REGULAR ARTICLE

Can I Buy My Health? A Genetically Informed Study of Socioeconomic Status and Health

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Abstract

Background A large literature demonstrates associations between socioeconomic status (SES) and health, including physiological health and well-being. Moreover, gender differences are often observed among measures of both SES and health. However, relationships between SES and health are sometimes questioned given the lack of true experiments, and the potential biological and SES mechanisms explaining gender differences in health are rarely examined simultaneously.

Purpose To use a national sample of twins to investigate lifetime socioeconomic adversity and a measure of physiological dysregulation separately by sex.

Methods Using the twin sample in the second wave of the Midlife in the United States survey (MIDUS II), biometric regression analysis was conducted to determine whether the established SES-physiological health association is observed among twins both before and after adjusting for potential familial-level confounds (additive genetic and shared environmental influences that may underly the SES-health link), and whether this association differs among men and women.

Results Although individuals with less socioeconomic adversity over the lifespan exhibited less physiological dysregulation among this sample of twins, this association only persisted among male twins after adjusting for familial influences.

Conclusions Findings from the present study suggest that, particularly for men, links between socioeconomic adversity and health are not spurious or better explained by additive genetic or early shared environmental influences. Furthermore, gender-specific role demands may create differential associations between SES and health.

Keywords Physiological risk · Socioeconomic adversity · Twins · Familial confounds

Introduction

Whether it be money, education, or occupational prestige, more is better for health [1]. This adage has received empirical support for decades and from studies conducted across the globe [2]. Along with this message, however, has been a concern that findings are only correlational and that "third variables" may explain this link [3]. Researchers have addressed this concern by adjusting for a wide array of factors that may account for these observations [4]. Relations between socioeconomic status (SES) and health often persist even after taking these factors into account, strengthening the conviction that higher SES results in better health. Nevertheless, tests of causality are hindered when random assignment is impossible or unethical. Family data is a means for conducting quasi-experiments, allowing researchers the opportunity to control for many (measured and unmeasured) confounds; one twin from each family serves as an age-matched control for his or her co-twin [5, 6]. Critically, unmeasured genetic and environmental factors can be controlled for in family research designs, as twins share part of their genotype (or all in the case of monozygotic twins) and were likely exposed to the same rearing household environments. Recent twin research has indicated that the observed relations between educational attainment and occupational position with health are not causal, but rather explained by familial processes [7, 8], for example, and additional research is needed to determine the degree to which other (i.e., economic,

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subjective financial) indicators of SES predict health after controlling for familial-level confounds.

There are also several observed gender differences in both health and SES that may further explain the observed differential effects of SES on health between men and women [9, 10]. From an epidemiological perspective, gender-specific role-related demands (i.e., primary breadwinner status) may render SES a stronger predictor of health for men than women. From a behavioral genetic perspective, there also remains the possibility that genetic or early environmental factors differentially confound SES-health relationships between the sexes. The purpose of the present study was to conduct a quasi-causal study of a previously established relationship between a composite socioeconomic adversity measure comprised of both education and financial indicators, and a measure of multisystem physiological risk containing a simultaneous constellation of regulatory indicators [4] that adjusts for unmeasured genetic and environmental confounds shared by twins. This previous report addressed twin dependency via a generalized equation model that clusters twins within families. The current report builds on those findings via a biometric regression that simultaneously models the between-family fixed effects (as was done in previous reports) and within-family random effects (a novel contribution of the current report). Random effects allow for the investigation of twin differences in SES that may predict twin differences in health holding constant all other measured and unmeasured confounds shared in twin pairs [6]. Using the subsample of twins from the second wave of the Midlife in the United States Study (MIDUS II) [11], we tested the hypothesis that the twin with higher socioeconomic adversity in adulthood would also exhibit greater multisystem physiological risk, an early sign of impending health problems [12]. We also investigated whether the relationship between adulthood socioeconomic adversity and physiological risk varied between men and women, adjusting for effects of age and childhood socioeconomic position that may similarly confer bias.

SES and Health

SES disparities in health are not only observed when comparing individuals at the lowest and highest extremes of the socioeconomic spectrum but also incrementally [2, 13]. This pattern emerges across various dimensions of SES including income, education, and occupational prestige [1]. Despite the vast evidence supporting links between SES and health, the lack of true experiments challenges causal inferences. Both SES indicators [14, 15] and aspects of physiological health [16–18] are partially heritable, and growing evidence suggests the same

family-level factors may partially explain the established links between SES and health.

Researchers have addressed this concern in various ways, including statistical adjustment for potential confounds [4]. Examples of studies that adjust for potential confounds abound. Rather than providing an exhaustive review of these (reviews elsewhere) [1, 13], we will describe a report that provides the basis for the present study. Using a national sample of U.S. adults, researchers identified an inverse relationship between a composite SES index consisting of education and financial indicators and allostatic load [4]. Allostatic load is a summary measure representing dysregulation across multiple physiological regulatory systems, with higher scores indicating poorer physiological well-being. In a generalized estimating equation (GEE) that clustered siblings within families, thus adjusting for potential family-level confounds, a significant relationship between lower SES and greater allostatic load was observed.

Twin and Family Studies

The ability to include as covariates an *exhaustive* collection of potential confounds is implausible, making inference of the causal effect of SES on health difficult to disentangle from social selection (e.g., random assignment to SES is unethical). Family designs, like twin and sibling studies, thus offer an alternative way to examine health in the context of people's SES while controlling for unobserved genetic and environmental selection factors [5, 6]. Whereas fixed-effect approaches investigate predictor-outcome relationships while adjusting for family-level averages of the predictor, conventional twin models (i.e., ACE models) also allow for between- and within-family variance to be decomposed into genetic and environmental sources of variance [6]. The power of twin and sibling designs is that pair differences in SES on health cannot be attributed to genetic and environmental factors shared by twins, an effect often considered "quasi-causal" [6]. As an example, if an identical twin with higher SES than their co-twin also has better health, on average, the effect cannot be attributed to genotype (identical twins share all of their genes) or their shared environments (i.e., familial environments that influence them similarly). In this way, twin designs offer the most rigorous test of hypothesized causal pathways in studies where random assignment is not possible. We note, however, that because of the correlational nature of twin studies, the third variable confound concerns linger.

Using Swedish twin pairs, researchers observed significant relations between education and income and self-rated health in a classic OLS regression, but the strength of these associations was weaker in a fixed-effects model

that clustered twins within families [19]. Others observed that sick leave granted to Norwegian adults with mental or physical health problems was utilized more among those with lower levels of education and income [20]. This relation was attenuated, however, among dizygotic twins who share approximately half of their genetic background and was diminished to non-significance among monozygotic twins who share 100% of their genes. Reports on Danish and Australian twins have demonstrated that more education was associated with greater longevity and lower risk of overweight status, respectively, but only among men [21, 22].

Several studies in the United States have utilized family data to adjust for genetic and shared environmental confounds in SES-health research. A study of 308 female twins living in California demonstrated that the twin with a working-class occupation had higher systolic and diastolic blood pressure and low-density lipoprotein cholesterol than her professional co-twin [23]. Similar to others [7], however, twin differences in physiological health were not related to twin differences in educational attainment. In a national sample of twins from the MIDUS II Study, researchers reported a significant inverse association between years of education and allostatic load [7]. In a follow-up analysis that separately modeled fixed and random effects, however, only the fixed effect was significant. The random effect was essentially zero, suggesting that any relation between education and health was explained entirely by familial processes. Another report using MIDUS Study twins indicated that twin differences in education were related to twin differences in perceived global health, but not perceived physical or mental health, body mass index, hip-to-waist ratio, or depressive symptoms [24]. Finally, MIDUS twins have been used to examine allostatic load in the context of adulthood socioeconomic disadvantage, a construct that, in addition to education, considers multiple indices of financial well-being [4]. Greater socioeconomic adversity in adulthood was related to greater physiological dysregulation across a constellation of biomarkers representing seven regulatory systems simultaneously, and this finding was observed in a GEE model that clusters siblings within families. These studies demonstrated that the family data can be used to support a causal connection between dimensions of SES and aspects of peoples' health and that the importance of SES for health may vary by dimension (i.e., income vs. education).

The Present Study

We used a sample of twins from the MIDUS II to test the hypothesis that the twin with greater socioeconomic adversity in adulthood would have higher physiological risk. We included childhood SES and age as covariates in our models to adjust for their potential between-family influence. This analysis builds on previous work in the following ways:

1. The current measure of adulthood socioeconomic adversity includes indices of education and financial well-being;
2. The current measure of physiological dysregulation includes objectively assessed biomarkers representing multiple regulatory systems;
3. The current twin approach, namely a biometric regression, decomposes both socioeconomic adversity and allostatic load into additive genetic, shared environmental, and unique environmental factors; and
4. Given historical gender differences in income despite similar education [25], coupled with a paucity of research on potential sex moderation of the genetic or shared environmental processes underlying SES-health relationships, we investigated whether there would be gender differences in genetic or environmental influences on socioeconomic adversity or physiological risk, or the relationship between these factors.

Method

Sample and Procedures

MIDUS II is the second wave in a longitudinal study of health at midlife, started in 1994. A large sample of U.S. adults identified via random digit dialing procedures represent the majority, and siblings or twins of these participants represent another large portion of the sample. The core MIDUS survey evaluates the psychological, behavioral, and social correlates of mental and physical health in mid-life and older age. A subset of the original MIDUS participants ($N = 1,043$) completed the Biomarker Project which adds biological data to participant records. The Biomarker Project consisted of an overnight stay in one of three General Clinical Research Centers (GCRC; at University of California, Los Angeles; University of Wisconsin; and Georgetown University) [26]. Eligibility for the Biomarker Project was determined by willingness and ability to travel to one of the GCRCs. Information from physical exams and assayed blood and urine samples allowed for the assessment of an array of indicators of the cardiovascular, sympathetic and parasympathetic nervous, hypothalamic pituitary adrenal axis, inflammatory, and lipid and glucose metabolism systems. A final allostatic load variable was calculated for those with data available for at least half of the biomarkers in each physiological system, resulting in an analytic sample of 1,039.

Furthermore, given the aims of the current study were to investigate whether an observed SES-physiological health association persists after adjusting for familial-level confounds, the analytic sample included 140 complete and 17 incomplete monozygotic female (MZF) twin pairs, 128 complete and 17 incomplete monozygotic male (MZM) twin pairs, 152 complete and 24 incomplete dizygotic female (DZF) twin pairs, 89 complete and 16 incomplete dizygotic male (DZM) twin pairs, and 183 complete and 37 incomplete dizygotic opposite sex (DZOS) twin pairs. Complete pairs included twin pairs in which both twins supplied some data whereas incomplete pairs included twin pairs in which only one twin supplied data (the co-twin supplied no data). The study was completed using ethical guidelines with the approval of each of the review boards of the institutions involved.

Measures

Multisystem physiological risk

The composite physiological risk variable in the present study was represented by seven regulatory systems, each of which comprised various numbers of physiological biomarkers, 24 biomarkers in total [4]. An ordinal variable was created for each of the 24 biomarkers, based on the distribution of values of the biomarker, indicating whether biomarker values fell into the quartile of "risk" or not. The highest quartile was considered the quartile "at risk" (higher values represent more physiological wear-and-tear) for most indices. For two exceptions, dehydroepiandrosterone sulfate and high-density lipoprotein cholesterol, lower scores are more health-compromising, and values in the lowest quartiles were considered "at risk."

The 24 biomarker indicators were then used to construct seven separate system-specific composite variables [4, 27]. Because each system was indicated with differing numbers of biomarkers, each system variable was constructed as the proportion of within-system indicators for which participant values fell into the "risk" categories. The resulting system variables thus ranged from 0 (none of the biomarkers within the system had values in the quartile of risk) to 1 (all of the biomarkers within the system had values in the quartile of risk). The composite physiological risk variable was constructed by summing across the seven 0–1 system-specific variables. This measure of overall "risk" ranged from 0 (none of the physiological systems have any at-risk indices) to 7 (all indices within all seven systems are at risk).

Socioeconomic adversity

Adult SES disadvantage was calculated using similar methods reported elsewhere [4]. Disadvantage scores were calculated with summed values on five indicators: education level (0 = bachelor's degree or higher, 1 = some college/associate arts degree, 2 = high school/GED or less,); family-size adjusted income-to-poverty ratio (0 = 600% or more, 1 = 300%–599%, 2 = less than 300%); current financial situation (0 = best possible to 2 = worst possible); availability of money to meet basic needs (0 = more than enough to 2 = not enough); and difficulty level of paying bills (0 = not at all difficult to 2 = very or somewhat difficult). Summing across these five 0–2 variables created a socioeconomic adversity variable ranging from 0 (no disadvantage suggested from any of the five SES variables) to 10 (greatest disadvantage reported across all five measures of SES disadvantage).

Covariates

Age was coded in years. Retrospective reports of childhood socioeconomic adversity were given during MIDUS II. Childhood socioeconomic adversity was calculated with three indicators: financial level growing up (0 = better off than others to 2 = worse off than others), the highest level of parental education (0 = some college or higher, 1 = high school/GED, 2 = less than high school), and childhood welfare status (0/1 = never/ever on welfare). The final childhood socioeconomic adversity variable thus ranged from 0 to 6, with higher values representing greater childhood socioeconomic adversity [4].

Statistical Analyses

We present descriptive statistics on analytic variables in Table 1 by gender for the subset of MIDUS II twins in the Biomarker Project. The MIDUS II full sample was used in the present analyses to test the hypothesis that greater socioeconomic adversity would be associated with more physiological risk. The MIDUS II twin sample was then used to assess whether the socioeconomic adversity-physiological risk association would persist after adjustment of potential family-level confounds. We used linear mixed-effects regression with SAS proc mixed to make gender comparisons on all key variables in the twin sample, given that twin-related dependency in the data violates the independence of observations assumption.

To examine our first question, whether adulthood socioeconomic adversity would be related to physiological risk, we included Biomarker Project participants with complete data on our analytic variables ($n = 1,039$). Given that the sample contains twins, we conducted GEEs to examine the relationship between socioeconomic adversity and physiological risk, adjusting for potential family-related dependency in the data. We first tested for a socioeconomic adversity main effect, and then a potential interaction with gender in relation to physiological risk, adjusting for childhood socioeconomic adversity

Table 1 Description of analytic twin sample by gender

| | Twin sample | | | | |
| | Men (n = 227) | | Women (n = 308) | | |
	M (SE)	Range	M (SE)	Range	Est (SE)
Allostatic load	1.65 (0.07)	0–4.50	1.73 (0.06)	0–5.03	−0.08 (0.09)
Adulthood socioeconomic adversity	3.59 (0.17)	0–9	4.41 (0.15)	0–10	−0.82*** (0.23)
Childhood socioeconomic adversity	1.67 (0.09)	0–6	1.81 (0.08)	0–6	−0.14 (0.11)
Age	54.83 (0.59)	34–83	55.14 (0.59)	34–81	−0.31** (0.12)

*$p < .05$; **$p < .01$; ***$p < .001$.

and age. Models were estimated using the *gee* package (version 4.13-20) in R 4.2 [28].

To examine our second question, whether the hypothesized link between socioeconomic adversity and physiological risk would persist after taking into account family-level confounds, we used a sample of twins who completed the Biomarker project (n = 692 twin pairs; 140 MZF, 128 MZM, 152 DZF, 89 DZM, and 183 DZOS). We first present twin correlations to illustrate genetic and environmental influences on our analytic variables. We additionally present cross-twin, cross-trait correlations to describe the genetic and environmental influences underlying the hypothesized association between SES and health. We interpret MZ twin correlations that are greater than DZ twin correlations as evidence for additive genetic influences, with the reverse providing evidence for a shared environmental influence. MZ correlations that are not at least twice as great as DZ correlations indicate evidence of both additive genetic and shared environmental influences.

Biometric regressions were then conducted with a five-group modeling approach which examines differences by zygosity and gender in relationships between adulthood socioeconomic adversity and physiological risk (MZM, MZF, DZM, DZF, and DZOS) [29]. We used a biometric regression model implementing full-information maximum likelihood estimation with robust standard errors in the M*plus* 7.11 program. These models allow for a test of unique associations between SES and health while controlling for familial factors that may relate to both SES and health. Variance in both physiological risk and socioeconomic adversity was decomposed to additive genetic (A), shared environment (C), and unique environment (E) components for each twin [5] to determine the proportion of variance in these outcomes that is explained by these (A, C, and E) factors. Additive genetic (A; correlated 1.0 for monozygotic [MZ] and 0.5 for dizygotic [DZ] twins) effects which account for the assumption that these twin pairs share 100% and 50% of their genes, respectively, and shared environmental (C; correlated 1.0 for both MZ and DZ

twins) effects contribute to the similarity between twins. Any unique environmental effects, which are represented by E, contribute to differences between twins and include measurement error (uncorrelated between twins). Model assumptions include that the A, C, and E latent variables neither correlate nor interact with one another, and that parental genetic backgrounds are uncorrelated [29]. The physiological risk was regressed on the A, C, and E components of socioeconomic adversity (see Fig. 1) to test the hypothesis that higher socioeconomic adversity would associate with higher physiological risk, controlling for A and C, as well as age and childhood socioeconomic adversity. Growing research suggests that factors related to individual SES, including both income [15] and education [14], have genetic bases and are transmitted intergenerationally within families [30]. By controlling for additive genetic and shared environmental influences that inform the development of individual SES—which are shared by twins—we can determine the degree to which links between socioeconomic adversity and health are confounded by these familial factors.

The initial multiple regression model examined whether the hypothesized socioeconomic adversity-health association would be detected within the twin sample before adjusting for A and C latent variables. Coefficients from this initial model (Model 1) served as baseline (total) effect from which we could compare hypothesized effects adjusted for effects of A and C. Biometric models were used to indicate whether the twin with greater socioeconomic adversity would also have higher physiological risk, controlling for A and C. We next constrained the A and C paths to be the same to examine whether genetic and environmental confounds could be distinguished in male and female groups separately (Model 2). We constructed a final model in which regression effects between men and women were equated (Model 3). This final model allowed for an investigation of whether socioeconomic adversity influenced physiological risk equally between men and women.

To assess model fit, we used chi-square difference tests of nested models, root mean square error of
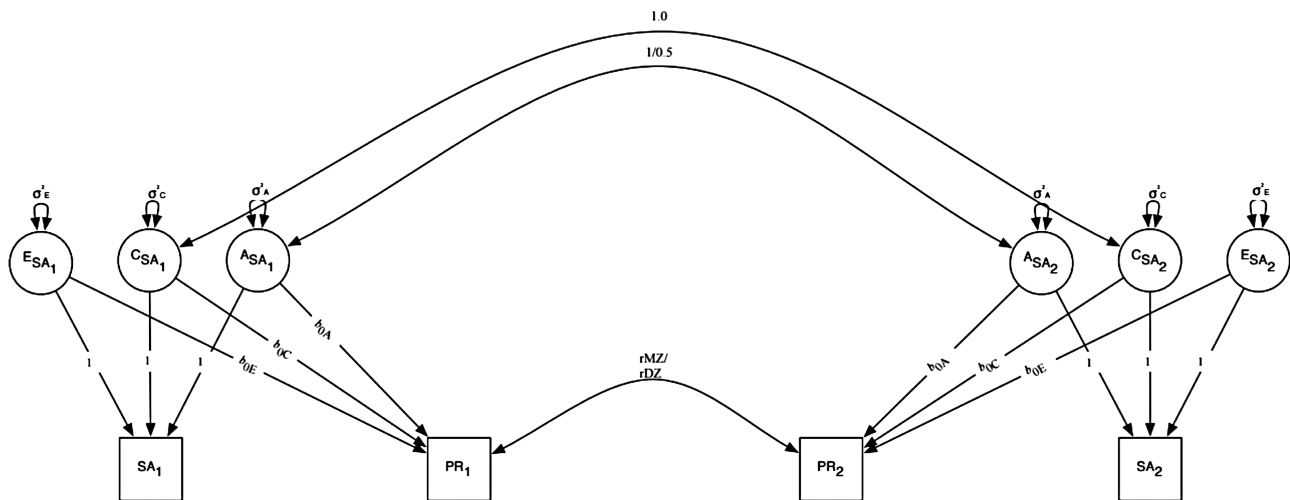
Fig. 1. Biometric regression model for adulthood socioeconomic adversity and allostatic load. Shared environmental components are constrained to 1.0 for monozygotic (MZ), dizygotic (DZ), and opposite sex (DZOS) twin pairs. Additive genetic components are constrained to 1.0 for MZ and 0.5 for DZ and DZOS twin pairs. SA_1 = Twin 1 socioeconomic adversity; PR_1 = Twin 1 physiological risk; E_{SA_1} = Twin 1 unique environmental component of socioeconomic adversity; C_{SA_1} = Twin 1 shared environmental component of socioeconomic adversity; A_{SA_1} = Twin 1 additive genetic component of socioeconomic adversity; E_{PR_1} = Twin 1 unique environmental component of physiological risk; C_{PR_1} = Twin 1 shared environmental component of physiological risk; A_{PR_1} = Twin 1 additive genetic component of physiological risk. Subscript 2 indicates the same latent and observed variables for Twin 2.

approximation (RMSEA, 0.05 is good and 0.08 is acceptable fit) [31], the Tucker-Lewis Index (TLI; greater than 0.95 is good fit) [32], Akaike Information Criterion (AIC; lower values imply better fit), and Bayesian Information Criterion (BIC; lower values imply better fit). Given the small sample size and non-normal distribution of the socioeconomic adversity, we used the Satorra-Bentler χ^2 (S-B χ^2) difference test [33] to compare nested models (e.g., comparing ACE to AE models and models in which male and female parameters were freely estimated or equated).

Results

The analytic samples, first the full Biomarker and then the twin subset, are described separately by gender in Table 1. Both men and women had, on average, low levels of physiological risk. Participants generally reported lower socioeconomic adversity in childhood than adulthood. In the full Biomarker sample, women had higher socioeconomic adversity than men in adulthood [$t = -4.21_{(1026.5)}$, $p < .0001$]. There were no other gender differences in the full Biomarker sample, and a similar pattern was observed among the subset of twins, with greater adulthood socioeconomic adversity among women than men.

Our first goal was to examine links between socioeconomic adversity and physiological risk in adulthood and to investigate potential gender differences in this hypothesized link. As shown in Table 2, results of Model 1 indicated that greater socioeconomic adversity

Table 2 Generalized estimating equations predicting multisystem physiological risk among full Biomarker sample ($n = 1,039$)

	Model 1	Model 2
Intercept	1.73*** (0.04)	1.74*** (0.04)
Adulthood socioeconomic adversity	0.17*** (0.03)	0.11** (0.04)
Childhood socioeconomic adversity	0.07** (0.03)	0.08** (0.03)
Age	0.03*** (0.003)	0.03*** (0.003)
Gender	−0.04 (0.06)	−0.04 (0.06)
Adulthood socioeconomic adversity × gender		0.13* (0.06)

Age was centered around the sample mean. To maximize comparison with results presented in Gruenewald et al. [4], adulthood socioeconomic adversity and childhood socioeconomic adversity were transformed into z-scores. Gender was coded so that female = 0 and male = 1.

*$p < .05$, **$p < .01$, ***$p < .001$.

in both childhood and adulthood and older age were significantly associated with higher physiological risk. Additionally, results of Model 2 indicated a significant interaction with gender, suggesting that the link between adulthood socioeconomic adversity and physiological risk was stronger among men than women (Fig. 2). Simple slopes analysis indicated that the effect of adulthood socioeconomic adversity on physiological risk in both men ($b = 0.24$, $SE = 0.05$, $t = 5.19$, $p < .05$) and women ($b = 0.11$, $SE = 0.04$, $t = 2.89$, $p < .05$) was statistically significant. The unstandardized difference effect, however, was more than twice as great in men
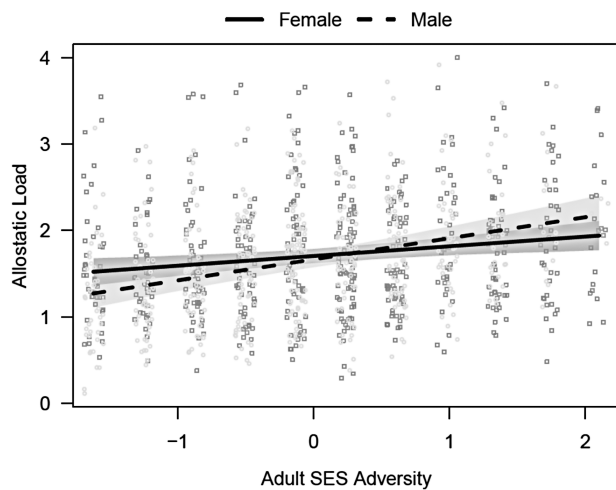
Fig. 2. Socioeconomic adversity × sex interaction on physiological dysregulation in the full sample. *SES* socioeconomic status.

Table 3 Socioeconomic adversity and physiological risk within-pair and cross-twin, cross-trait correlations

	Socioeconomic adversity	Physiological risk	Socioeconomic adversity-Physiological risk
MZM	0.49	0.50	0.14
DZM	0.33	0.35	0.69
MZF	0.34	0.56	0.04
DZF	0.30	0.13	0.19
DZOS	0.08	0.49	0.10

MZM monozygotic male; *MZF* monozygotic female; *DZM* dizygotic male; *DZF* dizygotic female; *DZOS* dizygotic opposite sex pair.

as in women and was statistically significant ($b = 0.13$, $SE = 0.06$, $t = 2.18$, $p < .05$).

Our second goal was to examine whether the association between socioeconomic adversity and physiological risk persisted after taking into account potential familial selection confounds. Twin correlations are shown in Table 3. Among men, there was evidence for both additive genetic and shared environmental influences on socioeconomic adversity and physiological risk, as the MZ twin correlations were less than double those of the DZ twin correlations. Although the same could be said among women for socioeconomic adversity, there was a stronger additive genetic influence on physiological risk among female twin pairs, given that DZ twin correlations were less than half that of MZ twin pairs. Cross-twin, cross-trait correlations indicated that common familial processes explain at least some of the association between socioeconomic adversity and physiological risk; among both men and women, MZ and DZ correlations were generally larger than DZOS correlations.

Before reporting the results of the biometric regressions, the results of a series of tests of model fit will be

Table 4 Unstandardized parameter estimates for multiple regression and biometric models of adulthood socioeconomic adversity and multisystem physiological risk

	Men	Women
Regression estimates		
Multiple regression		
b_{phen}	0.11*** (0.03)	0.01 (0.03)
Full biometric model (Model 1)		
b_{0A}	0.03 (0.08)	0.16 (0.34)
b_{0C}	0.25 (0.57)	−0.04 (0.18)
b_{0E}	0.16** (0.06)	−0.00 (0.04)
AE biometric model (Model 2)		
b_{0A}	0.03 (0.06)	0.03 (0.08)
b_{0C}	0.03 (0.06)	0.03 (0.08)
b_{0E}	0.16** (0.06)	0.00 (0.04)
Covariates		
Childhood SES$_1$	0.10 (0.06)	0.01 (0.07)
Childhood SES$_2$	0.04 (0.06)	0.04 (0.06)
Age	0.30*** (0.06)	0.43*** (0.06)

b_{phen} is the full phenotypic effect; b_A and b_C are indirect effects of socioeconomic adversity on allostatic load.

*$p < .05$; **$p < .01$; ***$p < .001$.

described. The baseline model, Model 1, suggested adequate fit to the data ($X^2 = 171.77$, $df = 124$; TLI = 0.86; RMSEA = 0.05; AIC = 12,121.0; BIC = 12,353.30). Model 2, however, provided better fit to the data, as indicated by a nonsignificant S-B χ^2 difference test and lower AIC and BIC values ($X^2 = 171.86$; $df = 126$, ΔS-B $\chi^2 = 0.28$, $\Delta df = 2$, $p = 0.87$; TLI = 0.87; RMSEA = 0.05; AIC = 12118.10; BIC = 12340.5). Model 2 suggests that A and C effects cannot be disentangled. Finally, Model 3 was compared to Model 2 to test for gender differences. The S-B χ^2 difference test was statistically significant and the TLI, RMSEA, and AIC values were worse for Model 3 than Model 2 ($X^2 = 181.96$, $df = 128$, ΔS-B $\chi^2 = 42.08$; $\Delta df = 2$, $p < .001$; TLI = 0.85; RMSEA = 0.06; AIC = 12121.50; BIC = 12334.80). Model 2, thus, was considered the best fitting model, that is, equating A and C within gender.

We next report the parameter estimates of the biometric models in Table 4. In the phenotypic model, greater adulthood socioeconomic adversity but not childhood socioeconomic adversity was related to higher physiological risk among men only, a finding that is similar to the overall Biomarker sample (Table 2). No significant effect of adulthood socioeconomic adversity on physiological risk was observed in the female twins. Older age was significantly associated with higher physiological risk among both men and women. Results

of the full biometric model (Model 1) and the constrained biometric model (Model 2) indicated that the relationship between socioeconomic adversity and higher physiological risk among men persisted after adjusting for familial confounds.

Discussion

Prior studies of unrelated individuals have found greater physiological risk among those with lower SES [34, 35]. Recent research suggests, however, that relations between lower education and greater allostatic load are not causal, but explained by shared genetic and environmental processes [7]. There are several reasons why we might expect twins raised in the same family to be similar on SES. First, growing evidence in both molecular and behavioral genetic fields indicates that social characteristics, including those highly relevant for SES such as years of education, have a genetic component [15] and are heritable [14]. Research has further illustrated that health outcomes, including measures of physiological risk, also have a genetic basis [16–18]. Certain genetic markers increase the risk for various cardiovascular outcomes [36–38], outcomes which are often shared by members of the same family [39]. It is, therefore, possible that common genetic markers may confound SES-health associations.

Despite the need for quasi-causal models to further investigate these controversial findings, few data sets, to our knowledge, have large samples of twins *and* adequate biological data. Using the small set of male and female twins in the MIDUS II Biomarker Study, the purpose of the present study was to leverage twin SES discordance to examine whether unique experiences in adulthood (i.e., SES) among members of twin pairs who share some degree of their genetic inheritance and early environments may explain twin health differences. We observed greater levels of physiological risk among those with lower adult SES among men, adjusting for additive genetic and shared environmental factors. This association was not observed among women. The unique environmental effect observed among men in our study is consistent with the argument that greater socioeconomic adversity results in greater physiological risk among men. Replications with larger samples of twins are needed to demonstrate the robustness of this observed sex variation.

Socioeconomic adversity in both childhood and adulthood is independently associated with physiological risk, with greater socioeconomic adversity linked to greater physiological dysregulation [4]. We replicated this finding in the present study when using a large sample of adults. When we extended this investigation to the subset of twins in our sample, only socioeconomic adversity in adulthood was significantly associated with greater physiological risk. However, the null effect for childhood socioeconomic adversity is because twins raised in the same family share their early SES; aside from twin-pair variance due to recall bias, twins should theoretically have the same childhood SES.

Why the Gender Difference?

Gender differences in the effects of SES on health have been observed in various international studies, including those that utilize health data before and after implementation of educational policies [22] and data collected from twin pairs [23]. Significant associations between years of schooling and both overweight status and mortality have been observed, but only among men [22, 23]. Consistent with these findings, in the present study, greater socioeconomic adversity predicted higher physiological risk among men, but not women. Results of the simple slope analysis suggested that the effects of adult socioeconomic disadvantage on physiological risk is more than twice as great in men as in women.

Our results suggest a possible causal role of socioeconomic adversity including education and income in relation to a scale of physiological dysregulation, particularly for men. Although we acknowledge that the effect of socioeconomic adversity on physiological dysregulation among men in this study is small, with one whole unit difference in physiological scores when comparing men at the lowest and highest range of socioeconomic adversity, we believe this difference holds clinical meaning. First, and although not reported in Table 4, we observed that every one-standard-deviation increase in adulthood socioeconomic adversity was related to an increase in physiological dysregulation of about a quarter standard deviation among men. Second, indices of physiological dysregulation, like the ones included in the present measure, often co-occur [40]. Obesity, for instance, is known to involve a "compendium" of additional physiological CVD risk, including hyperglycemia, a pro-inflammatory state, atherosclerosis, hypertension, elevated adrenergic activity, and dyslipidemia. Importantly, each physiological factor contributes independent risk for CVD [41]. As such, elevated risk in one index of physiological functioning may set in motion a cascade of additional pathophysiology leading to disease. Our hope is that the findings reported in the present study will encourage further investigation of SES-physiological health links, particularly among older samples of men among whom greater pathophysiological accumulation is likely to have occurred, and greater variability in physiological health likely will be observed.

The observed gender difference may be explained by characteristics of the cohort of adults used in the present analyses, born between 1921 and 1970. Some have argued that gender differences in the relation between socioeconomic adversity and health may reflect the opportunity to shift from blue- to white-collar work among men, whereas women—at least those represented in the current sample of midlife to older adults——were less likely to be employed [22]. Moreover, men in the current sample, perhaps more so than those born after the period encompassed between 1921 and 1970, may maintain conventional gender ideologies regarding the provider role [42], with low SES providing a more substantial threat to men' sense of self-worth. As such, it is possible that the threat to self-worth among male providers in the current study resulted in an activation of multiple physiological regulatory systems and greater physiological deterioration among men, relative to women.

The present data set provided a unique opportunity to examine both the SES and biological (genetic) factors that may predict health, and gender differences in health. Although one of the primary aims was to determine whether the SES-health link persisted after adjusting for familial confounds, another was to determine whether there are gender-specific patterns of genetic prediction of SES and health as well as gender-specific patterns of SES-health relationships. We are interpreting the observed interaction between SES and sex on physiological risk to mean that men and women, particularly in this older cohort of adults, were not raised in similar environments with regard to access to education, occupations, or higher incomes. The MIDUS II sample used in the present analyses represents a generation of people in which women were generally homemakers, and men were expected to obtain college degrees and financially support their families. If women in this cohort were raised in environments that did not support the same educational and professional outcomes afforded to men, it is possible that their genetic predispositions for these pursuits had been attenuated. This model of environmental modulation of genetic influence has been observed elsewhere [40], where there is a stronger cognitive genotype-phenotype association among children raised in affluent homes, relative to those raised in impoverished homes.

Limitations and Future Directions

Despite using a twin design to adjust for genetic and environmental selection effects, reverse causation and unobserved third variables are of concern [3]. Moreover, given the small size of our twin sample, we were unable to include an extensive number of covariates used in investigations among unrelated individuals [4]. The small sample of twins available in the MIDUS II Biomarker study also limited the ability to model twin correlations with great precision. Replications of the work in the current study should be extended to larger twin registers, and/or should use additional quasi-experiments that assist when random assignment is implausible. Additionally, we have argued that the gender difference in the present study may be cohort-specific. It may also be the case, however, that the MIDUS II twin sample is not representative of the population of U.S. adults. Future research should examine the robustness of—and explore factors that may explain—the observed gender difference, address whether specific aspects of SES relate to health differentially between men and women, and examine relations between socioeconomic adversity and physiological risk among more recent cohorts. Our sample is ethnically homogenous, and given a vast literature describing racial/ethnic differences in SES and health, replications with more representative samples are needed.

Supplementary Material

Supplementary material is available at *Annals of Behavioral Medicine* online.

Compliance With Ethical Standards

Authors' Statement of Conflict of Interest and Adherence to Ethical Standards Authors Jennifer W. Robinette, Christopher R. Beam, and Tara L. Gruenewald declare that they have no conflict of interest. All procedures, including the informed consent process, were conducted in accordance with the ethical standards of the responsible committee on human experimentation (institutional and national) and with the Helsinki Declaration of 1975, as revised in 2000.

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