Published in final edited form as:

Psychosom Med. 2018 April ; 80(3): 278–293. doi:10.1097/PSY.0000000000000561.

Socioeconomic Status and Cardiovascular Responses to Standardized Stressors: A Systematic Review and Meta-Analysis

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Abstract

Objective—Disparities in cardiovascular health by socioeconomic status (SES) are a pressing public health concern. Hypothesized mechanisms linking low SES to poor health are large cardiovascular responses to and delayed recovery from psychological stress. The current study presents a meta-analysis of the literature on the association of SES with blood pressure and heart rate reactivity to and recovery from acute stress tasks.

Methods—The PubMed database was searched, and 26 unique studies with relevant data were identified ($k = 25$ reactivity ($n = 14,617$), $k = 6$ recovery ($n = 1,324$)).

Results—Using random effects models, no significant association between SES and cardiovascular reactivity to stress emerged ($r = .008$, 95% CI: $-.02, .04$), although higher SES was associated with better recovery from stress ($r = -.14$, 95% CI: $-.23, -.05$). Stressor type moderated the reactivity effect, wherein higher SES was associated with greater reactivity to cognitive stressors ($r = .036$, $p = .024$), not with reactivity to interpersonal stressors ($r = -.02$, $p = .62$), but was associated with lower reactivity to tasks with combinations of cognitive, interpersonal, and physical challenges ($r = -.12$, $p = .029$). Accounting for publication bias revealed a significant association between SES and reactivity in the opposite direction of hypotheses.

Conclusions—Cardiovascular recovery from acute stress, but not reactivity to stress, may be a key pathway between low SES and risk for cardiovascular diseases. Heterogeneity in effect size and direction, challenges related to working across temporal dynamics, and recommendations for future research are discussed.

Keywords

socioeconomic status; cardiovascular reactivity; cardiovascular recovery; blood pressure; heart rate

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The authors declare no conflicts of interest.

INTRODUCTION

Disparities in health as a function of socioeconomic status (SES) are a worldwide phenomenon. Within the United States, it is estimated that billions of dollars in health care expenditures are directly and indirectly related to socioeconomic and racial disparities in health (1). Addressing these disparities represents a top public health priority, as indicated in the Healthy People 2020 guidelines. Individuals from disadvantaged backgrounds or those with less education, income, or less prestigious occupational status exhibit shorter life spans, higher rates of most chronic diseases, and show more adverse profiles across a number of subclinical measures and physiological markers of disease risk (2–7). This association is monotonic in nature, spanning across the spectrum of SES. Similar patterns emerge when SES is assessed during childhood, rather than adulthood, and some evidence suggests that childhood SES is associated with poor physical health in adulthood independent of adult SES (8,9).

There are myriad factors contributing to the association between lower SES and increased health risks, including less access to and poorer quality of health care, more environmental exposures, and more adverse psychosocial factors (2). These environmental, interpersonal, and intrapersonal differences affect behavioral and physiological pathways that have more proximal effects on health. With a goal of identifying biological mechanisms that underlie the link between SES and health, plausible physiological processes need to be sensitive to the environmental and psychosocial contexts that vary across socioeconomic strata and play a role in disease pathology. Candidate biological pathways include the autonomic nervous system (ANS), the hypothalamic-pituitary-adrenal (HPA) axis, and the immune system, specifically related to inflammatory processes (10–12). These systems mount coordinated physiological responses to prepare the body to respond to acute physical stressors. As such, their actions mobilize energy and direct important resources, such as oxygen and glucose, to the brain and muscles, and prepare to deal with acute infection or injury. However, repeated activation or prolonged activity in these stress response systems, which are also activated by psychological stressors, has physiological costs. Alterations in HPA axis and ANS functioning precede and contribute to dysregulation in other physiological systems, including immune and metabolic outcomes (13), and immune and metabolic dysregulation further contribute to chronic disease pathology, such as cardiovascular disease (CVD).

The aim of the current paper is to synthesize evidence for one of these biological pathways thought to contribute to socioeconomic disparities in health: cardiovascular stress physiology. Specifically, the main objective is to evaluate the evidence linking SES to blood pressure and heart rate responses to standardized stressors, a response coordinated primarily by the ANS. CVD is the leading cause of death in the United States, and socioeconomic disparities in cardiovascular morbidity and mortality are dramatic. For example, college graduates show nearly a 6-year longer life expectancy at age 25 than adults without a high school degree, and evidence suggests consistent income and education gradients in health status, rates of diabetes, and coronary heart disease (4). Despite theoretical models implicating ANS and cardiovascular functioning as mechanisms linking SES to morbidity and mortality (2,10,11), the empirical literature is mixed, and there has not been a quantitative summary of the empirical support between SES and blood pressure and heart

rate responses to stress to date. Here we examine whether SES predicts consistent changes in systolic blood pressure (SBP), diastolic blood pressure (DBP), and heart rate (HR) in response to and following acute standardized stressors.

Researchers employ a variety of tasks to elicit physiological stress responses. A key assumption that researchers rely on is that the ways in which physiological systems respond to the contrived stressors in the lab mirrors the reactivity and recovery processes that occur in the real world (14). With relevance to SES, it is important that the laboratory challenges are equally familiar and challenging across socioeconomic strata (15). Common psychological stimuli include public speaking with or without a social evaluation component, arithmetic, or other cognitive challenges, such as mirror tracing or video games. Other researchers use the anticipation of exercise or a physiological challenge (e.g., cold pressor task) as a stimulus to elicit a cardiovascular response. Despite their diversity, each of these tasks reliably lead to measurable increases in blood pressure and heart rate (16). In addition, despite potential issues of generalizability, examining physiological systems in response to acute stress has important advantages related to experimental control and precision of measurement of individual differences in reactivity and recovery.

There are considerable individual differences in magnitude of reactivity to a laboratory stressor as well as individual differences in the rate of recovery following stressor termination (17). The reactivity hypothesis posits that prolonged or exaggerated cardiovascular reactivity to psychological stress can promote the development of CVD due to structural and functional changes in the heart (18). Further, cardiovascular responses to acute stress may cause endothelial dysfunction (19), and impaired cardiovascular recovery is associated with elevated fibrinogen and interleukin-6 (20), both of which contribute to atherosclerosis and thrombus development. Finally, and perhaps most importantly, epidemiological data show that individual differences in blood pressure and heart rate reactivity and recovery predict subsequent hypertension and coronary heart disease (e.g., (21,23,24)). The effect sizes reported tend to be small to medium, and they remain significant over extended follow-up periods (i.e., 10 years). Relative to the reactivity literature, a much smaller literature has examined poor cardiovascular recovery from stress as a risk factor for adverse outcomes. Nonetheless, recent meta-analyses of this literature find that impaired recovery is associated with later adverse cardiovascular outcomes, and that the effect sizes are comparable with that for reactivity (21,23). The associations between recovery and later cardiovascular outcomes were stronger among older adults and were stronger when the stressor task was physical, rather than psychological, in nature.

It has not yet been established if low SES reliably predicts exaggerated blood pressure and heart rate reactivity or poor recovery from stress. Therefore, the objective of the present study is to perform a meta-analysis of available data on differences in SBP, DBP, and HR reactivity and recovery, respectively, to acute stressors as a function of SES. We examine the overall direction and magnitude of the relevant effects and the impact of study characteristics on effect sizes (e.g., age of sample, measure of SES utilized). If individual differences in cardiovascular reactivity and/or recovery to stress are a plausible psychobiological pathway linking SES to CVD, then associations between SES and individual differences in blood pressure and heart rate responses to stress should be apparent. Specifically, current

explanatory models suggest that lower SES is associated with higher SBP, DBP, and HR during and following exposure to acute stress, reflecting exaggerated cardiovascular reactivity and poor cardiovascular recovery, respectively (11,15).

Method

Literature Review Strategy

The Preferred Reporting Items for Systematic reviews and Meta-analyses (PRISMA) guidelines were followed in conducting this review (25). To identify relevant articles, the PubMed database was searched between July 2016 and April 2017. The following search terms were utilized: (“socioeconomic status” OR “socioeconomic position” OR “social status” OR “education*” OR “income” OR “occupation*” OR “subjective social status” OR “social standing”) AND (“cardiovascular reactivity” OR “cardiovascular recovery” OR “blood pressure reactivity” OR “blood pressure recovery” OR “stress reactivity” OR “heart rate reactivity”). All reference lists of identified papers were further scanned to identify other potentially relevant manuscripts. The search returned 201 possible abstracts (see Figure 1 for the number of articles identified, screened, and included as well as the reasons for exclusion). Identified studies were screened and selected for inclusion based on the following criteria: (1) peer-reviewed article published in the English language, (2) described changes in either heart rate, systolic blood pressure, or diastolic blood pressure in response to a standardized psychological stressor, (3) included at least one measure of socioeconomic status at the individual or neighborhood level, and (4) sample comprised of either adolescents or adults (mean age of at least 11 years). Studies with child samples were not included given the focus on stress reactivity and recovery as mediating pathways from SES to disease outcomes that typically occur in midlife and old age, as well as concerns of developmental maturation affecting results and needing to ensure that stress tasks were developmentally appropriate across age groups. Studies that examined only heart rate variability (HRV) reactivity or recovery to stress or impedance cardiography were excluded, as the link between stress-induced phasic changes in these measures and cardiovascular disease is not as well established (21). Further, HRV and impedance cardiography parameters are underlying autonomic determinants of HR and BP thought to influence physical health through their eventual influence on HR and blood pressure, which are directly assessed here. A total of 94 full-text articles were assessed for eligibility, and 26 unique studies were included in the meta-analysis (25 for reactivity; 6 for recovery). In total, 68 articles were excluded based on not including SES ($n = 16$), not reporting or providing relevant effects ($n = 29$), utilizing a child sample ($n = 10$), not including appropriate SBP, DBP, or HR measures ($n = 5$), presenting data from a shared sample obtained from another (included) study ($n = 6$), and not publishing in the English language ($n = 2$). Studies that were retrieved and excluded are listed in Table S1 (Supplemental Digital Content 1).

Data Extraction

The first author performed the data searches and coding in consultation with the second and third authors. Further, the first and second authors reviewed the coding of all studies prior to the final analysis to check for errors and resolved any discrepancies. The following information was extracted from each study: (1) sample size; (2) sample demographics (age,

sex, racial makeup); (3) study design (prospective/longitudinal v. cross-sectional collection of SES measures relative to outcome); (4) outcome (HR, SBP, or DBP reactivity or recovery); (5) type of acute stressor (cognitive, interpersonal, physical, or combination of the aforementioned types); (6) SES measure (Hollingshead index, education, occupation, income, neighborhood SES); (7) reactivity/recovery calculation (change score v. residualized change); (8) SES analytic strategy (i.e., continuous SES compared to group comparisons); (9) study focus (stated study aim to assess SES and cardiovascular reactivity/recovery v. not a stated aim; it's important to note that this is independent of whether data collection was designed to test SES differences); and (10) total number of covariates included in analytic models. When data could not be obtained from the original article, the corresponding author was contacted by email two times, with at least one month between communications. If no data could be obtained, the study was not included in the analysis. Detailed information about the included studies for the reactivity and recovery meta-analysis are presented in Tables 1 and 2, respectively.

Analysis Overview

For each outcome examined, we extracted the reported effect size. Effect sizes were reported as Pearson correlations, regression coefficients, odds ratios (OR), or standardized mean differences (Cohen's *d*). The direction of effects was consistently coded such that positive values indicate a positive relationship between SES and reactivity/recovery (e.g., those with higher SES had higher HR or blood pressure in reaction to the task or in the recovery post-task). Data were analyzed using Comprehensive Meta-Analysis (CMA) software, version 3.0 (BiostatTM, USA). Random effects modeling was employed to examine associations between SES and SBP, DBP, and HR reactivity and recovery outcomes, respectively. Random effects models are appropriate, given the variation in effect sizes and populations included, and they give relatively less weight to sample size and appear to produce more accurate confidence intervals than fixed-effects models (26,27).

The heterogeneity statistic Q_T was used to determine an estimate of the variability of effect sizes. If Q_T was significant, several moderating effects were considered as potential explanatory variables for the effect size variability. For reactivity, these included whether findings differed by racial composition of the sample, by outcome (HR v. SBP v. DBP), age group of the sample (adolescent v. adult sample), measure of SES (Hollingshead/education/occupation v. all other; neighborhood v. individual level indicators), study design (prospective or longitudinal v. not), stress task type (cognitive v. interpersonal v. combination), SES analysis approach (continuous v. extreme groups), study aim (SES and reactivity/recovery was a stated key aim of study v. not) and reactivity/recovery calculation type (change score v. residualized change). For recovery, these included whether findings differed by racial composition of the sample, by outcome (HR v. SBP v. DBP), age group of the sample (adolescent v. adult sample), study design (prospective or longitudinal v. not), and SES measurement (continuous v. extreme groups). Fewer available studies and a lack of variability on certain moderators precluded considering all moderating variables for the recovery analysis. Tested moderator variables were considered significant when the test of the between groups difference (Q_M) was significant at $p < .05$.

Proposed guidelines were used to assess the presence and magnitude of publication bias (i.e., bias resulting from null and negative findings being less likely to be submitted for publication by authors or less likely to be accepted for publication by editors and reviewers; (28)). First, forest plots were examined for individual effects that appear to be outliers. Second, funnel plots of analyzed effects were examined, as these compare published effect sizes by sample size (29). Since funnel plots are based on a combination of statistical analyses and scientific judgment (28), we also statistically tested whether standard errors and standardized effect sizes are significantly associated using Kendall's tau (which is similar to a correlation; (30)) and Egger's regression (where the intercept reflects the slope of the association; (31)). If publication bias appeared to be present, we quantified the magnitude of this bias using Duval and Tweedie's (32) trim and fill procedure, which provides a bias-corrected estimate of the cumulative effect size.

Results

Is SES associated with cardiovascular reactivity to acute stress?

Results of random effects meta-analyses for SBP, DBP, and HR reactivity outcomes are presented in Table 3. There were 14,617 total participants in the reactivity analysis. A forest plot of the individual effects aggregated within study ($K = 25$) is presented in Figure 2 and a forest plot examining all effects individually ($k = 75$) is presented in Figure S1 (Supplemental Digital Content 2). Regardless of whether effects were aggregated within study or treated individually, the cumulative effect size for the association between SES and SBP, DBP, and HR reactivity to acute stress was positive and not significantly different from zero ($r = .010$ and $.008$, respectively, both $p > .10$; Table 3).

Is SES associated with cardiovascular recovery from acute stress?

Results of random effects meta-analyses for SBP, DBP, and HR recovery outcomes are presented in Table 4. There were 1,324 total participants in the recovery analysis. A forest plot of the individual effects aggregated within study ($K = 6$) is presented in Figure 3 and a forest plot examining all effects individually ($k = 17$) is presented in Figure S2 (Supplemental Digital Content 3). Regardless of whether effects were aggregated within study or treated individually, there was a significant, inverse association between SES and recovery from acute stress ($K = 6$, $r = -.14$, $p = .003$; $k = 17$, $r = -.11$, $p = .004$; Table 4). Specifically, higher SES individuals exhibited better blood pressure and heart recovery (i.e., lower BP and HR following an acute stressor).

Moderators

Results of moderator analyses are also presented in Table 3 (reactivity) and Table 4 (recovery). Analyses revealed two significant moderators of the cumulative association between SES and cardiovascular reactivity (Table 3). First, there was a significant difference in cumulative effect size depending on the nature of the stress task ($p = .013$). Specifically, SES was significantly and positively associated with reactivity to cognitive stress tasks ($k = 48$, $r = .036$, $p = .024$), significantly and negatively associated with reactivity to combination stress tasks ($k = 17$, $r = -.12$, $p = .029$), and not significantly associated with reactivity to interpersonal stressors ($k = 10$, $r = -.02$, $p = .62$). Second, there was a significant difference

in cumulative effect size depending on the method of calculating reactivity (i.e., difference score v. residualized change; $p = .016$). SES and reactivity were negatively associated when reactivity was calculated as a residual ($k = 14$, $r = -.07$, $p = .051$), whereas the SES-reactivity association was not significant when calculated as a change score ($k = 60$, $r = .02$, $p = .12$). There were no significant differences in cumulative effect size for reactivity as a function of outcome assessed (SBP, DBP, HR), race, age of sample (adolescent v. adult), study design (prospective or longitudinal v. not), measurement of SES (continuous v. extreme group comparison), SES measure analyzed (Hollingshead/education/occupation v. all others; neighborhood indices v. individual indices), or study aim (SES and reactivity as stated study aim v. not).

Moderator analyses also revealed one significant moderator of the cumulative association between SES and recovery (Table 4). Race moderated SES to recovery associations ($p < .001$), such that Black participants evidenced no significant association between SES and recovery ($k = 3$, $r = .13$, $p = .17$); however, White participants show a moderate inverse relationship ($k = 8$, $r = -.21$, $p < .001$), indicating that high SES was associated with better recovery. Notably, results for Blacks were based on only one independent sample and results for Whites were based on only three independent samples. There were no significant differences in cumulative effect size for recovery as a function of outcome assessed (SBP, DBP, HR), age of sample (adolescent v. adult), study design (prospective or longitudinal v. not), or measurement of SES (continuous v. extreme group comparison).

Publication Bias

First, we examined the forest plots (Figures 2–3; S1–S2) for individual effects that appear to be outliers and may significantly bias results. We found no obvious outliers for reactivity or recovery. Second, we examined a funnel plot of analyzed effects (Figures 4 and 5; (29)). Examination of the funnel plot for reactivity effects suggests a possibility for bias, such that smaller studies (i.e., studies with larger standard errors, closer to the bottom of the plot) evidenced somewhat larger effects (because effects would need to be larger to reach statistical significance) and were less symmetrically distributed around the mean, with effects in the hypothesized direction (e.g., higher SES $\rightarrow$ lower reactivity) more likely to be published. For these reactivity effects, results for Tau did suggest significant publication bias (Tau = $-.21$, $p = .007$); however, results for Egger's regression did not (Intercept = $-.44$, $SE = .38$, $p = .25$). Examination of the funnel plot for recovery effects does not strongly suggest the possibility of bias in published reports, as smaller studies do not show larger effects. Further, results for both Tau and Egger's regression did not suggest significant publication bias for recovery (Tau = $.07$, $p = .74$; Egger's regression: Intercept = 2.2 , $SE = 1.72$, $p = .29$).

Given ample evidence that our reactivity results may be influenced by publication bias, we evaluated the magnitude of this bias using Duval and Tweedie's (32) trim and fill procedure. Results revealed a larger and significantly positive bias-corrected estimated effect size ($r = .042$, 95% CI: $.01$ to $.07$) compared to our originally observed positive but non-significant estimate ($r = .008$, 95% CI: $-.02$ to $.04$), suggesting that publication bias may be present and

that the true, unbiased association between SES and reactivity may be positive, counter to predictions.

Discussion

The primary objective of this paper was to use meta-analysis to summarize the evidence linking SES to blood pressure and heart rate responses to stress. Despite the important public health implications of testing theoretical models of how SES “gets under the skin” to affect morbidity and mortality, empirical examples that incorporate predictors, mediators, and outcomes in the same model are few. Therefore, building on prior support for established links between cardiovascular reactivity and recovery to acute stress and later cardiovascular disease and mortality (21,23), the conclusions from the present study are that (a) low SES is not associated with higher BP and HR reactivity; (b) relationships are altered by type of stress such that higher SES is reliably associated with greater blood pressure and heart rate reactivity to acute cognitive stressors and lower SES is associated with greater blood pressure and heart rate reactivity to a combination of acute stressors (cognitive, interpersonal, and physical challenges), and (c) higher SES is associated with better blood pressure and heart rate recovery following acute stress. Therefore, based on the available evidence, we do not find robust support for cardiovascular *reactivity* as a mediator linking lower SES and downstream poor health at this time, although how the cardiovascular system *recovers* from acute stress may be an informative mechanism between SES and morbidity and mortality. Further, other intermediate pathways, such as health behaviors, HPA axis reactivity, or inflammation, may be stronger mediating processes between SES and health than blood pressure and heart rate reactivity (33).

Relative to the reactivity literature, fewer studies have investigated how cardiovascular recovery from stress may be linked with SES and poor health outcomes. This is an important oversight, as it is adaptive to mount a physiological reaction, though not excessively, when faced with an acute challenge or threat. However, failing to down-regulate this response upon stressor cessation may tax biological systems and cause wear-and-tear (e.g., (34)). In the context of emotion regulation and cortisol responses to stress, the recovery window is posited to be more strongly linked with downstream health (35–37). Indeed, consistent with the results of this meta-analysis, the few studies that have examined reactivity and recovery together indeed show stronger associations between SES and recovery than with reactivity (17,38). Results should be interpreted with caution, however, as the meta-analytic effect for recovery was based on only six independent studies. Further, we were unable to test length of recovery assessment or measurement timing (e.g., continuous vs. intermittent) as effect moderators given limited variability in these factors across recovery studies. Finally, while there was evidence that the recovery effect was not significant among Black participants but significantly stronger among White participants, small numbers of studies ($K = 1$ for Blacks, and $K = 3$ for Whites) preclude drawing conclusions regarding race as a moderator. Of the remaining recovery studies, Boylan and colleagues (38) reported findings in a mixed-race sample, barring its inclusion in the race moderation test. Importantly, Boylan and colleagues reported testing for race differences in associations between SES and recovery and failed to find empirical support for such differences. However, if the pattern for stronger associations between SES and recovery in Whites remained with more empirical support, it would be

consistent with prior literature indicating that higher SES Blacks do not derive the same health benefits from higher SES as their White counterparts (39–42).

Publication bias was evident in the reactivity literature examined here. After accounting for publication bias, a small, *positive* association between SES and reactivity emerged. That is, higher SES individuals demonstrated more exaggerated blood pressure and heart rate reactions to acute stressors. Further, it is noteworthy that there was considerable heterogeneity in the direction of effects for reactivity. Of the 25 studies with available reactivity data, ten of these reported at least one inverse effect (i.e., lower SES had more exaggerated blood pressure or heart rate responses), ten reported at least one positive effect, and seven reported at least one null effect.

Stressor type moderated the association between SES and reactivity. Higher SES was associated with *higher* reactivity to cognitive stress tasks but *lower* reactivity when reactivity was combined across multiple types of stress tasks (i.e., average reactivity across cognitive, physical, and/or interpersonal task). Reactivity to interpersonal stressors alone was not statistically significant though very few studies examined interpersonal stressors only ($K=10$). Of note, among the studies utilizing combination stressors ($K=17$), the majority ($K=11$) included at least one interpersonal stressor. Cognitive stressors (e.g., mental arithmetic) were the most common stressor type employed in this literature, including in large, epidemiological studies (i.e., CARDIA, Dutch Famine Birth Cohort Study, West of Scotland Twenty-07 Study). One contributing factor to lower SES being associated with greater reactivity in studies that combined across task type may be that this approach more reliably captures a reactivity assessment, accounting for distinct types of skills and greater flexibility to the different tasks.

The findings for reactivity raise important questions regarding the hypothesis that reactivity may mediate SES disparities in cardiovascular outcomes. Unfortunately, very few opportunities were available to empirically test the full mediation model of interest (i.e., SES to reactivity/recovery to disease). Despite theoretical interest in cardiovascular reactivity and recovery as a potential biological pathway contributing to SES disparities in disease (2,11,43), there are only a handful of studies with relevant data to test mediation, and these studies have not reported on such models to date. A previous meta-analysis found that both reactivity to and recovery from laboratory stressors, particularly cognitive stressors, were associated with subsequent (prospective) cardiovascular risk (21). This meta-analysis also reported that blood pressure reactivity was the primary predictor of subsequent CVD and not HR reactivity; relatively few studies evaluated HRV but they were by and large null. Extrapolating from the current meta-analysis, our results appear to support the viability of a pathway from lower SES to *lower* stress reactivity and, thus, better cardiovascular health. However, lower SES is clearly not protective for cardiovascular health (e.g., (4,44)). The most parsimonious conclusion is that while stress reactivity to cognitive tasks in the laboratory predicts future risk for CVD, which were predominantly hypertension and subclinical CVD (21), it is not a viable explanatory factor in the SES-CVD relationship. Potential explanations for why cognitive stress reactivity may not help explain the SES-CVD association are explored below.

The method used to calculate reactivity also moderated effect sizes. Among effects calculated with a residualized approach, there was a significant, inverse association between SES and blood pressure and heart rate reactivity, whereas the association between SES and reactivity calculated as a difference score was positive and not significantly different from zero. While 14 individual effects utilized a residual approach for calculating reactivity, these effects came from only 4 studies, as compared to 60 individual effects from 20 studies utilizing a difference score approach (half of which controlled for baseline values). As such, it is difficult to discern whether the residual approach is responsible for an inverse association between SES and reactivity, or if this finding is spurious due to the small number of studies. Reactivity calculation method was tested as an effect modifier given that adjustment for baseline parameters is handled differently with the two approaches. With a residualized approach, reactivity is, by definition, uncorrelated with baseline physiology. However, controlling for baseline physiology is not always done when using a difference score approach. Not adjusting for baseline parameters may change associations of interest with change score reactivity, especially if baseline physiology is correlated even modestly with change (45). In healthy samples, there is often regression to the mean, such that higher baseline, less reactivity, whereas in normotensives with a family history of hypertension or hypertensives, baseline and reactivity may be positively correlated (e.g. (46,47)). This results in interpretative difficulties because the greater changes during stress for those with higher blood pressure can be due to structural changes in the vessel based on Poiseuille's law rather than to a greater adrenergic response to stress.

A significant obstacle for research focused on uncovering pathways from something as pervasive as SES to health decades later pertains to temporal dynamics. The measures utilized in this meta-analysis operate on several different time-scales. Some measures of SES, such as educational attainment, and health risk factors are relatively stable across the adult life-course, or show change only over the long-term. Other SES measures, like income and occupational status, are relatively stable or change gradually over the years, though are also susceptible to abrupt changes more so among lower as compared to higher SES individuals. Blood pressure and heart rate reactivity and recovery, on the other hand, change within milliseconds to minutes. As such, we must consider the extent to which profiles of reactivity and recovery can be construed as traits, given the linkages to disease processes that develop over decades. There are several demonstrations of short-term reliability and stability of cardiovascular reactivity and recovery (reviewed in (16)), including in children and adolescents (48). A more recent study showed that blood pressure and heart rate responses to mental stress and cold pressor are stable over an 18-year follow-up among young men (49). However, the long-term reliability and consistency across settings and tasks, especially for recovery measures, have not been sufficiently demonstrated to date. Importantly, multiple assessments of both SES and reactivity and recovery are needed to determine if the association is truly prospective. While it is unlikely that cardiovascular reactivity and recovery would bias SES assessments, the validity of retrospective SES assessments may change over time, affecting how conclusions may be drawn from a causal modeling standpoint.

Another relevant concern is that laboratory stressors may not reflect real world stressors faced by lower SES individuals on important dimensions such as the magnitude and duration

of the stressor as well as available resources for coping (50). There may be limited engagement of physiological stress responses to artificial, lab-based stressors among low SES individuals, and determining what constitutes a challenge or threat may be differentially calibrated as a function of SES based on prior life experiences. Higher SES individuals may demonstrate greater reactivity to cognitive stressors, especially the commonly used serial subtraction task, due to greater task familiarity or engagement. We know from laboratory studies that tasks that are easy or hard are less likely to elicit substantial responses, compared to medium difficulty tasks (51). Hence, it is important that the stress tasks employed are equally familiar and challenging across socioeconomic strata (15,51). Future research may consider employing stress tasks that mimic real-world stressors faced by low and high SES individuals alike or that may disproportionately affect lower SES individuals, thus potentially explaining disparity (52). Notably, this may come at the cost of the benefits of standardization across labs and experimenter control. Researchers are also encouraged to obtain objective measures of task performance as well as ratings of task difficulty and to test for SES differences in these factors. Employing tasks that change difficulty level within person depending on performance are also appropriate. Finally, employing multiple types of stressors may also reduce potential effort and engagement bias related to SES.

In reviewing the SES-health literature, Matthews and colleagues (33) found limited support for stress, assessed primarily as self-reported life events or perceived stress, as a mediating pathway from SES to health. This finding along with the current finding that cognitive stress reactivity (used in most large epidemiological studies) does not appear to be a viable explanatory pathway, suggests that either our measurement of stressors or the types of stress typically measured are not capturing the reliable variance in stress physiology by SES. In other words, we appear to be measuring stress in a number of ways that do not usefully explain SES-health pathways. Research would likely benefit from exploration of novel measurement of stress and stress reactivity in hopes of uncovering stressors that do appear to be viable mediators of the SES-health association. Notably, higher reactivity to a combination of types of stressors was associated with lower SES in this meta-analysis, perhaps suggesting that assessment of reactivity to multiple types of stressors may improve the utility of blood pressure and heart rate reactivity in explaining SES disparities.

There are limitations of the present analysis that warrant additional consideration. First, nearly 30 studies with potentially appropriate data were not included in this meta-analysis because the relevant effects were not reported or provided by the authors upon request, which is more than half of all studies with relevant data (26 unique studies were included in the present meta-analysis). While this is a common issue for all meta-analyses, improving data sharing practices, such as providing a table of bivariate correlations for key study variables, may lessen this concern. Further, most of the associations included in the meta-analysis were cross-sectional in nature, making causal claims about SES and cardiovascular responses to stress untenable, despite prospective epidemiological evidence that reactivity and recovery are reliably linked to later disease and mortality outcomes. Though it presents unique challenges, it is imperative that future research addresses complete pathways from SES to mediators to health to allow for empirical comparisons across multiple mediators and contribute to eventual intervention development (11,43). Finally, we were unable to evaluate whether associations between SES and blood pressure and heart rate reactivity and recovery

differed by sex, as many studies only included men in their sample or did not provide sex-stratified associations.

In conclusion, this meta-analysis addressed whether there were consistent differences in systolic and diastolic blood pressure and heart rate in response to and following acute stress as a function of SES. We did not find significant meta-analytic associations between SES and blood pressure and heart rate reactivity, though estimated effects were significant and positive (higher SES to higher reactivity) after accounting for publication bias. This positive association was driven by reactivity to cognitive stress tasks. Taken together, results weaken the hypothesis that blood pressure and heart rate reactivity is a key intermediate pathway between low SES and risk for CVD and by inference suggest that additional biobehavioral mechanisms (e.g., health behaviors, HPA axis reactivity, inflammation) may be stronger intermediate pathways. However, prolonged blood pressure and heart rate recovery from acute stress was reliably linked with low SES. As such, this is an important avenue to pursue in future research, including how cardiovascular physiological parameters track with psychological and behavioral recovery processes following stressors. We have also identified several important directions for research in this area to consider as we continue to pursue how socioeconomic disparities in morbidity and mortality outcomes get under the skin to affect morbidity and mortality outcomes.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

This research was supported by the National Heart, Lung, and Blood Institute (HL07560).

Abbreviations

ANS	autonomic nervous system
CARDIA	Coronary Artery Risk Development in Young Adults
CVD	cardiovascular disease
DBP	diastolic blood pressure
HPA	hypothalamic-pituitary-adrenal
HR	heart rate
HRV	heart rate variability
OR	odds ratio
PRISMA	Preferred Reporting Items for Systematic reviews and Meta-analyses
SBP	systolic blood pressure
SES	socioeconomic status

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Figure 1.
Flow diagram of literature search and article selection process.

Figure 2.
Forest plot for all reactivity effects aggregated within study ($k = 25$).

Figure 3.
Forest plot of all recovery individual effects aggregated within study ($k = 6$).

Figure 4. Funnel plot for reactivity effects examining the likelihood of publication bias. Open circles are observed values and black circles are values imputed to correct for potential publication bias.

Figure 5.
Funnel plot for recovery effects.

Table 1
List of included studies for reactivity meta-analysis and description of sample and measures

First Author, Year (Ref)	Sample Characteristics	Measure of SES	Type of Stressor	Reactivity Outcomes	Covariates	Findings
Boylan, 2016 (38)	N = 246 100% male 55.7% Black M _{age} = 32.1 years	Parental Hollingshead	Cognitive	SBP, DBP, HR	Age, race, marital status, BMI, current smoking, current SES (Hollingshead), psychological resources, average task demand	Null
Brummett, 2009 (53)	N = 328 38.2% male M _{age} = 31.4 years	Income	Cognitive	SBP, DBP	Baseline systolic or diastolic blood pressure, BMI, smoking status, race, sex, age, positive affect, negative affect	Inverse
Carroll, 1997 (54)	Whitehall II cohort N = 1,091 100% male M _{age} = 43.6 years	Occupation	Cognitive	SBP, DBP	Age, gender	Inverse
Carroll, 2000 (55)	West of Scotland Twenty-07 Study N = 1,657 45.8% male 100% White n = 594 23 year olds; n = 630 43 year olds; n = 433 63 year olds	Occupation	Cognitive	SBP, DBP	Age, task performance	Positive
Chen, 2001 (56)	N = 90 42.2% male 45.6% African American M _{age} = 13.6 years	Paternal Hollingshead	Cognitive Physical	SBP, HR	Concurrent resting systolic blood pressure or heart rate, Systolic blood pressure or heart rate reactivity from wave 1	Inverse
Chen, 2004 (57)	N = 100 36% male 53% African American M _{age} = 17.3 years	Parental Hollingshead	Interpersonal	SBP, DBP, HR	Race	Inverse (DBP and HR reactivity)
de Rooij, 2012 (58)	Dutch Famine Birth Cohort N = 665 47% male M _{age} = 58 years	Education	Cognitive	SBP, DBP, HR	None	Positive
Evans, 2007 (59)	N = 207 52% male 100% White M _{age} = 13.4 years	Poverty duration	Cognitive	SBP, DBP	None	Positive

First Author, Year (Ref)	Sample Characteristics	Measure of SES	Type of Stressor	Reactivity Outcomes	Covariates	Findings
Evans, 2013 (60)	$N = 185$ 53% male 95.1% White $M_{age} = 17.3$ years	Income to needs ratio	Cognitive	SBP, DBP	Age, sex, race	Positive
Evans, 2015 (61)	$N = 428$ 51% male $M_{age} = 15.6$ years	Parental occupation	Cognitive Interpersonal	HR	Age, sex, BMI, urbanicity	Positive
Everson, 2001 (62)	Kuopio Ischemic Heart Disease Risk Factor Study $N = 2,303$ 100% male $M_{age} = 52.8$ years	Education	Cognitive	SBP, DBP	Age, resting systolic blood pressure, BMI, alcohol, smoking, HDL and LDL cholesterol, fibrinogen, prevalence of diabetes, use of medication for hypertension or hypercholesterolemia	Inverse
Gianaros, 2002 (63)	$N = 251$ 100% male 78.5% White $M_{age} = 56.1$ years	Education	Cognitive	SBP, DBP, HR	None	Null
Gordon, 2016 (64)	$N = 133$ 53.3% male 43.6% African American $M_{age} = 37.4$ years	Education	Interpersonal	SBP, HR	Discrimination, childhood sexual abuse, tangible support, restraint coping, turning to religion, church attendance, church support, importance of faith	Positive
Gump, 1999 (65)	$N = 147$ ~50% male 52.4% African American ~50% 8–10 years, ~50% 15–17 years	Hollingshead Financial strain Neighborhood SES composite	Cognitive and Physical	Factor score with SBP, DBP, Total peripheral resistance	Age, sex	Inverse
Jackson, 1999 (66)	$N = 272$ 50.4% male 59.6% Black $M_{age} = 13.5$ years	Neighborhood SES composite	Cognitive and Interpersonal	SBP, DBP, HR		Inverse for Whites, Positive for Blacks
Kapuku, 2002 (67)	$N = 24$ 100% male 100% African American $M_{age} = 18.8$ years	Hollingshead Neighborhood SES composite	Cognitive and Physical	SBP, DBP	None	Inverse
Lovallo, 2012 (68)	Oklahoma Family Health Patterns Project $N = 354$ 44.6% male >70% White	Education Hollingshead	Cognitive and Interpersonal	HR	None	Null

First Author, Year (Ref)	Sample Characteristics	Measure of SES	Type of Stressor	Reactivity Outcomes	Covariates	Findings
	$M_{age} = 23.1-24.6$ years					
Matthews, 2006 (69)	Coronary Artery Risk Develop in Young Adults Study (CARDIA) $N = 4,126$ 49.7% male 54.0% Black $M_{age} = 27$ years	Education	Cognitive	SBP, DBP	None	SBP reactivity: Positive DBP reactivity: Null
McLaughlin, 2014 (70)	$N = 157$ 44.0% male 40.8% White $M_{age} = 14.9$ years	Poverty status	Interpersonal; Cognitive	SBP, DBP, HR	None	Null
Owens, 1993 (71)	$N = 49$ 30.6% male 100% White $M_{age} = 44.1-49.7$ years	Education Hollingshead	Cognitive, Interpersonal, and Physical	SBP, DBP, HR	BMI	Inverse
Shaffer, 2012 (72)	1995 Nova Scotia Health Survey $N = 1,470$ 50.7% male $M_{age} = 45.31$ years	High school education (y/n)	Interpersonal	SBP, DBP	None	Null
Suchday, 2005 (73)	$N = 83$ patients with known coronary artery disease 86.7% male 70.1% White $M_{age} = 61.8$ years	Education Neighborhood Per Capita Income	Cognitive	SBP, DBP, HR	Baseline blood pressure, age, sex, race, BMI, marital status, medication usage	Positive
Williams, 2008 (74)	$N = 165$ 55.2% male 57.0% African American $M_{age} = 35.1$ years	Education	Cognitive	SBP, DBP, HR	Father's education	Inverse
Wilson, 2000 (75)	$N = 76$ 46.1% male 100% African American $M_{age} = 14$ years	Parental education, income, neighborhood SES composite	Interpersonal	SBP, HR	BMI, family history of essential hypertension	Inverse
Wright, 2005 (76)	$N = 133$ 42.9% male $M_{age} = 69.8-71.2$ years	Education	Cognitive	SBP, DBP, HR	None	Null

Note. BMI = body mass index. Ref = reference number. "Positive" findings indicate a positive relationship between SES and reactivity (e.g., those with higher SES had higher HR or BP in reaction to the task). Additional information was extracted from each study, including study design (prospective/longitudinal v. cross-sectional), SES analytic strategy (i.e., continuous SES compared to group comparisons), study focus (stated aim to assess SES and cardiovascular reactivity/recovery v. not a stated aim), and method of calculating reactivity (residualized change v. difference score). This information is available from the authors upon request.

Table 2

List of included studies for recovery meta-analysis and description of sample and measures

First Author, Year (Ref)	Sample Characteristics	Measure of SES	Type of Stressor	Recovery Outcomes	Covariates	Findings
Boylan, 2016 (38)	N= 246 100% male 55.7% Black $M_{age} = 32.1$ years	Hollingshead	Cognitive	SBP, DBP, HR	Age, race, marital status, BMI, current smoking, current SES (Hollingshead), psychological resources, average task demand	Inverse (SBP and HR recovery)
Evans, 2013 (60)	N= 185 53% male 95.1% White $M_{age}=17.3$ years	Income to needs ratio	Cognitive	SBP, DBP	Age, sex, race	Inverse
Evans, 2015 (61)	N= 428 51% male $M_{age}=15.6$ years	Parental occupation	Cognitive Interpersonal	HR	Age, sex, BMI, urbanicity	Inverse
Jackson, 1999 (66)	N= 272 50.4% male 59.6% Black $M_{age} = 13.5$ years	Neighborhood SES composite	Cognitive and Interpersonal	SBP, DBP, HR		Inverse for Whites, Positive for Blacks
Steptoe, 2002 (17)	Whitehall II study N= 228 53.9% male $M_{age}=50.9-53.9$ years	Employment grade	Cognitive	SBP, DBP	Sex, age, baseline blood pressure, task reactions, stress during tasks	Inverse
Wright, 2005 (76)	N= 133 42.9% male $M_{age}: 69.8-71.2$ years	Education	Cognitive	SBP, DBP, HR	None	Null

Note. BMI = body mass index. Ref = reference number. "Positive" findings indicate a positive relationship between SES and recovery (e.g., those with higher SES had higher HR or BP in the recovery post-task). Additional information was extracted from each study, including study design (prospective/longitudinal v. cross-sectional), SES analytic strategy (i.e., continuous SES compared to group comparisons), study focus (stated aim to assess SES and cardiovascular reactivity/recovery v. not a stated aim), and method of calculating reactivity (residualized change v. difference score). This information is available from the authors upon request.

Table 3

Cumulative effect sizes of the association between SES and cardiovascular reactivity.

	Cumulative Effect Size (<i>r</i>)	95% CI	<i>Q_T</i> (heterogeneity)
All effects (<i>k</i> = 75)	.008	-.02, .04	313.1 *
Using study as the unit of analysis (<i>K</i> =25)	.010	-.03, .05	102.9 *
<u>Cardiovascular Outcome</u>			
Systolic blood pressure (<i>k</i> =27)	.013	-.03, .06	129.9 *
Diastolic blood pressure (<i>k</i> =23)	.002	-.04, .04	78.7 *
HR (<i>k</i> =21)	.032	-.03, .09	63.4 *
<i>Q_M</i> (2,68)			.70
<u>Race</u>			
Black samples only (<i>k</i> =15)	-.09	-.20, .02	35.1 *
White samples only (<i>k</i> =27)	.022	-.02, .06	168.3 *
<i>Q_M</i> (1,40)			3.25
<u>Age of Sample</u>			
Adolescent samples (<i>k</i> =30)	-.026	-.09, .04	121.5 *
Adult samples (<i>k</i> =45)	.017	-.01, .05	191.3 *
<i>Q_M</i> (1,73)			1.44
<u>Comparing SES Measures</u>			
Hollingshead/edu/occ (<i>k</i> =44)	-.003	-.04, .03	224.9 *
All Others (<i>k</i> =31)	.030	-.03, .09	87.0 *
<i>Q_M</i> (1,73)			.99
Neighborhood indices (<i>k</i> =13)	-.010	-.12, .10	34.8 *
Individual indices (<i>k</i> =62)	.010	-.02, .04	278.2 *
<i>Q_M</i> (1,73)			.11
<u>Study Design</u>			
Not prospective or longitudinal (<i>k</i> =62)	.011	-.02, .04	180.9 *
Prospective or longitudinal (<i>k</i> =13)	-.001	-.06, .06	119.3 *
<i>Q_M</i> (1,73)			.14
<u>Nature of Stress Task</u>			
Cognitive (<i>k</i> =48)	.036 *	.01, .07	209.6 **
Interpersonal (<i>k</i> =10)	-.02	-.08, .05	25.8 *
Combination (<i>k</i> =17)	-.12 *	-.22, -.01	73.3 *
<i>Q_M</i> (2,72)			8.6 *
<u>SES Measurement</u>			
Continuous (<i>k</i> =50)	0	-.04, .04	270.2 *
Extreme Groups Comparison (<i>k</i> =25)	.03	-.01, .06	47.5 *

	Cumulative Effect Size (<i>r</i>)	95% CI	<i>Q_T</i> (heterogeneity)
<i>Q_M</i> (1,73)			.90
<u>SES and Reactivity is Stated Study Aim</u>			
Stated aim (k=45)	.005	-.04, .05	161.3 *
Not stated aim (k=30)	.009	-.03, .04	145.6 *
<i>Q_M</i> (1,73)			.02
<u>Method of Calculating Reactivity</u>			
Change score (k=60)	.023	-.01 – .05	251.6 *
Residual (k=14)	-.07 *	-.14 – .00	36.3 *
<i>Q_M</i> (1,72)			5.8 *

K denotes studies and k denotes effects. *Q_M*= test of between group difference. Bolded values highlight significant moderator variables.

* *p* .05.

Edu = education; occ = occupation. “Positive” effect sizes indicate a positive relationship between higher SES and greater reactivity (e.g., those with higher SES had larger HR or BP responses to the task).

Table 4

Cumulative effect sizes of the association between SES and cardiovascular recovery.

	Cumulative Effect Size (<i>r</i>)	95% CI	<i>Q_T</i> (heterogeneity)
All effects (k = 17)	-.11	-.18, -.03	52.2 *
Using study as the unit of analysis (K=6)	-.14	-.23, -.05	12.1 *
<u>Cardiovascular Outcome</u>			
Systolic blood pressure (k=6)	-.14 *	-.22, -.06	7.2
Diastolic blood pressure (k=6)	-.09	-.24, .06	24.6 *
HR (k=5)	-.08	-.23, .07	19.1 *
<i>Q_M</i> (2,14)			.57
<u>Race</u>			
Black samples only (k=3)	.13	-.05, .30	4.7
White samples only (k=8)	-.21 *	-.27, -.15	9.3
<i>Q_M</i> (1,12)			12.2 *
<u>Age of Sample</u>			
Adolescent samples (k=9)	-.07	-.18, .04	24.1 *
Adult samples (k=8)	-.14 *	-.23, -.04	25.9 *
<i>Q_M</i> (1,15)			.82
<u>Study Design</u>			
Not prospective or longitudinal (k=6)	-.17 *	-.27, -.06	17.3 *
Prospective or longitudinal (k=11)	-.07	-.15, .02	26.1 *
<i>Q_M</i> (1,15)			2.1
<u>SES Measurement</u>			
Continuous (k=8)	-.10 *	-.16, -.03	9.9
Extreme Groups Comparison (k=9)	-.10	-.22, .03	40.7 *
<i>Q_M</i> (1,15)			0

K denotes studies and k denotes effects. *Q_M*= test of between group difference. There were no significant moderator variables.

*
p .05.

“Positive” effect sizes indicate a positive relationship between higher SES and worse recovery (e.g., those with higher SES had higher HR or BP in the recovery post-task).