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Protective Factors for Adults from Low Childhood Socioeconomic Circumstances: The Benefits of Shift-and-Persist for Allostatic Load

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Abstract

Objective—Low socioeconomic status (SES) early in life is one of the most well-established social predictors of poor health. However, little is understood about why some adults who grew up in low SES environments do not have poor health outcomes. This study examined whether the psychological characteristic of “shift-and-persist” protects adults from the physiological risks of growing up in low SES households. Shift-and-persist consists of reframing appraisals of current stressors more positively (shifting), while simultaneously persisting with a focus on the future. We hypothesized that this characteristic would be associated with reduced physiological risk in low childhood SES individuals.

Methods—A national sample of 1,207 adults (ages 25–74) from the Survey of Midlife Development in the U.S. (MIDUS) completed psychological questionnaires, and were queried about parent education. Biological assessments consisted of 24 different measures across seven physiological systems, from which a composite measure representing cumulative physiological risk (allostatic load) was derived.

Results—Among adults who grew up in low SES households, those who engaged in high shift and high persist strategies had the lowest allostatic load ($b = -.15$, $p = .04$). No benefit of shift-and-persist was found for those from higher childhood SES backgrounds ($p = .36$).

Conclusions—Identifying the health-related protective qualities that naturally occur in some low SES individuals represents one important approach for developing future health improvement interventions for those who start out life low in SES. Moreover, the psychological qualities that are protective from future disease risk for those from low SES backgrounds are different from those beneficial to high SES individuals.

Keywords

childhood socioeconomic status; allostatic load; protective factors; psychological; physiological

INTRODUCTION

Socioeconomic status (SES) is one of the most powerful social determinants of physical health in developed countries. For example, individuals living in low SES circumstances are

consistently at greater risk for a variety of chronic medical illnesses, including cardiovascular disease (CVD), arthritis, and some cancers (1-3). The effects of low SES are evident across numerous countries throughout the world, including both those with and without universal health care (4).

Furthermore, research has identified the early years of life as a sensitive period, during which low SES seems to have especially potent and lasting effects on health. Indeed, low childhood SES increases people's risks of developing infectious, respiratory, and cardiovascular diseases much later in life (5-8). Over the years, researchers have focused their efforts on investigating the pathways that can explain these effects of childhood SES (9-13).

However, one important question that has gone largely unexplored is why some adults are able to maintain physically healthy profiles despite having grown up under difficult life circumstances. This is the notion of resilience - that is, thriving under adversity (14, 15). Resilience has been extensively discussed within the mental health literature (16-18); however, the notion of resilience has not often been tested with respect to physical health-related outcomes.

Our group has been working to develop a theoretical account of the specific psychological characteristics that may protect low SES individuals from detrimental physical health consequences (19). Under traditional models, low SES typically evokes stress responses that over time can promote long-term pathogenic processes that result in chronic illnesses such as cardiovascular disease years later (1, 20-24).

In contrast, our model states that there is an approach to life that is adaptive specifically in a low SES context. That is, a "shift-and-persist" approach to dealing with life demands will be beneficial physiologically to those who come from low SES backgrounds. This approach balances adapting the self to life stressors together with maintaining a focus on the future. It entails both shifting (adjusting oneself to stressors through cognitive reappraisals and emotion regulation) and persisting (enduring life with strength by holding onto hopes for the future). This combination of approaches to dealing with life stressors is hypothesized to reduce physiological responses to stress acutely, and by doing so, mitigates the long-term progression of pathogenic processes that lead to chronic disease (19).

The shift-and-persist theory is based in part on lifespan theories of control which postulate that human beings strive for primary control (being able to change the environment so that it fits one's needs and desires), but that when this type of control is not possible, they engage in secondary control efforts (attempts to bring oneself in line with one's environment) (25, 26). Given the multitude of constraints that low SES individuals face in life, they are hypothesized to value efforts to control the self and to adjust oneself to the world and others around them (conjoint agency) more so than efforts to control the world (27, 28). Consistent with this notion, in the health psychology literature, active efforts to cope with stressors and being given control over the parameters of a stressor are not beneficial to cardiovascular risk profiles amongst low SES individuals (29-33).

In addition, efforts to maintain a focus on the future are also thought to be beneficial for those low in SES. This idea is derived from theories of resilience that postulate that finding meaning in life helps individuals to cope with traumatic or life-threatening events (34-36). Low SES may represent another circumstance in which broader perspectives on life, such as meaning making and a future orientation is beneficial, not only to well-being, but also to physical health. Consistent with this notion, low SES adults who reported greater purpose in life showed lower levels of systemic inflammatory markers (37).

Finally, we argue that it is not just the presence of one of the above approaches, but the combination, that is critical – that is, possessing both an approach that values shifting the self in response to stress together with persisting with hopes for one’s future will be more beneficial than either trait on its own for physiological responses to stress specifically among those who come from low SES backgrounds. This is because shift-and-persist is postulated to represent a good fit with the general environmental constraints for those low in SES. In contrast, among high SES individuals, proactive efforts at coping that are aimed at eliminating stressors may be more effective, given the greater resources, on average, that high SES individuals possess for engaging in preventive behaviors, resolving situations, and influencing outcomes (38-41). Furthermore, shifting without persisting has the potential to lead to learned helplessness (passive acceptance of all stressors), which has been associated with increased risk of depression and functional disability in patient populations (42, 43). Hence the label that we use, “shift-and-persist,” is intended to connote the fact that it is the combination of these characteristics, rather than either one on its own, that is most beneficial to health among those low in SES.

The present paper sought to empirically test the shift-and-persist theory in a national sample of midlife adults in the U.S. To examine the notion that shift-and-persist strategies evolve out of adverse childhood circumstances and can have long-term health benefits, we focused on participants who grew up in low SES households during childhood, and examined whether shift-and-persist strategies were associated their physiological profiles as adults. Because SES disparities are observed for a wide range of health outcomes, we focus in this paper on multiple system indices of physiological risk, captured by allostatic load. The concept of allostatic load refers to the long-term physiological wear and tear that results from the body’s efforts to maintain homeostasis in response to stressors (44). Over time, this wear-and-tear manifests in the dysregulation of multiple physiological systems, including the cardiovascular, autonomic, metabolic, and inflammatory systems (45). Allostatic load composites reflecting these systems predict the onset of diseases such as CVD, as well as all-cause mortality (46-48). Capturing the biological effects of low SES across multiple regulatory systems is important, given that low SES has been linked to a wide array of health conditions that are likely affected by multiple physiological systems. In the present study, we hypothesized that the combination of shift-and-persist strategies would be associated with lower allostatic load among adults who grew up in low SES households, but not among adults who grew up in higher SES households.

METHODS

Participants

Participants were from the Midlife in the United States (MIDUS) survey, a national sample from the 48 contiguous states of individuals ages 25-74, selected via random-digit telephone dialing (Brim, Ryff, & Kessler, 2004). MIDUS I was conducted in 1995–1996 and MIDUS II in 2004–2006. In MIDUS I, 7,108 noninstitutionalized adults were selected, with 1,914 being twins. 75% of these individuals participated in MIDUS II. Biological data were collected on a subset of MIDUS II participants (N=1,255) who completed the telephone and mail surveys and were able and willing to travel to one of three General Clinical Research Centers (GCRC) for an overnight visit. 43% of those invited agreed to participate. Those who participated in the biological protocol were not different from the overall sample in terms of age, sex, race, marital status, or income (though they were more likely to have a college degree) (49). This study was approved by the Institutional Review Boards at UCLA, University of Wisconsin Madison, Georgetown University, and Brandeis University.

The current analyses focused on 1,207 MIDUS II participants who both provided biological data and information about their childhood SES. 300 of these participants were low SES, and 907 were higher SES (see below for SES definitions). See Table 1.

Protocol

Participants completed demographic and psychosocial questionnaires by mail and telephone interview. At the GCRC, individuals provided a complete medical history and medication information, underwent a physical exam with a physician, and provided blood, urine, and saliva samples, along with cardiovascular and heart rate variability measurements. Fasting morning blood was collected around 7:00am (prior to any caffeine or nicotine consumption). Urine was collected during a 12-hour overnight protocol beginning at 7:00pm and ending at 7:00am. Informed consent was obtained for all research participants.

Measures

SES—To index childhood SES, participants were queried about their parents' highest level of educational attainment. We used parent educational attainment because this is recommended for use in retrospective SES studies of adult samples and is more likely to be accurately remembered from childhood than other SES measures (such as family income) (50). Second, we sought to identify a group of clearly low SES adults, so we classified those who reported both parents as having less than a high school diploma as low SES ($n=300$). The remainder of the MIDUS II sample was categorized as higher SES ($n=907$). Current SES was used as a covariate in analyses, and was coded based on the participant's own level of educational attainment (less than high school diploma, high school grad, some college, or bachelor's degree or higher). This allowed us to test whether effects were due to childhood circumstances, or whether childhood SES merely served as a proxy for current SES.

Measures of shift—Two measures were combined to capture the tendency to shift oneself in response to stressors. One was the tendency to reappraise stressful situations more positively. This was measured at time 2 using the Positive Reappraisals scale of the Primary and Secondary Control questionnaire (51). Four items (e.g., "I can find something positive, even in the worst situations") were queried on a 4 point scale (ranging from "not at all" to "a lot"). Items were coded such that higher scores indicated higher tendency to positively reappraise stressful situations. Cronbach's alpha for the scale in MIDUS I was .78. Ten year test-retest reliability (from MIDUS I to MIDUS II) was .57, a value that is comparable to the stability of personality traits over this type of interval (52). This measure has been shown to have validity in previous studies in terms of correlating significantly with other related constructs, such as mastery (51), and in terms of predicting positive adjustment and positive affect among those who have more constrained life opportunities (e.g., older adults; (51, 53, 54)).

Second, we used a questionnaire measure of emotional reactivity to stress to get at emotion regulation (55). The stress reactivity subscale of the Multidimensional Personality Questionnaire (MPQ) taps the extent to which participants control their emotions in response to the stressors of daily life (e.g., "My mood often goes up and down," and "Minor setbacks sometimes irritate me too much"). Three items were rated on a 4 point scale (true, somewhat true, somewhat false, false) at time 2. Items were recoded such that higher scores indicated a greater ability to control one's emotions in the face of hassles. These items represent a shortened version of the MPQ-BF (55), which was necessary, given the constraints on questionnaire length in MIDUS. The 3 items were chosen in consultation with the author of the original scale (Tellegen) and using factor analysis and item response theory to analyze the original items from other datasets. Cronbach's alpha for this scale was .74 (compared to .84 in the original, longer version). Validity is indicated by virtue of the short

version correlating significantly with other measures of negative affect in MIDUS, such as neuroticism ($r=.69$, $p<.001$) and negative affect from the Positive and Negative Affect Scales ($r=.56$, $p<.001$). This is comparable to validity data on the original scale, in which the stress reactivity scale correlated .76 with neuroticism, and .33-.63 with negative emotion measures related to anxiety, anger, and distress (55, 56).

To create a total shift score, responses to the two questionnaires were first standardized (because they have different ranges), and then summed. Thus higher scores indicated greater use of shift strategies.

Measure of persist—A measure of future orientation was included as an indicator of persistence at time 2 (57). The “Live for Today” subscale of the Planning and Making Sense of the Past questionnaire in MIDUS consisted of four items, each rated on a 4 point scale (from “not at all” to “a lot”). These items tapped the extent to which individuals think about their future (e.g., “I have too many things to think about today to think about tomorrow”). Items were reverse coded such that higher scores indicated higher persistence with thinking about the future. Cronbach’s alpha for the scale in MIDUS I was .73. Ten year test-retest reliability (from MIDUS I to MIDUS II) was .55, comparable to the stability of personality traits over this type of interval (52). Validity is indicated by virtue of the Live for Today scale correlating significantly with other measures from MIDUS that one would expect to co-occur with a future orientation, such as greater mastery ($r=.27$, $p<.001$), optimism ($r=.21$, $p<.001$), greater life satisfaction ($r=.12$, $p<.001$), and greater conscientiousness ($r=.13$, $p<.001$), similar to the pattern of correlations documented for a related measure of future planning (57).

Allostatic load—Allostatic load was calculated as a risk score based on measures taken of seven different physiological systems at time 2. Measures of *cardiovascular* functioning included resting systolic and diastolic blood pressure (SBP, DBP; average of the two most similar of three successive measurements with a 30 sec rest period in between) and resting pulse (pulse count taken for 15 sec and multiplied by 4 to obtain resting beats per min). Indicators of *sympathetic nervous system* (SNS) activity included overnight urinary measures of epinephrine and norepinephrine (12-hour overnight urine collections via high-pressure liquid chromatography (HPLC), and adjusted by urine creatine levels to control for hormone output as a function of body size). Measures of *parasympathetic nervous system* (PNS) activity included the following heart rate variability parameters (assessed with electrocardiographic (ECG) electrodes placed on the left and right shoulders and in the left quadrant, and with ECG activity monitored during an 11 min seated baseline period): low and high frequency spectral power, the standard deviation of R-R (heartbeat to heartbeat) intervals (SDRR), and the root mean square of successive differences (RMSSD). Indicators of *hypothalamic pituitary adrenal (HPA) axis* activity included an overnight urinary measure of the hormone cortisol (measured via HPLC) and a serum measure of the hormone dehydroepiandrosterone sulfate (DHEA-S, assessed with a Roche Modular Analytics E170 analyzer with an Elecsys kit). Measures of *inflammation* included plasma C-reactive protein (CRP, assayed with a particle-enhanced immunonephelometric assay using a BNII nephelometer), fibrinogen (BNII nephelometer), and serum measures of interleukin-6 (IL-6, assayed with high-sensitivity enzyme-linked immunosorbent assay, ELISA) and the soluble adhesion molecules e-selectin and intracellular adhesion molecule-1 (ELISAs). Indicators of *lipid and general metabolic activity* included high density lipoprotein (HDL) cholesterol, low density lipoprotein (LDL) cholesterol, triglycerides, body mass index (BMI), and waist-hip ratio (WHR). Cholesterol panels were obtained using a Cobas Integra analyzer. LDL cholesterol levels were estimated using the Friedewald equation based on measures of total cholesterol, triglycerides, and HDL cholesterol. WHR is the ratio of waist (assessed at the narrowest point between ribs and the iliac crest) to hip circumference (measured at the

maximum diameter of the buttocks), and BMI was calculated based on height and weight. Measures of *glucose metabolism* included levels of glycosylated hemoglobin (measured using a Cobas Integra analyzer), fasting glucose (measured with an enzymatic assay on an automated analyzer, Roche Modular Analytics P), and the homeostasis model of insulin resistance (HOMA-IR, calculated based on glucose and insulin, which was measured with a two-site sandwich immunoassay using direct chemiluminescent technology).

To calculate allostatic load, a multi-system physiological risk score was computed as the sum of the seven separate physiological system (SNS, PNS, HPA, cardiovascular, glucose metabolism, lipid metabolism, and inflammation) risk indices. System risk indices were computed as the proportion of individual biomarker indicators for each system (ranging from 2 to 6 biomarkers) for which participant values fell into high-risk quartile ranges; scores were only computed for individuals with values on at least half of the biomarkers within a system. System risk scores could range from 0 to 1 (indicating 0 - 100% of system biomarkers in high-risk range for a given participant). As the number of biomarker indicators varied across physiological systems, this average risk scoring method produced a similar 'scaling' of risk scores across the different systems. A multi-system physiological risk index was computed as the sum of the seven physiological system scores (possible range from 0 to 7). Multi-system risk scores were only computed for participants with information on at least 6 of the 7 systems. Similar approaches to conceptualizing and calculating allostatic load have been used in numerous previous studies (45-47).

Covariate—Covariates included in statistical analyses included the demographic covariates of age, gender, and race (White or other). Medical covariates included a history of diseases that could affect the above systems, including diabetes (yes/no) and cardiovascular disease (yes/no). Health behavior covariates included current smoking status (yes/no). To address the possibility that childhood SES merely served as a proxy for current SES, we also included current education as a covariate.

Statistical analyses—Our first analysis involved a test of the 3-way interaction between childhood SES, shift strategies, and persist strategies in predicting allostatic load. This allowed us to test our overarching hypothesis that the combination (interaction) of shift and persist strategies would have different effects across SES groups. Hierarchical multiple regression analyses were conducted in which allostatic load was predicted from (1) covariates; (2) main effects of childhood SES (low, high), shift, and persist variables; and (3) the interaction of childhood SES, shift, and persist variables. Significant 3-way interactions were then followed-up by testing 2-way interactions (shift $\times$ persist) within each childhood SES group (low, high). The 2-way interactions allowed us to ask the question of how the combination of shift-and-persist strategies related to allostatic load within each SES group. Within SES groups, hierarchical multiple regression analyses were conducted in which allostatic load was predicted from (1) covariates; (2) main effects of shift and main effects of persist variables; and (3) the interaction of shift and persist variables. Including this interaction allowed us to test the hypothesis that it would be specifically the combination of high shift and high persist strategies (rather than either on its own) that would be protective for low childhood SES individuals. Tests of interactions were conducted according to the recommendations of Aiken and West (58), whereby variables are first centered, and then the interaction is calculated as the product of centered variables.

RESULTS

Table 1 presents descriptive information on the study sample. Adults from low childhood SES backgrounds were less likely to use persist strategies than adults from high childhood SES backgrounds ($t=5.13$, $p<.001$), although they were equally likely to use shift strategies

($t=0.11$, $p=.90$). Adults from low childhood SES backgrounds had higher allostatic load scores than adults from high childhood SES backgrounds ($t=5.26$, $p<.001$).

Do the Effects of Shift-and-Persist Differ by Childhood SES?

We first tested the 3-way interaction of childhood SES, shift, and persist variables predicting allostatic load. After controlling for demographic variables, medical variables, smoking, and current SES, the 3-way interaction of childhood SES, shift, and persist variables significantly predicted allostatic load ($\beta=-.07$, $p=.02$), indicating that the combination of shift and persist strategies had different associations with allostatic load, depending on childhood SES. To clarify the nature of this 3-way interaction, we next investigated how the 2-way interactions between shift and persist strategies predicted allostatic load for low versus higher childhood SES participants. This analytic approach allowed us to test whether the combination of shift and persist strategies was important specifically for buffering low SES adults from elevations in allostatic load.

Associations of Shift-and-Persist Strategies with Allostatic Load in Low Childhood SES Participants

Table 2 presents results for shift-and-persist analyses in low childhood SES participants. In Model 1, the demographic variables of age, gender, and ethnicity were controlled. Neither the main effects of shift strategies ($p=.73$) or persist ($p=.53$) strategies predicted allostatic load. However, the interaction between shift and persist significantly predicted allostatic load ($p=.02$), such that high shift in combination with high persist resulted in the lowest levels of allostatic load among adults who came from disadvantaged childhood SES environments.

In Model 2, medical history variables were added as covariates. In Model 3, current smoking was added as a covariate. And in model 4, current SES was included as a covariate. Patterns remained identical in all models, such that even in the fully adjusted model, after controlling for demographic variables, medical variables, smoking, and current SES, the interaction between shift and persist strategies remained significant ($p=.04$). As with Model 1, high shift in combination with high persist resulted in the lowest levels of allostatic load among adults who came from disadvantaged childhood SES environments.¹ See Figure 1.

Influence of Twin Status

To determine whether the presence of siblings in our sample affected our results, we redid analyses using Generalized Estimating Equations, specifying an exchangeable covariance matrix. This specification relaxes the assumption of independent and identically distributed observations. Patterns remained the same, in that over and above covariates, the interaction of shift-and-persist strategies ($p=.03$), but not the main effects of either shift ($p=.92$) or persist ($p=.72$) strategies predicted allostatic load.

Effects in Higher Childhood SES Participants

To test the specificity of these findings to low childhood SES participants, analyses were repeated for the 907 MIDUS participants who were higher in SES in childhood. The interaction between shift-and-persist strategies did not predict the allostatic load composite in this sample ($\beta=.01$, $p=.36$). See Figure 2. In addition, there was no main effect of shift strategies ($\beta=.02$, $p=.40$), and a marginal effect of persist strategies ($\beta=-.07$, $p=.06$),

¹These effects were specific to the allostatic load composite, as interactions were not significant for the individual measures that comprised allostatic load.

predicting allostatic load. This suggests that the combination of shift-and-persist strategies is beneficial only among those adults who came from low SES circumstances early in life.

DISCUSSION

These findings revealed that among adults who grew up in low SES households, engaging in shift-and-persist strategies was associated with reduced allostatic load. This shift-and-persist strategy entailed an approach to life that aims to positively reappraise stress and regulate negative emotions (shifting) in combination with maintaining a focus on the future (persisting). The association of shift-and-persist with lower allostatic load scores among adults who grew up in low SES households persisted after controls were implemented for a number of demographic, medical, and behavioral covariates. In addition, neither shifting nor persisting alone was associated with allostatic load in low SES individuals, suggesting that the combination of tendencies is what is important for reducing cumulative physiological risk in adults from low childhood SES backgrounds.

In this study, allostatic load was defined by a comprehensive battery of 24 measures taken from seven different physiological systems. The notion behind allostatic load is that risk may be better captured through a multisystem perspective that acknowledges the non-independence of physiological systems (45). The fact that shift-and-persist strategies were associated with this cumulative index of allostatic load suggests that shift-and-persist strategies may have implications over the long term for a number of chronic diseases of aging.

We note that shift-and-persist strategies were hypothesized to be beneficial specifically to those who live, on average, under circumstances of frequent stressors with few resources (i.e., low SES). In contrast, for those who live, on average, with fewer stressors and more abundant resources (high SES), an approach to life that in general emphasizes proactive efforts to change stressors themselves may be most effective for mitigating physiological responses to stress. In line with this theory, we found that shift-and-persist strategies were beneficial to allostatic load only in adults from low childhood SES backgrounds, but not in the much larger group of higher childhood SES adults.

Taken together, these findings suggest that the psychological qualities that are beneficial to low SES individuals' health may be different from those that are beneficial to high SES individuals. That is, for low SES individuals who, on average, face more uncontrollable stressors in daily life (22), an approach that in general emphasizes shifting oneself by accepting and adapting the self to stressors may be most effective for slowing down the pathogenic processes that contribute to chronic diseases. In combination, persistence in terms of maintaining a focus on one's future may provide meaning in life that promotes beneficial cardiovascular, neuroendocrine, and immune profiles. Over a lifetime, an accumulation of reduced psychosocial and physiological responses to acute stressors may slow the progression of longer-term pathogenic processes for chronic diseases of aging that low childhood SES normally sets into motion.

Our findings are consistent with the few studies that have examined positive psychological traits that buffer the effects of low current SES on current physiological or physical health outcomes. For example, adults who are low in SES but high in perceived control have profiles of self-reported health, acute health symptoms, and functional limitations that are similar to high SES individuals, and better than those of low SES individuals with low perceived control (59). Although the distinction between primary and secondary control coping was not made in this earlier study, the patterns are consistent with our notion that certain types of control strategies will be beneficial to low SES individuals. Similarly,

another study found that adults who are low in SES but have high purpose in life showed lower levels of the CVD risk marker IL-6, and their levels were similar to those of high SES individuals (37). These patterns are consistent with the notion that persisting in terms of finding meaning is beneficial to low SES individuals' health. Finally, these results are consistent with another recent paper published by our group in which we documented that among low SES children with asthma, high shift-and-persist scores predicted lower levels of asthma inflammation and reduced clinical impairment over time. In contrast, high SES children with asthma showed no benefit from shift-and-persist strategies (60). Our work extends these findings by focusing on the long-term health consequences of low early-life SES, and by identifying a unique constellation of traits that is protective among adults who grew up in low, but not high, SES families.

Our work is also consistent with previous literature that has focused on the broader social environment and that has identified protective effects of social support from others. This research has demonstrated that support and warmth from others is associated with reduced allostatic load in low SES children (61), and with beneficial cardiovascular and immune profiles among currently low SES adults (62). It is also consistent with research that has documented that positive relationships can buffer the effects of low childhood SES on adult physiological risk and gene expression profiles (63, 64). It may be the case that experiencing positive relationships with others over time helps to facilitate the development of a shift-in-persist approach within individuals. In the present study, rather than focusing on external social environments, we focused on low SES individuals themselves, and the characteristics that they can acquire to protect themselves from adverse health outcomes.

The fact that findings emerged with respect to the childhood economic circumstances from which study participants came is consistent with previous epidemiological literature documenting that childhood may represent a sensitive, or critical, period with long-lasting effects on health outcomes (6, 8, 65). One reason why this may be is that adversity that happens during certain sensitive periods of children's development may calibrate how certain bodily systems operate going forward, establishing more permanent proclivities, such as the tendency for immune cells to exhibit stable pro-inflammatory tendencies throughout life, via mechanisms such as epigenetics, post-translational modifications, and tissue remodeling (66).

Strengths of the present study include the large sample of participants from low SES childhoods, and the extensive battery of allostatic load measures. Limitations include the cross-sectional design that makes the directionality of findings difficult to discern. Ideally, we would have had a lifecourse design so that we could assess shift-and-persist strategies during childhood, and its ability to prospectively predict trajectories of physiological profiles over adulthood, and so that we could assess an array of childhood SES measures (in addition to parent education) and potential confounding variables, such as child health, early in life. In the absence of this type of longitudinal data, it is certainly possible that shift-and-persist strategies as measured in adulthood could reflect a consequence of certain childhood factors, such as being healthier or having experienced less stressful events earlier in life. However, we do note that the measures chosen for this study have long-term stability (high test-retest reliability over 10 years), suggesting that they represent characteristics that developed earlier in life. Second, we note that our findings are consistent with another study in which we also took approximations of the shift-and-persist construct from an existing dataset and demonstrated that shift-and-persist strategies protected low SES children from asthma inflammation and impairment (60). Taken together, these two studies suggest that there are benefits to shifting-and-persisting in childhood, and that their effects are also evident into adulthood, as suggested by the present study.

Second, this study was not designed explicitly to test the shift-and-persist hypothesis. As with many new theories, the first tests often come from existing datasets in which one utilizes reasonable approximations of a construct. Hence we relied on measures within the MIDUS study that tapped constructs related to shift-and-persist. We are currently in the process of developing and validating a measure of shift-and-persist, and plan to test ability of this measure to predict physiological and health-related outcomes in a low SES sample in a future study.

Finally, we did not have the ability to verify the controllability of stressful situations when probing shift strategies, although we know that low SES individuals, on average, report more perceived constraints in life (59).

In sum, adults who came from low SES backgrounds as children and who engaged in a combination of shift-and-persist strategies (dealing with stressors by positively reappraising them, engaging in emotion regulation, and at the same time, persisting with a future orientation) showed lower allostatic load scores. Understanding the specific psychosocial qualities that naturally contribute to physiological resilience among low SES individuals represents one important approach toward developing future interventions aimed at reducing health disparities. That is, if we can identify characteristics that some low SES individuals already possess that promote positive physiological profiles, these may be ones that would be effective to alter through intervention in other low SES individuals, allowing our society to help bolster individual coping resources among those who grow up with socioeconomic disadvantage. There are of course a variety of approaches that society could take toward reducing health disparities. The ones related to shift-and-persist suggested by this paper focus around efforts to help individuals deal with adversity themselves. Alternative approaches of great importance in the public health realm center around providing low SES individuals with basic resources to improve health. No one approach is likely to eliminate the health disparities that are so prominent in our society, and hence efforts to promote shift-and-persist would ideally be utilized in conjunction with societal efforts to provide basic economic resources and health services, which together could be marshaled to begin to make a difference in improving the health of those confronting disadvantaged circumstances early in life.

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Abbreviations

SES	socioeconomic status
CVD	cardiovascular disease
MIDUS	Midlife in the United States
SDRR	standard deviation of the RR interval
RMSSD	root mean squared successive difference
LFHRV	low frequency heart rate variability
HFLRV	high frequency heart rate variability
DHEA-S	dehydroepiandrosterone sulfate
CRP	C reactive protein
IL-6	interleukin 6
sICAM-1	soluble intercellular adhesion molecule 1
SBP	systolic blood pressure
DBP	diastolic blood pressure
HbA1c%	hemoglobin A1c %
LDL	low density lipoprotein
HDL	high density lipoprotein
BMI	body mass index
WHR	waist hip ratio
SNS	sympathetic nervous system
PNS	parasympathetic nervous system
HPA	hypothalamic pituitary adrenal axis
ECG	electrocardiogram
HPLC	high pressure liquid chromatography
DHEA-S	dehydroepiandrosterone sulfate
ELISA	enzyme-linked immunosorbent assay
HOMA-IR	homeostasis model of insulin resistance

Figure 1. Interaction of shift-and-persist strategies in predicting allostatic load among low childhood SES MIDUS participants. Estimated allostatic load scores are plotted at ± 1 SD of the shift and persist variables.

Figure 2.

No significant interaction of shift-and-persist strategies predicting allostatic load among high childhood SES MIDUS participants. Estimated allostatic load scores are plotted at ± 1 SD of the shift and persist variables.

Table 1

Sample characteristics of MIDUS II participants (N=1,207)

	Low Childhood SES (n=300)			High Childhood SES (n=907)		
	%	M	SD	%	M	SD
Age		58.50	12.12		53.19	11.32***
Gender (% male)	40.8			44.6		
Ethnicity (% white)	68.6			84.2***		
Current education						
< high school	9.7			3.5***		
High school	33.6			17.8		
Some college	28.9			30.4		
≥ university degree	27.9			48.3		
Current smoker	15.7			12.5		
Diabetes (ever diagnosed)	14.1			8.7**		
Cardiovascular disease (ever diagnosed)	22.1			12.7***		
Shift – positive reappraisals		3.16	0.65		3.10	0.62
Shift – emotion regulation		8.38	5.86		8.85	2.29
Persist – future orientation		2.60	0.78		2.85	0.71***
Sympathetic Nervous System						
Epinephrine (ug/g creatine)		2.04	1.40		1.95	1.24
Norepinephrine (ug/g creatine)		29.36	13.89		26.79	13.83**
Parasympathetic Nervous System						
SDRR (msec)		34.39	17.03		36.34	17.86
RMSSD		23.64	18.14		22.72	18.10
LFHRV		357.86	501.69		453.52	655.59*
HFHRV		304.63	635.65		323.49	777.59
Hypothalamic Pituitary Adrenal Axis						
Cortisol (ug/g creatine)		15.32	16.13		15.61	15.19
DHEA-S (ug/dL)		96.03	72.04		108.92	78.75*
Inflammation						

	Low Childhood SES (n=300)			High Childhood SES (n=907)		
	%	M	SD	%	M	SD
CRP (ug/mL)		3.57	5.59		2.84	4.52 *
IL-6 (pg/mL)		3.60	3.47		2.82	2.90 **
Fibrinogen (mg/dL)		359.86	91.05		344.46	86.86 **
sICAM-1 (ng/mL)		302.63	124.24		284.86	110.92 *
sE-selectin (ng/mL)		45.05	24.16		42.62	21.80
Cardiovascular						
SBP (mmHg)		133.98	17.64		130.30	18.10 **
DBP (mmHg)		75.50	10.98		75.54	10.58
Heart rate (bpm)		71.67	12.08		70.84	10.91
Glucose metabolism						
Glucose (mg/dL)		105.08	29.45		101.01	28.01 *
Insulin resistance (HOMA-IR)		3.84	3.67		3.40	3.64
HbA1c%		6.35	1.34		6.00	1.08 ***
Lipid metabolism						
LDL (mg/dL)		102.23	33.72		106.84	35.82
HDL (mg/dL)		54.13	16.98		55.46	18.25
Triglycerides (mg/dL)		130.30	79.76		133.96	147.11
BMI		30.99	7.85		29.28	6.11 **
WHR		0.90	0.10		0.89	0.10 *
Allostatic load score		2.02	1.00		1.66	1.02 ***

*
p<.05.**
p<.01.***
p<.001.

p<.001, for differences between low childhood and high childhood SES groups.

SDRR=standard deviation of the RR interval. RMSSD=root mean squared successive difference. LFHRV=low frequency heart rate variability. HFHRV=high frequency heart rate variability. DHEA-S=dehydroepiandrosterone sulfate. CRP=C reactive protein. IL-6=interleukin 6. sICAM-1=soluble intercellular adhesion molecule 1. SBP=systolic blood pressure. DBP=diastolic blood pressure. HbA1c %=hemoglobin A1c %. LDL=low density lipoprotein. HDL=high density lipoprotein. BMI=body mass index. WHR=waist hip ratio.

Table 2

Multiple regression analyses of shift-and-persist strategies predicting allostatic load in MIDUS adults from low childhood SES backgrounds (N=300)

Predictor	Controlling demographics	Controlling demographics + med history	Controlling demographics + med history + smoking	Controlling demographics + med history + smoking + current SES
Age	.23**	.20**	.24**	.23**
Gender	.05	.05	.07	.04
Ethnicity	-.05	-.02	.01	.00
CVD diagnosis		.05	.06	.04
Diabetes		.20**	.20**	.20**
Smoking			.17**	.14*
Current SES				-.10
Shift	-.02	.00	.00	.01
Persist	-.04	-.02	.00	.01
Shift × Persist	-.16*	-.18*	-.16*	-.15*

Note: Coefficients presented are beta weights.

*
p<.05.

**
p<.01.

SES=socioeconomic status. CVD=cardiovascular disease. Gender (1=female, 0=male). Ethnicity (1=white, 0=nonwhite). CVD diagnosis=ever diagnosed with CVD (yes=1, no=0). Diabetes=ever diagnosed with diabetes (yes=1, no=0). Smoking=current smoker (yes=1, no=0). Current SES=educational attainment of participant.