparities in mortality have found substantively different results across various contexts. Similar to our estimates,

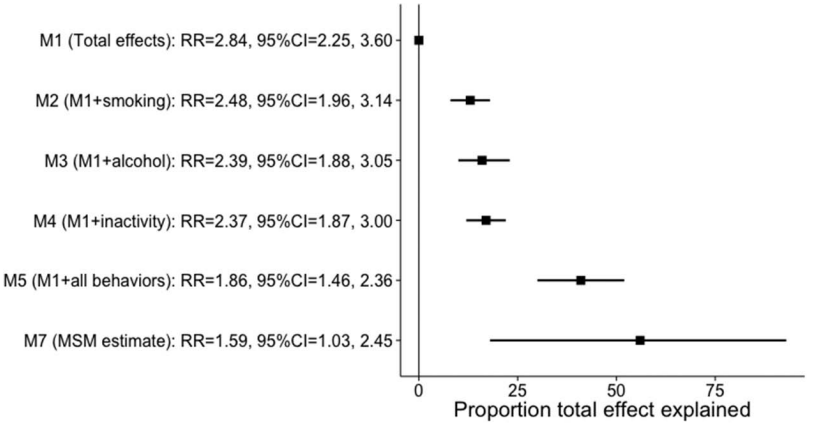

FIGURE. Proportion of the total effect of SES on mortality (model 1), represented by the RR comparing the most- to the least-disadvantaged quartiles of SES, explained on the excess relative risk scale by smoking, alcohol consumption, and physical inactivity modeled separately (models 2–4), concurrently (model 5), and concurrently after additionally accounting for time-varying confounding using a marginal structural model (model 7), health, and retirement study (n = 8,037).

Stringhini et al[11] showed that health behaviors (including smoking, heavy drinking, unhealthy diet, and physical inactivity) accounted for approximately one-half to three-quarters of the association between SES and mortality in the English Whitehall II cohort, depending on the particular indicator of SES. However, behaviors explained less than one-quarter of this association in the French GAZEL study. Although there are marked disparities in mortality by SES in our US study, as well as in the English and French studies, social disparities in health behaviors (particularly smoking) are substantially more pronounced in the United States and English studies,[11] which explains the greater contribution of health behaviors to social disparities in mortality in those contexts.

Divergent findings regarding the association among SES, health behaviors, and mortality in the broader literature may reflect differences in methodological approaches. First, the practice of simultaneously including multiple indicators of SES in models estimating the effects of SES on mortality (i.e., estimating the "independent" effect of education while holding income constant)[5–7] generates an ambiguous causal parameter.[25] For example, simultaneously adjusting for education and income likely underestimates the total effect of education on mortality, as well as the direct effect of education on mortality not through health behaviors, by controlling for variables on the pathway between education and mortality.[24] Second, with few exceptions,[7,9,11] studies have measured health behaviors as time-fixed. However, it has been recently shown that adjusting for health behaviors measured at one point in time may underestimate the contribution of these behaviors to socioeconomic disparities in mortality.[9] Third, unbiased estimation of the contribution of health behaviors by comparing the effect of SES on mortality before and after accounting for behaviors assumes no unmeasured confounding of (1) the association between SES and mortality and (2) the association between health behaviors and mortality.[26] Prior work has considered the former assumption; however, strategies for accounting for potential confounding of the association between health behaviors and mortality by prior health status have been inconsistent. In particular, European work has ignored potential confounding of the association between health behaviors and mortality,[9–11,27] whereas recent US work has adjusted for health status.[6,7] Importantly, if health status simultaneously confounds the association between health behaviors and mortality and mediates the association between SES and mortality,[28,29] then both strategies may be biased, and more advanced methods (e.g., marginal structural models)[13,30] are needed to account for time-varying confounding.

The results from our marginal structural models provided modest evidence of confounding of the relation between health behaviors and mortality by prior health status. Moreover, a comparison of the marginal structural model with the conventional regression approach of adjusting for prior health status suggests that the latter may considerably overestimate the contribution of health behaviors to socioeconomic disparities in mortality by controlling for variables on the pathway between SES and mortality, thus underestimating the direct effect of SES on mortality.

There were limitations to our study. First, although we included potential confounders of the association between SES and mortality and addressed potential confounding of the relation between health behaviors and mortality by time-varying health status, these approaches do not address unmeasured confounding. Second, we followed a sample of the 1931–1941 birth cohort, enrolled in the Health and Retirement Study in 1992, for mortality over the period from 1998 to 2008. As discussed in eAppendix C (http://links.lww.com/EDE/A752), selective survival and attrition may have biased the results. We incorporated respondent-level sample weights provided by the Health and Retirement Study to partially correct for bias introduced by differential attrition by sociodemographic characteristics between 1992 and 1998. Third, as discussed in eAppendix D (http://links.lww.com/EDE/A752), health behaviors were self-reported and may have been measured with error, particularly behaviors for which we lacked dose information (e.g., frequency and pack-years of smoking). In addition, we lacked history of smoking, alcohol consumption, and physical activity before enrollment in the study. Nondifferential errors in the measurement of health behaviors would tend

to underestimate the proportion of the total effect of SES on mortality mediated by health behaviors. Fourth, uncertainty in the estimation of the SES factor scores was not incorporated into the estimation of total and controlled direct effects. Fifth, the study did not collect information on diet; however, sensitivity analyses incorporating BMI as a proxy for nutrition did not increase the proportion of the total effect mediated by health behaviors. Sixth, we lacked the power to explicitly model three-way interactions among the health behaviors on mortality; as such, the sum of the proportions of the total effect explained by each health behavior modeled separately was greater than the proportion of the total effect explained by all behaviors modeled concurrently.

Socioeconomic disadvantage is associated with substantial mortality in the United States.[31] Our results suggest that reducing social disparities in unhealthy behaviors may mitigate socioeconomic disparities in mortality. Patient-level interventions have the potential to produce changes in health behaviors,[32] although structural barriers to sustained behavior change[33] suggest the need for complementary social approaches.[34,35]

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