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Original Contribution

Personality, Socioeconomic Status, and All-Cause Mortality in the United States

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The authors assessed the extent to which socioeconomic status (SES) and the personality factors termed the "big 5" (neuroticism, extraversion, openness to experience, agreeableness, conscientiousness) represented confounded or independent risks for all-cause mortality over a 10-year follow-up in the Midlife Development in the United States (MIDUS) cohort between 1995 and 2004. Adjusted for demographics, the 25th versus 75th percentile of SES was associated with an odds ratio of 1.43 (95% confidence interval (CI): 1.11, 1.83). Demographic-adjusted odds ratios for the 75th versus 25th percentile of neuroticism were 1.38 (95% CI: 1.10, 1.73) and 0.63 (95% CI: 0.47, 0.84) for conscientiousness, the latter evaluated at high levels of agreeableness. Modest associations were observed between SES and the big 5. Adjusting each for the other revealed that personality explained roughly 20% of the SES gradient in mortality, while SES explained 8% of personality risk. Portions of SES and personality risk were explained by health behaviors, although some residual risk remained unexplained. Personality appears to explain some between-SES strata differences in mortality risk, as well as some individual risk heterogeneity within SES strata. Findings suggest that both sociostructural inequalities and individual disposition hold public health implications. Future research and prevention aimed at ameliorating SES health disparities may benefit from considering the risk clustering of social disadvantage and dispositional factors.

cohort studies; health status disparities; mortality; personality

Abbreviations: CI, confidence interval; MCSA, Monte Carlo sensitivity analyses; OR, odds ratio; SD, standard deviation; SES, socioeconomic status.

All-cause mortality has consistently been associated with both socioeconomic status (SES) (1–4) and personality traits (5). These 2 factors are typically examined separately, as each represents a different fundamental cause of health outcomes: the societal stratification of wealth and resources on one hand (6) versus an individual's basic behavioral and psychological tendencies on the other (7).

At least 2 models may characterize the interface of SES, personality, and all-cause mortality. First, SES and personality may represent correlated or clustered risks. For instance, the notion of *indirect selection* (8–10) suggests that certain personality dispositions lead to both downward social mobility and poor health (a confounding relation). A *cultural/behavioral mechanism* model suggests that SES shapes individual personality tendencies, which in turn affect health (8) (a mediating relation). Either case indicates a clustering of social disadvantage with dispositional risk and the according possibility that personality explains differences *between* SES strata in all-cause mortality. Some findings support the notion of SES–personality correlation, suggesting that SES influences childhood personality development (11, 12), personality influences school achievement (13, 14), and educational (15) and occupational (16, 17) experiences shape personality and that adult economic attainment may be linked to personality (18, 19).

A second alternative is that SES and personality constitute independent mortality risks. Some studies report minimal correlation between SES and personality factors linked to all-cause mortality (20, 21). In this case, individual personality would explain heterogeneity in all-cause mortality risk *within*, rather than *between,* SES strata.

In a recent study of all-cause mortality in France, adjustment of SES estimates for personality attenuated SES relative risks by 24%–36% in men and 11% in women (9).

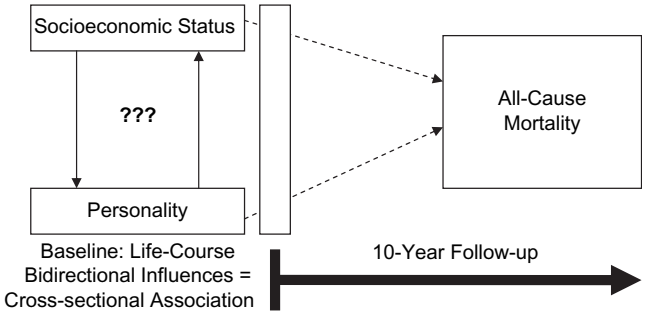

Figure 1. Conceptual model in which observed associations between personality and socioeconomic status at baseline are presumed to reflect bidirectional influences over the life course, MIDUS Study, 1995–2004. MIDUS, Midlife Development in the United States.

This has led to questions of whether similar evidence for correlated risk models exists in other populations, the public health significance of such correlated risks, and whether similar evidence can be obtained by using the comprehensive, empirically derived taxonomy of personality termed the ''big 5'' (22). The big 5 system groups specific traits along 5 superordinate dimensions (23, 24): neuroticism (composed of traits related to emotional distress), extraversion (composed of traits reflecting gregariousness, vigor, positive emotions), openness to experience (comprising traits such as intellect and novelty-seeking), conscientiousness (involving traits such as diligence, organization, reliability), and agreeableness (made up of traits reflecting compassion, cooperation, and trust).

We examined whether big 5 dimensions explained social inequalities in all-cause mortality in the United States. The causal direction of personality–SES associations—that is, indirect selection versus cultural-behavioral mechanism models (8)—cannot be distinguished when personality and SES are measured contemporaneously. However, life-course models would suggest bidirectional relations between SES and personality (and probably health) over development (10). Therefore, we consider any associations between personality and SES at baseline to reflect the product of reciprocal causal relations in operation since early development (Figure 1). In estimating all-cause mortality risk, we interpret attenuation of either personality or SES risk by the other conservatively as confounding, rather than invoking the strong temporal assumptions inherent in mediation.

MATERIALS AND METHODS

Study population

The Midlife Development in the United States national cohort study conducted baseline data collection in 1995, with 10-year all-cause mortality follow-up data released in 2007. Approved by ethical oversight boards, the study recruited noninstitutionalized, English-speaking adults aged between 25 and 74 years by using random digit dialing in 1995 (25), of whom 70% completed a phone interview. Of these, 87% returned an accompanying mail survey. Of the 4,244 individuals responding to at least the phone interview, 2,998 (71%) had data on all variables of interest, incomplete data being due primarily to the survey. Multivariate logit models indicated that the analysis sample did not differ from the larger sample in terms of age or gender but had a slightly higher average level of education (i.e., 3–4 years of college with no degree vs. 1–2 years of college) ($P < 0.001$). We assessed any bias arising from this with multiple imputation (see below).

Study measures

Mortality status. In 2004–2005 during the 10-year follow-up assessment, the names of individuals who could not be contacted for interview were submitted to the US National Death Index. The cause and date of death were not released to protect participants' confidentiality. Individuals identified as deceased by the National Death Index were coded as deaths, while those reached for follow-up or confirmed not deceased by the National Death Index were coded as alive.

Socioeconomic status. SES was assessed by a comprehensive set of indicators (26, 27). These were as follows: 1) annual household income, 2) total assets, 3) education, and 4) Duncan's socioeconomic index (28), a measure of occupational prestige. To eliminate measurement error in the observed SES indicators and overadjustment of 1 SES indicator for several others, as well as to utilize a single SES dimension with greater variability than its components, we used factor scores from a factor analysis of SES indicators (Web Table 1). (This information is described in the first of 8 supplementary tables; each is referred to as ''Web table'' in the text and is posted to the *Journal*'s website (http://aje.oxfordjournals.org/).) We note, however, that factor analysis cannot address measurement due to unobserved or omitted indicators of a latent dimension. Indicators loaded as expected on a general SES factor. We scaled factor scores by the interquartile range. In other words, they remain continuous, but the odds ratio corresponding to a 1-unit increase reflects the difference for an individual at the 25th versus 75th percentiles of SES.

Personality. The Midlife Development Inventory (29) assessed the big 5. Each dimension is tapped by 4–7 specific trait adjectives. Respondents rate how well each trait describes them on a 4-point Likert scale from ''a lot'' to ''not at all.'' The Midlife Development Inventory was developed from a large pool of big 5 trait adjectives (30) by identifying the smallest number that accounted for 90% of the variance in total scales scores, in an independent sample (29). Cronbach's alpha estimates of internal consistency for each scale were as follows: neuroticism, 0.74; extraversion, 0.78; openness, 0.77; agreeableness, 0.80; and conscientiousness, 0.58. To address measurement error in the scales due to the observed indicators, we utilized orthogonal factor scores. Items loaded as expected on the big 5 factors (Web Table 2). As with SES, factor scores were scaled by interquartile range.

Table 1. Selected Characteristics of the Sample by Survival Status, MIDUS Study, 1995–2004[a,b]

Variable	Survivor (n = 2,819)			Deceased (n = 179)			Total (N = 2,998)		
	Mean (SD) or Median (IQR)	No.	%	Mean (SD) or Median (IQR)	No.	%	Mean (SD) or Median (IQR)	No.	%
Demographic factors									
Female		1,377	49		74	41		1,451	48
Age at baseline, years	41 (13)			56 (11)			42 (13)		
Nonwhite race		317	11		20	11		337	11
SES factors									
Educational level[c,d]	6 (1, 12)			6 (5, 9)			6 (5, 9)		
Household income, $[c]	45,500 (27,000, 70,500)			38,000 (22,000, 61,500)			44,500 (27,000, 69,500)		
Assets, $[c]	32,500 (5,500, 125,000)			47,500 (3,500, 175,000)			32,500 (5,500, 125,000)		
Socioeconomic index[e]	43.4 (14.7)			37.7 (14)			43.1 (14.7)		
Health behaviors									
Current smoker		625	22		59	33		684	23
Former smoker		880	31		69	39		949	32
Heavy drinking history		506	17		30	17		467	17
Obesity		589	21		45	25		634	21
Inactivity		543	19		66	37		609	20

Abbreviations: IQR, interquartile range; MIDUS, Midlife Development in the United States; SD, standard deviation; SES, socioeconomic status.
[a] The follow-up period was approximately 9 years.
[b] Data for 25 specific personality traits are presented in Web Table 3 (http://aje.oxfordjournals.org/).
[c] Median value (25th quartile value, 75th quartile value).
[d] The education scale is composed of 12 intervals (1 = no school/some grade school; 2 = eighth grade/junior high; 3 = some high school; 4 = general equivalency diploma; 5 = high school graduate; 6 = 1–2 years of college, no degree; 7 = 3–4 years of college, no degree; 8 = 2-year college/vocational degree; 9 = 4-year college degree; 10 = some graduate school; 11 = master's degree; 12 = doctoral or other professional degree).
[e] Socioeconomic index examples: taxi cab = 22.46; railroad yard master = 36.47; air traffic controller = 50.11.

Behavioral risk factors and demographics. Behavioral risk factors were based on survey items and included current or former smoker, lifetime history of heavy drinking (a year during one's lifetime where, on average, women consumed ≥4 or men ≥5 drinks at 1 setting, ≥3 times a week), obesity (body mass index of ≥30 kg/m^2), and physical inactivity (moderate activity <1 time per month over the past year). Demographics included age, female sex, and nonwhite race.

Statistical analysis

We first estimated the association between SES and quintiles of each big 5 factor, using ordinal logit models. Next, we fit a series of logistic regressions predicting all-cause mortality. Although mortality rates were below the threshold (10%) at which odds ratios approximate relative risks, we retain the term "odds ratio" for precision. Model 1 contained only age, gender, and minority status. Model 2 included demographics plus SES. Model 3 included demographics plus the big 5. Model 4 included demographics, SES, and the big 5. On the basis of the odds ratios from models 2, 3, and 4, we computed the change in estimate of the odds ratio for SES due to personality and for personality due to SES (31) to quantify confounding. Model 5 adjusted model 4 for health behaviors, again computing change in estimates.

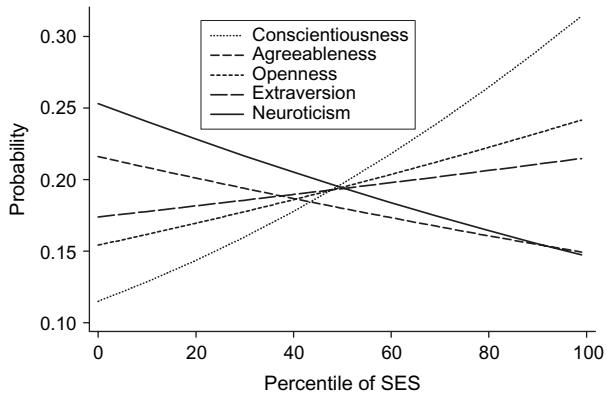

Figure 2. Marginal probabilities of membership in the top quintile of each "big 5" dimension (agreeableness, conscientiousness, extraversion, neuroticism, openness) across SES centiles, MIDUS Study, 1995–2004. Results are from demographic-adjusted ordinal logit models with covariates evaluated at the means. SES, socioeconomic status; MIDUS, Midlife Development in the United States.

Table 2. Adjusted Odds Ratios of Death Associated With Demographic, Socioeconomic, Personality, and Behavioral Characteristics, MIDUS Study, 1995–2004[a]

	Model 1		Model 2		Model 3		Model 4		Model 5	
	Odds Ratio	95% Confidence Interval	Odds Ratio	95% Confidence Interval	Odds Ratio	95% Confidence Interval	Odds Ratio	95% Confidence Interval	Odds Ratio	95% Confidence Interval
Demographic factors										
Female	0.71*	0.52, 0.99	0.65*	0.47, 0.91	0.63**	0.44, 0.89	0.60**	0.42, 0.85	0.62*	0.42, 0.91
Age (decades)	1.11***	1.10, 1.13	1.11***	1.09, 1.13	1.12***	1.10, 1.14	1.11***	1.10, 1.13	1.12***	1.10, 1.14
Nonwhite race	1.74*	1.04, 2.93	1.62	0.96, 2.74	1.84*	1.09, 3.11	1.74*	1.02, 2.95	1.70†	0.99, 2.91
Disadvantaged SES			1.43**	1.11, 1.83			1.34*	1.03, 1.74	1.14	0.87, 1.50
Personality										
Neuroticism					1.38**	1.10, 1.73	1.35***	1.07, 1.69	1.26*	1.00, 1.59
Extraversion					0.90	0.71, 1.14	0.91	0.72, 1.15	0.91	0.72, 1.15
Openness					0.90	0.69, 1.16	0.94	0.72, 1.22	0.91	0.70, 1.19
Agreeableness					1.51*	1.09, 2.09	1.46*	1.05, 2.03	1.45*	1.04, 2.02
Conscientiousness					1.03	0.78, 1.35	1.09	0.82, 1.43	1.16	0.87, 1.53
Conscientiousness × agreeableness					0.61**	0.44, 0.85	0.60**	0.43, 0.83	0.58***	0.42, 0.81
Behavior										
Current smoker									3.06***	1.96, 4.80
Former smoker									1.23	0.81, 1.85
Heavy drinking history									0.98	0.61, 1.57
Obesity									1.20	0.81, 1.77
Physical inactivity									1.68**	1.17, 2.39

Abbreviations: MIDUS, Midlife Development in the United States; SES, socioeconomic status.

* $P \leq 0.05$; ** $P \leq 0.01$; *** $P \leq 0.001$; † $P \leq 0.10$.

[a] Personality and SES are scaled by interquartile range: The odds ratio for personality traits corresponds to the 75th percentile versus the 25th percentile, while the odds ratio for SES corresponds to the 25th percentile versus the 75th percentile. Traits are centered so that values of 0 correspond to the 25th percentile, while values of 0 for SES correspond to the 75th percentile.

Using model 2–4 estimates, we also computed adjusted population attributable fractions (32, 33) to gauge the public health impact of disadvantageous SES and personality attributes. A reference level of the 25th percentile was used for factors conferring risk and the 75th for those conferring benefit. Secondary analyses examined the SES distribution and mortality risk of specific traits forming each composite big 5 dimension (34).

Sensitivity analyses examined the change in estimate in SES for all 32 combinations of adjustment for the big 5 factors. Linearity in the logit was assessed with fractional polynomials (35). Also screened were interactions between traits of the big 5 with one another and with gender, age, and SES on both multiplicative and additive scales (36). We examined missing data bias using multiple imputation (37) and whether correcting random measurement error in health behaviors increased their capacity to explain observed SES/personality risks, using simulation extrapolation (38). The simulation step of the simulation extrapolation method 1) fits the initial model and then 2) iteratively refits the model, each time multiplying the error term by an increasingly higher scaling factor representing increasing degrees of measurement error. The resulting trend in estimates under increasing measurement error is then used to extrapolate back to an estimate involving no measurement error (38). Finally, we used Monte Carlo sensitivity analyses (MCSA) (39, 40) to quantify the uncertainty in observed risk estimates due to the simultaneous operation of unmeasured confounders, selection bias differential with respect to both personality/SES and mortality, and systematic measurement error in personality and SES. In the absence of external information to validate the distributions of bias parameters, MCSA results may best be interpreted as approximations of the estimate uncertainty under different scenarios that might hypothetically characterize the data-generating process. Analyses were conducted in STATA, version 10 SE, software (StataCorp LP, College Station, Texas).

RESULTS

Descriptive data are presented in Table 1, with data for 25 specific personality traits presented in Web Table 3. Adjusted for demographics, the 25th versus 75th percentile of SES was associated with higher quintiles of neuroticism (odds ratio (OR) = 1.31, 95% confidence interval (CI): 1.19, 1.45) and agreeableness (OR = 1.24, 95% CI: 1.13, 1.36) and with lower quintiles of extraversion (OR = 0.91, 95% CI: 0.83, 1.00), openness (OR = 0.80, 95% CI: 0.72, 0.88), and conscientiousness (OR = 0.57, 95% CI: 0.51, 0.62). Figure 2 depicts demographic-adjusted marginal probabilities of membership in each big 5 quintile, across centiles of SES.

Sequential models adjusting SES for personality factors and vice versa are shown in Table 2. Unadjusted for personality, the 25th versus 75th percentile of SES was associated with a 43% increase in mortality risk. Unadjusted for SES, the 75th versus 25th percentile of neuroticism was associated with a 38% increase in mortality risk. An interaction was observed between agreeableness and conscientiousness. At high (75th percentile) agreeableness, the odds ratio for the 75th versus 25th percentile of conscientiousness was 0.63 (95% CI: 0.47, 0.84), while at low (25th percentile) agreeableness it was 1.02 (95% CI: 0.78, 1.35). The

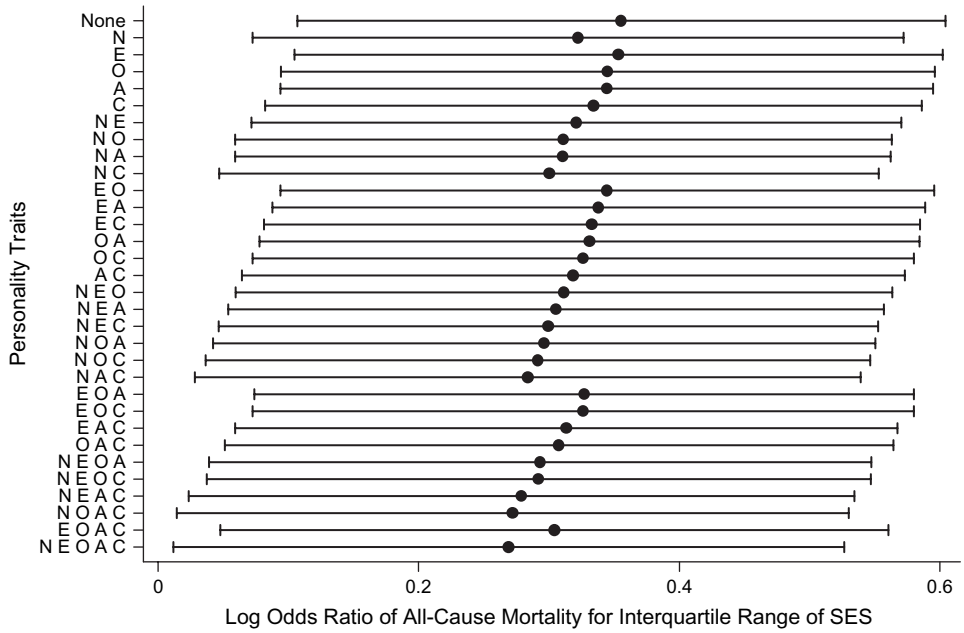

Figure 3. Impact of adjusting the SES odds ratio for 32 possible combinations of the "big 5" personality factors (agreeableness (A), conscientiousness (C), extraversion (E), neuroticism (N), openness (O)), MIDUS Study, 1995–2004. All estimates were also adjusted for demographics. MIDUS, Midlife Development in the United States; SES, socioeconomic status.

interaction also suggested that agreeableness was associated with elevated risk when conscientiousness was low (OR = 1.51, 95% CI: 1.09, 2.09) (Web Table 4).

Adjustment (model 4) revealed that personality attenuated the SES odds ratio by 20%. Figure 3 depicts the movement of the SES odds ratio observed during adjustment for all 32 possible combinations of big 5 traits. The largest attenuation appeared to be due to neuroticism and conscientiousness. By contrast, SES explained about 8% of the risk associated with neuroticism and conscientiousness (at high agreeableness) and 10% of the risk associated with agreeableness (at low conscientiousness). Absolute risks from model 4 for different configurations of SES and big 5 factors are shown in Figure 4. Persons of comparable SES but different personality showed nontrivial differences in absolute risk.

Health behaviors (model 5) explained roughly 59% of the risk associated with SES, 26% of that for neuroticism, 9% of that for conscientiousness (at high agreeableness), and 2% of that for agreeableness (at low conscientiousness). When random measurement error in health behaviors was adjusted, the percentage of risk explained became 92%, 43%, 21%, and 6%, respectively.

Demographic-adjusted, population-attributable fractions suggested that 15.9% of the population mortality was attributable to low SES, 11% to high neuroticism, and 8.6% to the combination of low agreeableness and conscientiousness. Mutually adjusting SES and personality for one another revealed population-attributable fractions of 13.3% for low SES, 9.1% for high neuroticism, and 8.6% for low agreeableness and conscientiousness. By comparison, population-attributable fractions for health behaviors mutually adjusted for one another and for demographics were 13.3% for physical inactivity, 4.7% for obesity, and 20.5% for current smoking.

Sensitivity analyses revealed regressor linearity in the logit, no other reliable additive or multiplicative interactions, and a very similar pattern of findings using multiple imputation. One exception was that the SES effect was about 10%–14% larger but, as before, personality explained 20% of this risk (Web Table 5). Stratification on gender revealed that personality explained 21% of the SES risk in men and 22% in women.

MCSA indicated that, across all possible combinations of unmeasured confounding, selection bias, and systematic measurement error, with bias parameter drawn from distributions approximating other associations within the data (Web Tables 6 and 7), log(relative risk) estimates differed from those observed by a mean of 0.59 (standard deviation (SD), 0.32) for neuroticism, 0.62 (SD, 0.37) for conscientiousness, and 0.59 (SD, 0.32) for SES. On average, confidence limit ratios around point estimates were roughly 4.5 times wider under the uncertainty entailed by these supposed combinations of biases. Full MCSA findings are presented in Web Table 8.

DISCUSSION

Our findings suggest that the interface of personality, SES, and all-cause mortality in the United States is best characterized by some degree of correlated risk. Three

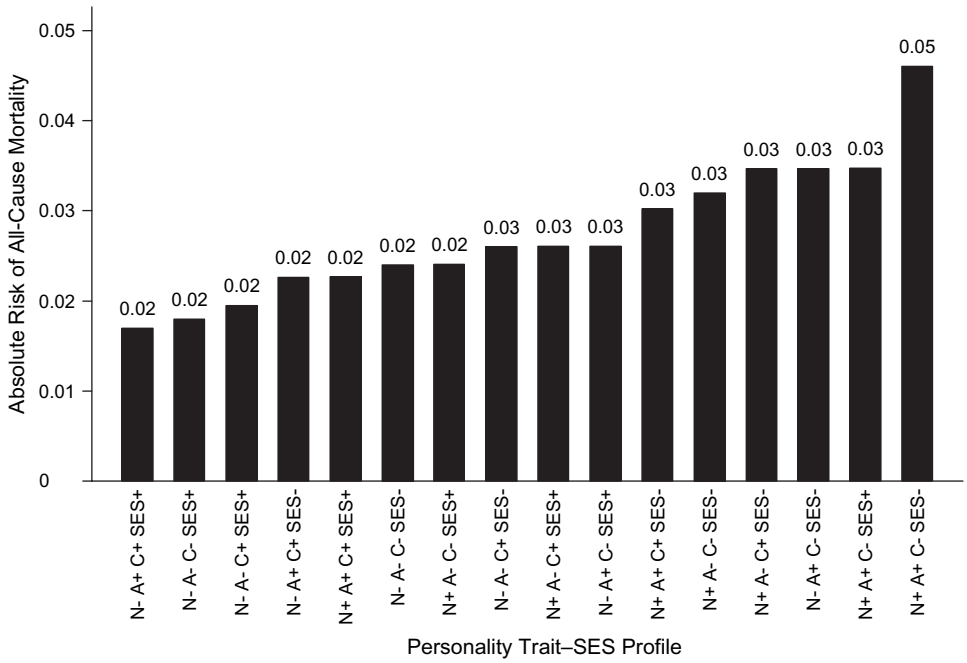

Figure 4. Absolute risk of all-cause mortality over a 10-year follow-up period from model 4 for different personality and SES risk-factor profiles, MIDUS Study, 1995–2004. +, the factor is at the 75th population percentile; −, the factor is at the 25th percentile. All other personality traits and demographic factors were held at sample means. A, agreeableness; C, conscientiousness; N, neuroticism; SES, socioeconomic status; MIDUS, Midlife Development in the United States.

aspects of the current findings are worthy of note. First, a recent study in France (9) reported that personality explained 24%–36% of all-cause mortality risk for men and 11% for women. That study used specific personality factors from classic psychosomatic medicine (41, 42). In the United States, using the more general big 5 personality framework, we found that personality accounted for about 20% of the risk associated with lower SES, a number comparable across men and women. Thus, despite large differences in populations and personality measures, our results suggest some generalizability of the correlated risk model.

Results from other studies on whether personality accounts for some amount of the social gradients in health outcomes vary. Personality constructs explain portions of social gradients in smoking and activity levels (43, 44). For obesity, some evidence suggests less confounding of risk (45, 46). Still other evidence suggests risk clustering for dispositional hostility and low SES with respect to allostatic load (47) (i.e., systematic wear on cardiovascular, neuroendocrine, and immune systems (48)). Our findings are also consistent with previous evidence of associations between personality and SES (11, 13, 19, 49, 50). We interpret the associations observed between SES and personality as the product of bidirectional influences over time, consistent with life-course epidemiologic models of risk clustering (51) and nature–nurture interplay (52).

The need to examine the public health impact of correlated personality and SES risks has been raised (22). Of the 15.9% population mortality attributable to low SES, roughly 16% could be accounted for by the big 5. Of the 11% attributable to high neuroticism, roughly 17% was attributable to SES. With adjustment for this confounding, nontrivial proportions of mortality were still associated with both low SES (13%) and personality (9.1% for high neuroticism, 8.9% for low agreeableness and conscientiousness). Thus, social inequalities and dispositional risk would both appear to be important distal etiologic factors in population survival.

Additionally, the risk clustering that we observed was modest, rather than total. Even after elimination of the confounding through adjustment, both personality and SES conferred residual, independently additive risk. This suggests that, despite the tendency for personality to explain some portion of *between*-strata SES variability in mortality risk, it also explains some portions of *within*-strata risk. In other words, some persons are able to offset the risk of social disadvantage through adaptive personality tendencies, some persons negate the advantage of high SES through risky dispositional tendencies, and others are duly advantaged or disadvantaged with respect to SES and personality.

The second central finding concerns the role of specific big 5 factors. The elevated mortality risk for persons higher in neuroticism that we observed has been previously noted (53–58). In addition, our findings indicated that conscientiousness was protective at high levels of agreeableness. This suggests that the health benefits of high conscientiousness may be most pronounced when people are also trusting and invested in creating interpersonal harmony, rather than cynical and prone to hostility. The self-disciplined, healthy behavior characterizing high conscientiousness may be socially reinforced or facilitated by the support of others that arises from an amicable, rather than antagonistic, approach to life. Such a personality style has been deemed ''effective altruists'' (59) for its balance between personal accomplishment and beneficence toward others. High agreeableness conferred mortality risk at low conscientiousness. This big 5 combination is denoted as the ''well-intentioned'' style and is characterized by the pursuit of interpersonal harmony at the expense of one's own diligence with respect to daily obligations and life goals (59). The quest for interpersonal harmony in the absence of self-discipline may signal a yielding to social pressures deleterious to health. Negative health consequences arising from a care-giving burden (60) or conflict-induced stress (61) may be particularly salient for such persons. Elevated mortality risk for higher agreeableness (independent of conscientiousness) has been observed in prior work on childhood personality (62–64).

Third, health behaviors explained substantial portions of the SES and neuroticism effects, consistent with prior reports (2). However, even after correction for measurement error, residual mortality risk was observed, particularly for configurations of conscientiousness and agreeableness. Cross-sectional, self-reported health behaviors may fail to fully capture health behaviors or changes in them over time. Remaining risk may also be a function of health behaviors not examined here, such as substance abuse, behavior inviting injuries, and health-care utilization. Residual risk may also signal the operation of biologic processes (65–67) such as allostatic load. Gene–environment interaction models (68) imply that personality phenotype may mark genetic vulnerability (69, 70) to such processes.

Our findings have several implications. Methodologically, risk estimates for low SES are likely to be overestimated without adjustment for personality tendencies. At the same time, absolute mortality risk for persons of lower SES may be underestimated, because other dispositional risks may be present and unaccounted for. At the level of prevention, our findings suggest that public health messages aimed at persons of lower SES may be additionally tailored and targeted according to dispositional factors (71–73). The efficacy and cost-effectiveness of clinical interventions with behavioral components may be similarly improved by such tailoring (74, 75). Personality research might inform initiatives to mitigate underuse or overuse of health services among socially disadvantaged individuals (76–78). Additionally, the clustering of both social disadvantage and personality risk for mortality suggests that evidence-based health policy stress both social (79) and personal (80) responsibility for health.

At a broader level, our results highlight the need to better understand mechanisms of personality–SES correlation. Such understanding can illuminate how and when to intervene in mutually reinforcing risk chains between social disadvantage and health-damaging personality tendencies. Population prevention involves not only reducing this risk clustering but also directly mitigating social disadvantage and chronic emotional distress (neuroticism), while also promoting prosocial self-discipline (conscientiousness and agreeableness). Can population shifts in socioeconomic structure and personality disposition occur? We believe that

such shifts are possible, but only over the long term. However, traditional public health targets of virtually eliminating smoking and obesity in the population are similar long-term goals.

Neuroticism declines, and agreeableness and conscientiousness increase naturally over the life course (81), meaning that, although personality is somewhat stable, most people are not impervious to adaptive changes in basic disposition. Birth cohort (82) and cross-national differences (83), as well as dispositional change during social upheaval (84–87), also suggest that sociocultural factors influence personality. Numerous social programs already endeavor to shape sociocultural factors. These include initiatives aimed at SES directly: programs for job training, early and life-long education, tax mechanisms for reducing the burden on the poor, and initiatives seeking to employ or educate persons from underprivileged backgrounds. Our findings raise the possibility that such social programs may directly and/or indirectly shape population disposition as well over the long term. Yet, it is important to note that personality variation will likely persist for evolutionary reasons (88), even if central tendencies of trait distributions shift. Efforts to better understand these complex issues appear warranted.

On the basis of a balanced assessment of study strengths and limitations, we encourage interpretation of these findings. We examined only all-cause mortality at 10 years and refrain from speculation about the interface of SES and personality risks for cause-specific mortality. Our findings are based on a US sample and the big 5 personality framework, representing both significant extensions of prior work and the limiting frame for generalization. We eagerly await further reports from other populations. The mortality rate in the Midlife Development in the United States (MIDUS) Study, while consistent with lower mortality rates often observed in mixed-age samples, also speaks to the need for future analyses when deaths have accumulated in this cohort. Date-of-death data would also permit time-to-event modeling. Finally, our MCSA indicate that risk estimates are precisely that—estimates based on available information, which might vary under unknown combinations of unobserved biases. We have no evidence supporting or dismissing the magnitude, direction, or even the operation of such factors, but we believe that the MCSA provide a useful reminder of the uncertainty involved in statistical estimation.

Study strengths involved the first examination of which we are aware of the contribution of personality to SES gradients in US all-cause mortality, careful treatment of random and systematic measurement error, analysis of missing data patterns and bias, and quantification of a range of other unobserved biases. The recency of this line of investigation highlights the need for more study. Personality, SES, and their interrelations have public health, clinical, and social policy implications.

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