Shift-and-Persist Strategies: Associations With Socioeconomic Status and the Regulation of Inflammation Among Adolescents and Their Parents

Edith Chen, PhD, Kate C. McLean, PhD, and Gregory E. Miller, PhD

ABSTRACT

Objective: Shift-and-persist is a resilience construct hypothesized to be beneficial to physical health among individuals with low socioeconomic status (SES). This shift-and-persist construct entails a combination of reframing stressors more positively while also enduring adversity through finding purpose in life. In this study, we investigated how shift-and-persist relates to key inflammatory processes that are implicated in cardiovascular and other diseases. We also obtained validation information on a new shift-and-persist measure.

Method: A sample of 122 adolescents and 122 parents from a diverse range of SES backgrounds completed our shift-and-persist measure, a battery of other psychosocial questionnaires and interviews, and provided blood samples. Parents also provided SES information.

Results: Reliability and validity of the shift-and-persist measure were demonstrated across both adolescents and adults. Shift-and-persist moderated the association between SES and indicators of inflammatory regulation. Specifically, as SES declined, shift-and-persist was associated with greater sensitivity to glucocorticoids' anti-inflammatory properties (interaction in adolescents: $\beta = .21$, $p = .033$; interaction in adults: $\beta = .25$, $p = .011$), and also with less low-grade, chronic inflammation (interaction in adolescents: $\beta = .18$, $p = .044$). Conversely, as SES increased, the opposite pattern was evident.

Conclusions: These findings suggest that adaptive psychosocial characteristics have the potential to regulate inflammatory processes in ways that may mitigate risk for a number of chronic diseases, particularly among disadvantaged groups.

Key words: socioeconomic status, resilience, health.

INTRODUCTION

Low socioeconomic status (SES) has potent effects on risk for mental and physical health problems across the life span. Low-SES individuals consistently experience greater morbidity and mortality from a variety of mental illnesses, ranging from depression to schizophrenia (1,2), as well as from chronic medical illnesses, including cardiovascular disease, arthritis, and some cancers (3–5). Low SES experienced in childhood also increases an individual's risk of developing depression (6), infectious (7), and cardiovascular diseases later in life (8,9).

Nonetheless, some individuals are able to maintain good outcomes despite experiences with adversity, a phenomenon labeled resilience. One previously proposed psychosocial model of physical health–related resilience focuses on a constellation of characteristics that buffer low-SES individuals from poor health, called "shift-and-persist" (10,11). In this article, we investigate the underlying biological mechanisms associated with shift-and-persist, focusing on inflammation-related processes related to a number of chronic diseases of aging. These processes include both how innate immune cells respond to microbial challenges and how effectively these cells can slow down inflammatory responses upon receiving inhibitory signals. Secondarily, we also present information on the validity of a new scale to measure shift-and-persist.

CRP = C-reactive protein, **ERQ** = Emotion Regulation Questionnaire, **IL** = interleukin, **LPS** = lipopolysaccharide, **RSQ** = Responses to Stress Questionnaire, **SES** = socioeconomic status, **SST** = serum separator tube, **TNF** = tumor necrosis factor

SDC Supplemental Content

From the Department of Psychology & Institute for Policy Research (Chen, Miller), Northwestern University, Evanston, Illinois; and Department of Psychology (McLean), Western Washington University, Bellingham, Washington.

Address correspondence and reprint requests to Edith Chen, PhD, Northwestern University, 2029 Sheridan Rd, Evanston, IL 60208. E-mail: edith.chen@northwestern.edu

Received for publication January 9, 2014; revision received January 4, 2015.

DOI: 10.1097/PSY.0000000000000157

Copyright © 2015 by the American Psychosomatic Society

Copyright © 2015 by the American Psychosomatic Society. Unauthorized reproduction of this article is prohibited.

Shift-and-Persist

Psychologically, the shift-and-persist model proposes that a lifetime of facing constraints with limited options leads some individuals living in a low-SES context to place value on the ability to accept and adjust oneself to stressors that occur in daily life (*shifting)*. Shifting is hypothesized to involve cognitive reappraisals (i.e., reframing the meaning of a stressor to be less threatening, seeing the good that can come from difficult life situations) as a form of emotion regulation. Shifting is a type of secondary control coping and is akin to positive reappraisals/reframing and cognitive restructuring from other coping measures (12–14). The term *shifting* is not intended to refer to a coping strategy unique from these other constructs; rather, it was intended as part of a single label (shift-and-persist) that conveys the idea that the combination is beneficial to health among low-SES individuals. Coping via efforts to accommodate the self to stressors may be particularly beneficial under conditions in which stressors are largely uncontrollable and when resources for dealing with stressors are limited.

At the same time, in this context, successful adaptation entails enduring adversity with strength by developing purpose in life and holding on to hope, despite adversity, that the future may be better (*persisting*). Persisting may be beneficial because it allows low-SES individuals to focus on a larger purpose to life when they are confronting current adversities, which may facilitate the maintenance of hope during times of difficulty (15).

Moreover, it has been argued that it is the combination of approaches (shifting + persisting) that over the long term can mitigate the inflammatory processes that contribute to various disorders in low-SES individuals (10). In contrast, among high-SES individuals who come from different life circumstances, shift-and-persist is not hypothesized to be beneficial, given other available strategies for resolving situations and influencing outcomes, such as active coping and problem solving (16).

In several previous studies, empirical evidence for shift-and-persist and its health-related benefits among those low in SES was demonstrated (17,18); however, all of these analyses were conducted in already completed studies in which shift-and-persist was approximated using available instruments. In the present study, we created a new measure specifically intended to probe shift-and-persist and provide information about the reliability and validity of this scale.

Biological Mechanisms: Role of Inflammation

The primary goal of this study was to explore biological mechanisms that might contribute to shift-and-persist's association with physical health. In previous studies, the benefits of shift-and-persist in low-SES individuals were documented for clinical outcomes (obesity, asthma) (17,18). However, more in-depth inflammatory mechanisms that might underlie shift-and-persist were not investigated.

Under exposure to chronic adversities, such as low SES, the immune cells that orchestrate inflammation (monocytes and macrophages) respond more aggressively to microbial threats and are less sensitive to inhibition by cortisol, which is normally a potent anti-inflammatory signal (19,20). Over time, these tendencies give rise to low-grade inflammation among low-SES individuals (21–24). Low-grade inflammation has been implicated in the pathogenesis and expression of a number of chronic diseases associated with aging (25–28).

Although there have been previous studies testing associations of SES with inflammatory processes, this study provides the first test of whether a psychological buffering factor can potentially affect SES disparities in how inflammatory responses occur and are regulated. In this study, we developed a new brief measure of shift-and-persist. We first provide information about the reliability and validity of this measure. Our primary objective was to investigate inflammatory mechanisms underlying resilience using this measure, hypothesizing that as SES declined, individuals who engage in higher levels of shift-and-persist would show reduced inflammatory responses to microbial challenge and increased sensitivity to cortisol signaling.

METHODS

Participants

A total of 122 adolescents and 122 parents were recruited from Vancouver, BC, Canada, from newspaper and transit advertisements. Eligibility criteria included the following: a) adolescents ranging in age from 14 to 18 years (all attending secondary school) plus one parent, b) no chronic illnesses for either parent or adolescents, c) free of acute respiratory illness at the time of visit (by parent and adolescent report), and d) fluent in English. Average (standard deviation [SD]) age for adolescents was 16 (1.18) years, and average (SD) age for parents was 46.65 (6.85) years. Fifty-one percent of adolescents were girls, and 80% of parents were mothers. Among adolescents, 43% were white, 40% Asian, 5% African, 5% Hispanic, and 7% First Nations. Among parents, 55% were white, 38% Asian, 2% African, 3% Hispanic, and 2% First Nations. Our sample was diverse in SES, reflecting a distribution typical of Canada. Our sample consisted of an average of a four-person family. In Canada, median income for family households in 2011 was $79,600 (www.statscan.gov) (For comparison, median family income for a family of four in the United States in 2005 was $67,000, www.census.gov). In our sample, family income ranged from less than $5000 to more than $200,000, with a median family income of $65,000. Twenty-one percent of our sample fell below the federally established Canadian low-income cutoff for a family of 4, and 26% of our sample earned family incomes more than $100,000. All values are in Canadian dollars. See Table 1 for descriptive information about the sample.

Procedures

Interested families contacted the laboratory and were screened for eligibility. If eligible, one adolescent and one caregiver were scheduled for each family, and consent was obtained for parents

Copyright © 2015 by the American Psychosomatic Society. Unauthorized reproduction of this article is prohibited.

TABLE 1. Descriptive Information of Sample

Characteristics	Parents (n = 122)	Adolescents (n = 122)
Age, y	46.65 (6.85)	16.00 (1.18)
Sex (female)	97 (79.5)	62 (50.8)
Ethnicity		
White	67 (54.9)	53 (43.4)
Asian	42 (38.5)	49 (40.1)
Other	8 (6.5)	20 (16.4)
Family income[a]	5.53 (1.75)	5.53 (1.75)
Shift-and-persist[b]	26.79 (3.55)	24.07 (4.18)
Waist circumference, cm	87.53 (12.87)	75.41 (13.22)
CRP, mg/L	1.67 (2.82)	0.75 (1.30)
IL-6, pg/ml	1.52 (2.13)	0.85 (0.74)
LPS stimulation		
IL-1β, pg/ml	5560.08 (4259.78)	6154.17 (5659.72)
IL-6, pg/ml	33,688.12 (16,628.07)	39,584.58 (20,305.6)
IL-8, pg/ml	11,913.35 (9995.24)	12,675.65 (8651.53)
TNF-α, pg/ml	11,150.77 (5564.59)	12,805.14 (6699.24)
IL-10, pg/ml	138.51 (73.80)	159.62 (89.37)
LPS + hydrocortisone stimulation		
IL-1β, pg/ml	813.11 (1162.52)	855.28 (1058.38)
IL-6, pg/ml	9230.43 (6942.76)	10667.81 (7059.91)
IL-8 (pg/ml)	4268.83 (4988.25)	4085.99 (3634.36)
TNF-α, pg/ml	2395.64 (1901.46)	2713.72 (2097.03)
IL-10, pg/ml	147.27 (87.25)	170.70 (97.54)

CRP = C-reactive protein; IL = interleukin; LPS = lipopolysaccharide; TNF-α = tumor necrosis factor α.

Values represent either mean (with standard deviation in parentheses) or numbers (with percentages in parentheses).

[a] Family income ranges from 1 to 9. An average of 5.53 falls in the $50,000 to $74,999 category.

[b] Shift-and-persist scores range from 8 to 32. Values for inflammatory markers are untransformed.

and assent for adolescents. Parents and adolescents were placed in separate rooms where they completed questionnaires and were then interviewed together, as described below. A trained phlebotomist drew 50 cc of blood from an antecubital vein in both adolescents and parents. The protocol was approved by the University of British Columbia Research Ethics Board. Parents and children were paid $75 (Canadian) each for the laboratory visit. Data collection occurred between September 2011 and June 2012.

Measures

Socioeconomic Status

Socioeconomic status (SES) was measured by asking parents about the amount of annual income their family earned. This measure is one that is recommended by the MacArthur Research Network on Socioeconomic Status and Health to measure SES (www.macses.ucsf.edu) and is identical to that used in previous studies (29,30). Families were asked to report a dollar amount, which was then converted into a 9-point scale. In previous research, resource-based measures of SES have been documented to have more robust associations with health-related outcomes than prestige-based measures (e.g., education) (31,32).

Shift-and-Persist

A battery of items reflecting shift-and-persist were developed by the first author, based on theoretical notions of shift-and-persist and drawing from other existing measures that tap similar constructs. The original measure was designed with five shift items, five persist items, and six distractor items. The measure of shifting contained items answered on a 4-point scale, with higher numbers indicating greater shifting. Items were developed to represent secondary control strategies similar to other measures (14,33). The measure of persisting contained items also answered on a 4-point scale, with higher numbers indicating greater persisting. Items were developed to tap purpose in life as well as future orientation and future expectations, similar to other measures (34). This measure was administered to both parents and adolescents.

INFLAMMATORY RESPONSE AND REGULATION

Inflammatory Response to Microbial Stimulation

Peripheral blood was drawn from adolescents and parents into lithium-heparin Vacutainers (Becton-Dickinson, Oakville, Ontario, Canada), to assess two aspects of inflammation. First, whole blood

Copyright © 2015 by the American Psychosomatic Society. Unauthorized reproduction of this article is prohibited.

was cultured with a bacterial stimulus, lipopolysaccharide (LPS), to assess the ability of immune cells to respond to microbial challenge. Production of cytokines, including interleukin-1β (IL-1β), IL-6, IL-8, IL-10, and tumor necrosis factor α (TNF-α), was assessed. Whole blood was incubated with LPS (50 ng/ml; Sigma, St Louis, MO) for 6 hours at 37°C in 5% carbon dioxide. After centrifugation, the supernatants were collected and frozen at −30°C until analysis. All cytokines were measured in duplicate with Meso Scale Discovery Human ProInflammatory 7-Plex Base Kits (MSD, Rockville, MD) on an MSD SECTOR Imager 2400. Average intra-assay coefficients of variance (CVs) for the cytokines ranged from 3.48 to 8.68.

Glucocorticoid Sensitivity

The sensitivity of immune cells to inhibitory signals from cortisol was measured by quantifying IL-1β, IL-6, IL-8, IL-10, and TNF-α production in cells that had been coincubated with LPS and hydrocortisone. Whole blood was diluted in a 10:1 ratio with saline and dispensed into culture plates (Sigma Chemicals) with LPS (50 ng/ml) and hydrocortisone (2.76×10^{-5} M). After 6 hours of incubation at 37°C in 5% carbon dioxide, supernatants were centrifuged, harvested, and frozen at −30°C until analysis. All cytokines were measured in duplicate as per above with the MSD platform.

Low-Grade Inflammation

Peripheral blood was drawn into SST tubes (Becton-Dickinson, Franklin Lakes, NJ), and two measures of systemic inflammation, IL-6 and C-reactive protein (CRP), were assessed. Serum IL-6 levels were measured using a high-sensitivity enzyme-linked immunosorbent assay kit (R&D Systems, Minneapolis, MN; average intra-assay CV <8%; detection threshold = 0.04 pg/ml). CRP assays were conducted using a high-sensitivity, chemiluminescent technique (inter-assay CV = 2.2%; detection threshold = 0.20 mg/L).

Other Psychosocial Measures

Constructs related to shift-and-persist that were assessed as part of construct validation included coping, emotion regulation, optimism, future orientation, purpose in life, and beliefs about the world.

Coping was measured using the Responses to Stress Questionnaire (RSQ), which contains a secondary control coping scale developed for adolescents (14), and via the Primary and Secondary Control Questionnaire (administered to parents (33)). The tendency to use disengagement strategies was measured via the goal disengagement scale, which has been used in adolescents and adults (35,36). Emotion regulation was measured via the Emotion Regulation Questionnaire (ERQ), which contains subscales of reappraisal and suppression and has been used in adults and adolescents (37).

Optimism was measured via the Resilience Inventory, a measure developed for adolescents with an optimism subscale (38), and via the Life Orientation Test–Revised (administered to parents (39)).

Future orientation was measured via the Consideration of Future Consequences (parents only; this measure has not been validated in children (40)).

Purpose in life was measured via the Purpose in Life Test, a measure developed for adults (34), and the Purpose in Life Scale, a comparable measure developed for adolescents (41).

Beliefs about the world were measures using the Just World Scale (parents only; not validated in children (42)), and beliefs about trust in others from the General Social Survey (43).

Discussion Task

To explore real-world experiences with shift-and-persist, parents and children were also asked to discuss life experiences they had had that were challenging. Families were asked to discuss one life experience shared by both the parent and the child and a challenge the child experienced in his or her past. All conversations were video and audio recorded then transcribed verbatim. A coding scheme was developed by the second author for shift-and-persist. Participants were coded in terms of the extent to which they described adjusting the self to difficult life experiences (shifting). For each event, they were coded as no shifting (0), some shifting (1), or shifting as the dominant strategy (2). Participants were also coded in terms of whether they described a future orientation (persisting), meaning that they discussed how they would think or behave in the future in a positive tone (reflecting optimism). This response was coded as absent (0) or present (1) for each event. As a second indicator of persisting, the extent to which participants made meaning from the event was coded on a 0 (no meaning) to 3 (insight) scale, as done in previous studies (44). Responses across the two challenges were summed.

Interrater reliability was assessed between two coders on 24 conversations. Reliability was acceptable for shifting: for parents, intraclass $r = 0.98$, and for child, intraclass $r = 0.94$. For future orientation, both parent and child codes were reliable at $\kappa = 1.00$. Reliability was acceptable for meaning making for the parents (intraclass $r = 0.99$) and for the child (intraclass $r = 0.97$).

Covariates

Age, sex, ethnicity, and waist circumference of the respective participant (e.g., parent demographics for parent outcomes) were included as covariates in all analyses with immune outcomes.

Analytic Strategy

To assess reliability and validity of the scale, internal consistency (Cronbach α) was computed for each scale (shifting, persisting). A principal components factor analysis with varimax rotation was conducted to determine whether the items were best represented as two distinct factors. Convergent and divergent validity was assessed by testing associations with questionnaire and interview measures described above.

We also tested whether SES interacted with shift-and-persist to predict proinflammatory phenotypes. To do this, we conducted hierarchical multiple regression analyses according to the recommendations of Aiken and West (45), in which inflammation measures were predicted from a) covariates described above, b) main effect of SES (family income) and main effect of shift-and-persist, and c) the interaction between SES and shift-and-persist. Shift-and-persist scores were calculated by summing shift scores together with persist scores. Testing the SES × shift-and-persist interaction allowed us to test the hypothesis that shift-and-persist would be inversely associated with inflammatory outcomes more strongly among lower-SES individuals. Effect size estimates for these analyses are presented in terms of R^2 change for the addition of the interaction term.

Copyright © 2015 by the American Psychosomatic Society. Unauthorized reproduction of this article is prohibited.

All of the inflammatory variables were log transformed to correct for nonnormal distributions. Cytokine values in culture supernatants were significantly and positively intercorrelated. The average correlation was $r = 0.58$, with a range from 0.32 to 0.91, and all p values were less than .001. (Some readers may have expected IL-10 to correlate inversely with the other cytokines. Although IL-10 does have anti-inflammatory properties in vivo, it is expressed under conditions of immune activation (46). Thus, empirically it tends to correlate positively with proinflammatory cytokines such as IL-1β, IL-6, and TNF-α.) Because the cytokines values clustered, we decided to create a composite end point, which also had the advantage of reducing the total number of analyses needed. The composite was formed by standardizing and then averaging values of each of the five cytokines. (Separate composites were created for the LPS stimulation and glucocorticoid sensitivity assays.) We also created a composite indicator of low-grade inflammation by standardizing and averaging serum CRP and IL-6 values (r values = 0.48–0.58 in parents and adolescents, respectively; p values < .001).

RESULTS

Questionnaire Properties

Participant characteristics are shown in Table 1, and Table 2 provides the factor structure of the shift-and-persist scale used in the analyses below. Of the five shift items, one loaded onto the shift factor only among parents, but not adolescents, and hence was removed ("I tell myself that everything will be all right"). Of the five persist items, one about future orientation reduced internal consistency and did not load onto the persist factor, and hence was removed ("I have too many things to think about today to think about tomorrow"). Hence the final questionnaire consisted of four shift and four persist items plus distractors (Appendix A).

Factor analysis of the eight remaining shift-and-persist items revealed two distinct factors with eigenvalues greater than 1 in both parents and children. Factor loadings are presented in Table 2. In parents, the two factors accounted for 60.21% of the variance. The four theoretically derived shift items loaded onto one factor with all factor loadings greater than 0.7. The four theoretically derived persist items all loaded onto a second factor with factor loadings all greater than 0.5. In children, the two factors accounted for 60.67% of the variance. The same patterns of loadings emerged as with parents (all factor loadings >0.6; Table 2).

Internal consistency for each factor was adequate, particularly given the short length of each scale. In parents, α was .82 for the shift items and .64 for the persist items. For children, α was .80 for the shift items and.73 for the persist items.

Convergent Validity—Questionnaires

Convergent validity for the shift subscale was assessed by testing associations with other coping measures. We hypothesized that shift scores would be positively associated with secondary coping and reappraisal strategies. Parent scores on the shift subscale were significantly and positively associated with secondary control coping scores on the adult Primary and Secondary Control Questionnaire ($r = 0.44$, $p < .001$). Child scores on the shift subscale were significantly associated with secondary control coping scores on the child RSQ ($r = 0.52$, $p < .001$). Both parent and child shift scores were also significantly associated with emotional reappraisal scores from the ERQ (parent: $r = 0.30$, $p = .001$; child: $r = 0.25$, $p = .005$).

With respect to persist, we anticipated that persist scores would be positively associated with purpose in life and with scales related to expectations or thoughts about the future (optimism, future orientation). Parent scores on the persist subscale were significantly associated with purpose in life scores on the Purpose in Life Test ($r = 0.71$, $p < .001$), with optimism scores on the Life Orientation Test–Revised

TABLE 2. Factor Loadings for Shift-and-Persist Questionnaire

	Parents		Adolescents	
Item	Factor 1	Factor 2	Factor 1	Factor 2
1. I feel my life has sense of purpose	0.22	0.80	0.15	0.81
2. My life feels worthwhile	0.22	0.80	0.19	0.82
3. I believe there is a larger reason or purpose for my life	0.32	0.58	0.06	0.63
4. I feel my life is going nowhere (R)	−0.22	0.61	0.10	0.71
When something stressful happens in your life…				
5. I think about what I can learn from the situation	0.72	0.17	0.81	0.03
6. I think about the positive aspects, or the good that can come from the situation	0.85	0.03	0.69	0.20
When something doesn't turn out the way you want, and you are not able to change it…				
7. I think about what good things could come from the situation	0.72	0.13	0.79	0.19
8. I think about what I can learn from the situation	0.87	0.13	0.84	0.10

(R) = reverse coded.

Copyright © 2015 by the American Psychosomatic Society. Unauthorized reproduction of this article is prohibited.

($r = 0.38$, $p < .001$), and with a greater future orientation on the consideration of first consequences ($r = 0.26$, $p = .005$). Child scores on the persist subscale were significantly associated with purpose in life scores on the Purpose in Life Scale ($r = 0.81$, $p < .001$) and with optimism scores on the Resilience Inventory ($r = 0.62$, $p < .001$).

Convergent Validity—Interviews

In parents, higher shift scores on the shift-and-persist questionnaire were marginally associated with greater descriptions during interviews of adjusting the self in response to challenging situations ($r = 0.17$, $p = .065$). In addition, higher persist scores on the shift-and-persist questionnaire were significantly associated with greater future orientation expressed in interviews in response to challenging situations ($r = 0.18$, $p = .049$). Child interview responses were not associated with shift-and-persist scores (all p values > .4).

Divergent Validity

Divergent validity was tested in two ways. First, we tested associations of shift and persist measures with scales we hypothesized to be conceptually distinct. Second, we tested for a differential pattern of associations between shift and persist scales to test whether associations were specific to the construct of interest and not just due to monomethod questionnaire approaches.

We note that coping subscales are often significantly correlated with one another, despite having theoretical distinctions, making discriminant analyses sometimes tricky. For example, primary and secondary control strategies are significantly and positively correlated with each other (12,14,33), so primary control coping cannot easily be included in discriminant analyses. However, across previous studies, secondary control strategies (e.g., positive reinterpretations) have either been negatively associated or not been associated with disengagement coping and suppression-related approaches (12,14). In addition, positive reinterpretations are not associated with venting, or expression, of emotions (12). Hence, in the present study, we hypothesized that shifting would not show associations with emotional expression, emotional suppression, or disengagement. In parents, shift scores were not significantly associated with disengagement ($r = 0.00$, $p = .99$) or with emotional suppression from the ERQ ($r = 0.15$, $p = .11$). In children, shift scores were not significantly associated with disengagement ($r = 0.11$, $p = .24$), or with emotional suppression ($r = -0.01$, $p = .93$), or with the emotional expression subscale of the RSQ ($r = 0.12$, $p = .21$).

Persisting represents a holding on to purpose and hope that things can get better, regardless of how life circumstances currently seem to be. This suggests that persisting will be inversely related to disengagement from goals, and also that persisting will be unrelated to beliefs about the world (this is the idea of staying strong even when things that happen in life seem unfair). In children, persisting was inversely related to disengagement ($r = -0.27$, $p = .004$). Persisting was unrelated to beliefs of others as trustworthy ($r = -0.07$, $p = .43$). In parents, persisting was not associated with disengagement ($r = -0.03$, $p = .75$). Persisting also was unrelated to just world beliefs ($r = -0.01$, $p = .93$) and unrelated to beliefs about others as trustworthy ($r = -0.15$, $p = .11$).

Finally, as additional evidence of divergent validity, we examined associations of key variables with both shift and persist, expecting differential patterns with each. In adolescents, emotional reappraisal was associated with shifting ($r = 0.25$, $p = .005$) but not persisting ($r = 0.09$,

TABLE 3. Hierarchical Multiple Regressions of SES and Shift-and-Persist on Inflammation Regulation in Adolescents

Variables	*b*	SE	*p*
LPS-stimulated cytokine production			
Age	0.07	0.06	.29
Sex	0.01	0.06	.91
Ethnicity	−0.01	0.06	.83
Waist circumference	0.14	0.06	.027
SES	−0.02	0.04	.49
Shift-and-persist	0.01	0.02	.38
SES × shift-and-persist	0.02	0.01	.085
Glucocorticoid sensitivity			
Age	0.18	0.08	.030
Sex	0.00	0.08	.97
Ethnicity	−0.02	0.08	.80
Waist circumference	0.05	0.08	.55
SES	−0.04	0.05	.44
Shift-and-persist	−0.01	0.02	.79
SES × shift-and-persist	0.03	0.01	.033
Low-grade inflammation			
Age	0.09	0.08	.30
Sex	0.04	0.08	.64
Ethnicity	0.11	0.08	.17
Waist circumference	0.29	0.08	.001
SES	−0.12	0.05	.016
Shift-and-persist	0.00	0.02	.96
SES × shift-and-persist	0.03	0.01	.044

SES = socioeconomic status; *b* = unstandardized regression coefficient from each step; SE = standard error; LPS = lipopolysaccharide; IL = interleukin; TNF-α = tumor necrosis factor α; CRP = C-reactive protein.

Covariates were entered in step 1, main effects of SES and shift-and-persist were entered in step 2, and the SES × shift-and-persist interaction was entered in step 3. LPS-stimulated cytokine production reflects a composite of IL-1, IL-6, IL-8, IL-10, and TNF-α (standardized and averaged). Glucocorticoid sensitivity reflects the same composite after incubation with LPS + hydrocortisone. Systemic inflammation reflects a composite of CRP and IL-6 in serum (standardized and averaged).

Copyright © 2015 by the American Psychosomatic Society. Unauthorized reproduction of this article is prohibited.

p = .30). The cognitive restructuring subscale of the RSQ was also associated with shifting (r = 0.27, p = .003) but not persisting (r = −0.07, p = .46) in adolescents. In parents, emotional reappraisal was associated with shifting (r = 0.30, p = .001) but not persisting (r = 0.10, p = .27). In adolescents, purpose in life was associated with both shifting and persisting, but associations were stronger with persisting (r = 0.81, p < .001) than with shifting (r = 0.37, p < .001). A similar pattern was found in parents between purpose in life and persisting (r = 0.71, p < .001) versus shifting (r = 0.35, p < .001).

SES, Shift-and-Persist, and Inflammation

Tables 3 and 4 present results of the regression analyses for immune outcomes, and Tables S1 through S4 (Supplemental Digital Content 1, http://links.lww.com/PSYMED/A191) present additional details of supplementary analyses related to inflammation.

Inflammatory Response to Microbial Stimulation

In children, there was no significant main effect of SES or shift-and-persist on cytokine production after LPS stimulation. There was a marginal interaction of SES × shift-and-persist on cytokine production (p = .085) in a direction similar to the interaction effects reported below for glucocorticoid sensitivity. In parents, there were no significant effects of SES, shift-and-persist, or their interaction on stimulated cytokine production.

Glucocorticoid Sensitivity

In children, there was no significant main effect of SES or shift-and-persist on cytokine production after incubation with LPS + hydrocortisone. However, there was a significant SES × shift-and-persist interaction (β = .21, p = .033, R^2 change = 0.042). This interaction is graphed in Figure 1 and demonstrates that higher levels of shift-and-persist are associated with an increased sensitivity to glucocorticoid inhibition as SES declined (depicted in the graph by a greater reduction of inflammatory cytokine production in the presence of hydrocortisone). The opposite pattern emerges as SES increased, as higher levels of shift-and-persist were associated with decreased sensitivity to hydrocortisone's anti-inflammatory properties.

In parents, a parallel pattern emerged. That is, there was a significant SES × shift-and-persist interaction (β = .25, p = .011, R^2 change = 0.058). This interaction is also graphed in Figure 1 and demonstrates that higher levels of shift-and-persist are associated with increased sensitivity to glucocorticoid inhibition as SES declined. The opposite was true as SES increased among parents.

Effects on Individual Cytokines

When the composite measure (cytokines produced in response to LPS + hydrocortisone) was disaggregated into individual cytokines, all patterns remained in the same direction for the interactions in children (IL-1β: β = .19, p = .058; IL-6: β = .21, p = .032; IL-8: β = .16, p = .099; TNF-α: β = .13, p = .18; IL-10: β = .20, p = .056) as well as for the interactions in parents (IL-1β: β = .20, p = .038; IL-6: β = .18, p = .059; IL-8: β = .17, p = .085; TNF-α: β = .23, p = .018; IL-10: β = .27, p = .006).

TABLE 4. Hierarchical Multiple Regressions of SES and Shift-and-Persist on Inflammation Regulation in Parents

Variables	b	SE	p
LPS-stimulated cytokine production			
Age	−0.06	0.08	.45
Sex	−0.01	0.08	.94
Ethnicity	−0.02	0.09	.78
Waist circumference	0.05	0.08	.58
SES	0.03	0.04	.43
Shift-and-persist	0.03	0.02	.19
SES × shift-and-persist	0.02	0.01	.19
Glucocorticoid sensitivity			
Age	−0.12	0.08	.12
Sex	0.10	0.08	.23
Ethnicity	−0.02	0.09	.80
Waist circumference	0.06	0.08	.50
SES	0.02	0.04	.73
Shift-and-persist	0.00	0.02	.97
SES × shift-and-persist	0.03	0.01	.011
Low-grade inflammation			
Age	0.14	0.07	.042
Sex	0.32	0.08	<.001
Ethnicity	0.13	0.08	.084
Waist circumference	0.57	0.08	<.001
SES	0.01	0.04	.88
Shift-and-persist	0.02	0.02	.36
SES × shift-and-persist	0.00	0.01	.72

SES = socioeconomic status; b = unstandardized regression coefficient from each step; SE = standard error; LPS = lipopolysaccharide; IL = interleukin; TNF-α = tumor necrosis factor α; CRP = C-reactive protein.

Covariates were entered in step 1, main effects of SES and shift-and-persist were entered in step 2, and the SES × shift-and-persist interaction was entered in step 3. LPS-stimulated cytokine production reflects a composite of IL-1, IL-6, IL-8, IL-10, and TNF-α (standardized and averaged). Glucocorticoid sensitivity reflects the same composite after incubation with LPS + hydrocortisone. Systemic inflammation reflects a composite of CRP and IL-6 in serum (standardized and averaged).

Low-Grade Inflammation

In children, there was a significant main effect of SES on the CRP/IL-6 composite (β = −.22, p = .016, R^2 change = 0.046), indicating that lower SES was associated with higher levels of CRP/IL-6. There was also a significant interaction of SES × shift-and-persist (β = .18, p = .044, R^2

Copyright © 2015 by the American Psychosomatic Society. Unauthorized reproduction of this article is prohibited.

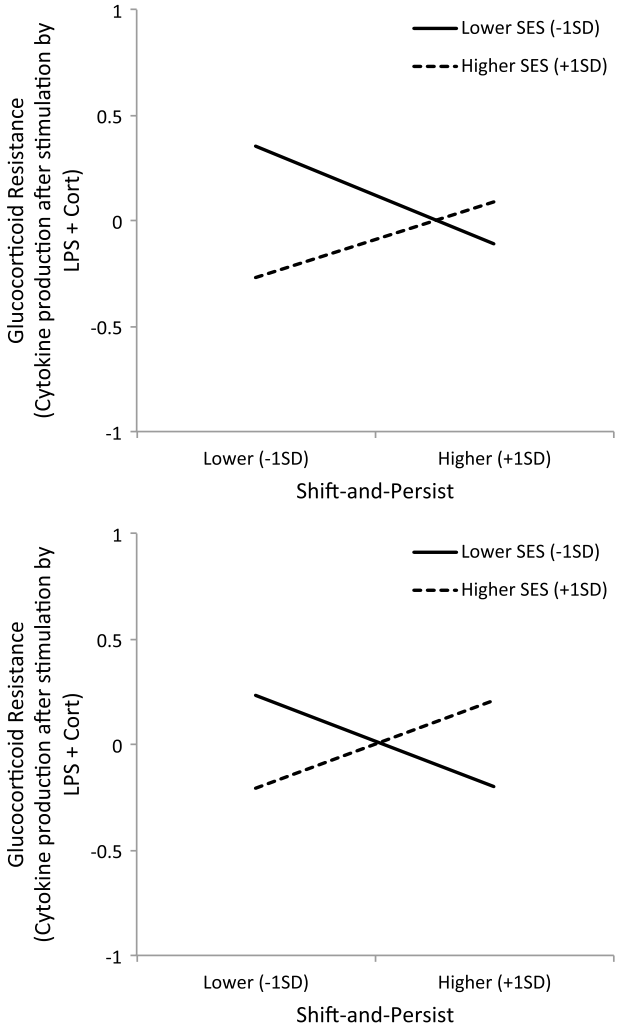

FIGURE 1. Interaction of SES with shift-and-persist predicting cytokine production after incubation with 50 ng/ml LPS and 2.76×10^{-5} M hydrocortisone. Cytokine production is a standardized composite of IL-1β, IL-6, IL-8, IL-10, and TNF-α. Graphs reflect estimated regression lines drawn at ±1 SD of each variable. Top panel: adolescents. Bottom panel: parents. SES = socioeconomic status; LPS = lipopolysaccharide; IL = interleukin; TNF-α = tumor necrosis factor α; SD = standard deviation; Cort = cortisol.

change = 0.031). This interaction is graphed in Figure 2 and illustrates that higher levels of shift-and-persist are associated with less low-grade inflammation as SES declined among adolescents. No associations with systemic inflammatory markers were found in parents.

DISCUSSION

This study investigated the inflammatory correlates of shift-and-persist. We found that SES interacted with shift-and-persist scores to predict the regulation of ex vivo monocyte inflammatory activity. As SES declined, higher shift-and-persist scores were associated with greater sensitivity to glucocorticoid's anti-inflammatory properties. In contrast, as SES increased, higher shift-and-persist scores were associated with decreased sensitivity to glucocorticoid inhibition.

These findings suggest that shift-and-persist enables lower-SES individuals to more effectively "slow down" monocyte inflammatory cytokine production once these cells have been activated by LPS, a common bacterial stimulus. This capacity may counteract the overabundance of inflammatory stimuli present in many low-SES environments. These stimuli can be social (stress, violence, conflict) and physical (cigarette smoke, air pollution, high-fat diets) and, through repeated activation of monocytes and macrophages, foster the kind of low-grade, chronic inflammation that contributes to mental illnesses such as depression and to chronic diseases of aging (25,47). Shift-and-persist may alter how lower-SES individuals respond to these stimuli, both psychologically and immunologically. By facilitating cortisol's regulation of these cells' activity, shift-and-persist may prevent inflammation from flourishing (19,48).

By psychologically reducing perceptions of stress in lower-SES individuals, shift-and-persist may promote glucocorticoid sensitivity through its effects on hypothalamic-pituitary-adrenal and/or autonomic nervous system responses to stress. Experiences of stress, when sustained over time, can elicit changes in hormone and cytokine profiles that prompt immune cells to down-regulate glucocorticoid receptor activity. This results in immune cells becoming more resistant to signaling from cortisol (glucocorticoid resistance). We hypothesize that when individuals have approaches to dealing with stress that are adaptive given the contexts they live in (e.g., higher shift-and-persist among lower SES; lower shift-and-persist among higher SES), this results in a reduced and more curtailed biological stress response that prevents the development of glucocorticoid resistance over time.

We found little evidence that shift-and-persist is associated with processes related to "turning on" cytokine production after microbial exposure. That is, shift-and-persist did not moderate the association of SES with cytokine production in response to LPS, suggesting that it does not affect how primed immune cells are to respond to this specific bacterial stimulus. Both mechanisms likely contribute to chronic, low-grade inflammatory burden in the body, but because they are regulated by different receptors and transcription factors, it is possible for psychosocial factors to affect one pathway but not the other. This idea is generally consistent with previous research that has demonstrated that social factors can regulate proinflammatory signaling patterns in monocytes and macrophages through different processes. For example, adverse social environments increase the expression of proinflammatory transcripts in adults in part through glucocorticoid resistance (20,49,50), whereas other factors such as maternal warmth achieve the same

Copyright © 2015 by the American Psychosomatic Society. Unauthorized reproduction of this article is prohibited.

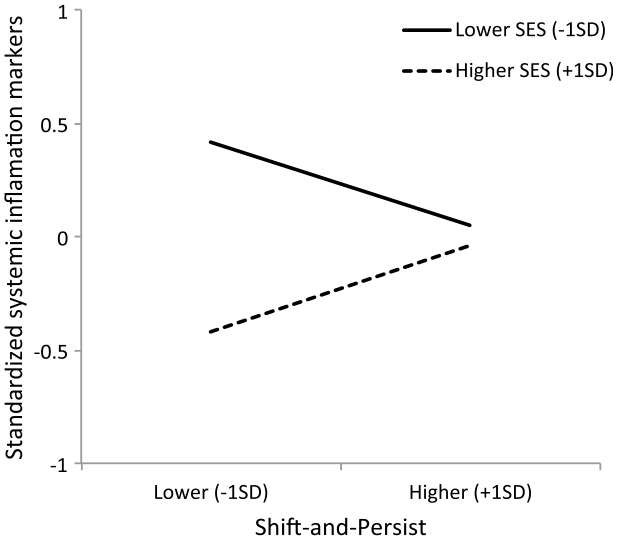

FIGURE 2. Interaction of SES with shift-and-persist predicting standardized composite of IL-6 and CRP in adolescents. Graphs reflect estimated regression lines drawn at ±1 SD of each variable. SES = socioeconomic status; IL = interleukin; CRP = C-reactive protein; SD = standard deviation.

effects through transcriptional pathways distinct from glucocorticoid resistance (e.g., cAMP response element-binding protein) (51).

Shift-and-persist also related to a downstream process, reflected in markers of chronic, low-grade inflammation in adolescents. That is, higher levels of shift-and-persist were associated with lower CRP/IL-6 as SES declined in adolescents. These findings are consistent with the possibility that shift-and-persist seems to mitigate the low-grade inflammation found in low-SES individuals in numerous previous studies (21–24,52). It is unclear why similar patterns with shift-and-persist and CRP/IL-6 did not emerge among parents, although this may have been related to the fact that there was no main effect of SES on CRP/IL-6 in parents in this sample. In addition, the clinical significance of these effects is difficult to discern because risk groups have traditionally been defined in midlife or older adults (not adolescents) and because this study did not consist of discrete groups that allow for direct comparisons.

We note that in this study, opposite patterns appeared at the other end of the SES spectrum, where as SES increased, shift-and-persist became associated with increased glucocorticoid resistance. Our theory proposes that shift-and-persist is a context-specific adaptive coping strategy, meaning that it is not beneficial to all individuals, but rather only beneficial in certain circumstances (e.g., low-SES environments where uncontrollable stressors frequently occur). In contrast, higher-SES individuals should have a wider repertoire of coping strategies available to them that can be effectively used (e.g., active coping, instrumental social support, and emotional expression), and under these circumstances, using a strategy best suited for largely uncontrollable life situations (shift-and-persist) may not be helpful and, in some cases, may even be detrimental for mitigating physiological stress responses. This may be because the use of strategies that are not a good fit for particular circumstances can thwart goal pursuits and increase frustration or negative affect. Examples of this are seen in studies showing that younger adults who engage in secondary control strategies (despite having more primary control strategies at their disposal) display more negative affect and poorer perceived health compared with older adults who engage in secondary control strategies (53,54). We also note that in a separate sample of asthma patients, shift-and-persist showed a similar pattern of appearing to be somewhat detrimental to higher-SES patients' inflammatory profiles (18).

In addition, we note that the new measure of shift-and-persist that we used in this study seems appropriate for both adolescents and adults, and demonstrated both reliability and validity. The development of such a questionnaire may be important for researchers interested in probing protective factors among disadvantaged groups, such as those who come from low-income families or underrepresented minority group members. That is, it is possible that there are constructs uniquely relevant to certain groups. For example, John Henryism refers to a coping style characterized by hard work and a single-minded determination to succeed even in the face of overwhelming odds, and has been associated with an increased risk of hypertension among low-SES, African American adults (55,56). The present study characterizes a similarly relevant, but positive, characteristic for low-SES individuals of shift-and-persist.

Although we tested the shift-and-persist scale in the context of low SES, this construct may also be relevant to other disadvantaged groups, or to other individuals experiencing ongoing adversities that are uncontrollable. For example, shift-and-persist may apply to disadvantaged minority groups (10). These possibilities should be tested in future studies with other diverse samples.

In our analyses, patterns were generally consistent across parents and adolescents. This suggests that our new measure can be used across both adolescents and adults, that shift-and-persist can benefit both younger individuals as well as adults who are low in SES, and that the inflammatory correlates of shift-and-persist are replicable across different age groups. One exception to the consistency in patterns was that discussion task ratings of shift-and-persist were correlated with shift-and-persist questionnaire scores in parents, but not adolescents. It may be that developmentally, adolescents are still in the process of constructing an understanding of the self, particularly when it comes to narrating life stories (57,58), and that the completely open-ended and unstructured nature of the discussion (parent and adolescent having a conversation with each other, but not guided by a research assistant) made that process

Copyright © 2015 by the American Psychosomatic Society. Unauthorized reproduction of this article is prohibited.

more difficult for adolescents. This may have resulted in adolescents having a less well-developed and less consistent description of coping with difficult life events and outlooks on life during open-ended conversations compared with their parents. We are currently testing the use of coding shift-and-persist from more structured interviews.

Limitations of the present study include the fact that it was cross sectional. Future research is needed that follows samples over time and determines when shift-and-persist develops, and how it changes across the life span. Administering the shift-and-persist questionnaire in larger samples to establish norms for this new measure would also be useful. In addition, creating paradigms for probing individual differences in shift-and-persist responses to an identical situation would be a useful complement to this questionnaire. Experimental manipulations of shift-and-persist would be an important next step for ascertaining causality and for determining the malleability of shift-and-persist and the impact of altering shift-and-persist for inflammatory processes as well as for longer-term health outcomes. We also acknowledge that there are multiple methods for calculating scale reliability, and although we used the most common approach (Cronbach α), there are others that may be useful as well (59). Finally, given that our multiple cytokine measures were highly associated with one another, we combined cytokines into one composite variable to reduce the number of analyses; however, this composite measure is limited in obscuring associations between individual cytokines and SES or shift-and-persist.

In sum, shift-and-persist moderated the relationship between SES and both glucocorticoid sensitivity and systemic inflammation, such that as SES declined, higher levels of shift-and-persist were associated with increased sensitivity to anti-inflammatory cortisol signaling and with lower levels of a CRP/IL-6 composite. These findings suggest that adaptive psychosocial characteristics may have the potential to regulate inflammatory processes in beneficial ways, particularly in more disadvantaged groups, and hence that targeting these types of strategies may be one useful approach to develop in future studies to begin to ameliorate the striking disparities in health outcomes that exist in our society.

Source of Funding and Conflicts of Interest: This project was supported by Canadian Institutes of Health Research Grant FRN: 97872 to E.C. All authors declare no conflicts of interest.

REFERENCES

1. Dohrenwend BP, Levav I, Shrout PE, Schwartz S, Naveh G, Link BG, Skodol AE, Stueve A. Socioeconomic status and psychiatric disorders: the causation-selection issue. Science 1992;255:946–52.
2. Johnson JG, Cohen P, Dohrenwend BP, Link BG, Brook JS. A longitudinal investigation of social causation and social selection processes involved in the association status and psychiatric disorders. J Abnorm Psychol 1999;108: 490–9.
3. Adler NE, Boyce T, Chesney MA, Cohen S, Folkman S, Kahn RL, Syme SL. Socioeconomic status and health: the challenge of the gradient. Am Psychol 1994;49:15–24.
4. Marmot MG, Shipley MJ, Rose G. Inequalities in death-specific explanations of a general pattern? Lancet 1984;i:1003–6.
5. Adler NE, Rehkopf DH. U.S. disparities in health: descriptions, causes, and mechanisms. Annu Rev Public Health 2008; 29:235–52.
6. Gilman SE, Kawachi I, Fitzmaurice GM, Buka SL. Socioeconomic status in childhood and the lifetime risk of major depression. Int J Epidemiol 2002;31:359–67.
7. Cohen S, Janicki-Deverts D, Chen E, Matthews KA. Childhood socioeconomic status and adult health. Ann N Y Acad Sci 2010;1186:37–55.
8. Galobardes B, Lynch JW, Smith GD. Childhood socioeconomic circumstances and cause-specific mortality in adulthood: systematic review and interpretation. Epidemiol Rev 2004;26:7–21.
9. Galobardes B, Smith GD, Lynch JW. Systematic review of the influence of childhood socioeconomic circumstances on risk for cardiovascular disease in adulthood. Ann Epidemiol 2006; 16:91–104.
10. Chen E, Miller GE. "Shift-and-persist" strategies: why being low in socioeconomic status isn't always bad for health. Perspect Psychol Sci 2012;7:135–58.
11. Chen E. Protective factors for health among low socioeconomic status individuals. Curr Direct Psychol Sci 2012;21: 189–93.
12. Carver CS, Scheier MF, Weintraub JK. Assessing coping strategies: a theoretically based approach. J Pers Soc Psychol 1989;56:267–83.
13. Folkman S, Lazarus RS, Dunkel-Schetter C, DeLongis A, Gruen RJ. Dynamics of a stressful encounter: cognitive appraisal, coping, and encounter outcomes. J Pers Soc Psychol 1986;50:992–1003.
14. Connor-Smith JK, Compas BE, Wadsworth ME, Thomsen AH, Saltzman H. Responses to stress in adolescence: measurement of coping and involuntary stress responses. J Consult Clin Psychol 2000;68:976–92.
15. Updegraff JA, Silver RC, Holman EA. Searching for and finding meaning in collective trauma: results from a national longitudinal study of the 9/11 terrorist attacks. J Pers Soc Psychol 2008;95:709–22.
16. Aspinwall LG, Taylor SE. A stitch in time: self-regulation and proactive coping. Psychol Bull 1997;121:417–36.
17. Kallem S, Carroll-Scott A, Rosenthal L, Chen E, Peters SM, McCaslin C, Ickovics JR. Shift-and-persist: a protective factor for elevated BMI among low socioeconomic status children. Obesity 2013;21:1759–63.
18. Chen E, Strunk RC, Trethewey A, Schreier HM, Maharaj N, Miller GE. Resilience in low-socioeconomic-status children with asthma: adaptations to stress. J Allergy Clin Immunol 2011;128:970–6.
19. Miller GE, Chen E, Parker KJ. Psychological stress in childhood and susceptibility to the chronic diseases of aging: moving toward a model of behavioral and biological mechanisms. Psychol Bull 2011;137:959–97.
20. Miller GE, Chen E, Fok AK, Walker H, Lim A, Nicholls EF, Cole S, Kobor MS. Low early-life social class leaves a biological residue manifested by decreased glucocorticoid and increased proinflammatory signaling. Proc Natl Acad Sci U S A 2009;106:14716–21.

Copyright © 2015 by the American Psychosomatic Society. Unauthorized reproduction of this article is prohibited.

21. Alley DE, Seeman TE, Ki Kim J, Karlamangla A, Hu P, Crimmins EM. Socioeconomic status and C-reactive protein levels in the US population: NHANES IV. Brain Behav Immun 2006;20:498–504.
22. Dowd JB, Zajacova A, Aiello AE. Predictors of inflammation in U.S. children aged 3-16 years. Am J Prev Med 2010;39: 314–20.
23. Phillips JE, Marsland AL, Flory JD, Muldoon MF, Cohen S, Manuck SB. Parental education is related to C-reactive protein among female middle-aged community volunteers. Brain Behav Immun 2009;23:677–83.
24. Petersen KL, Marsland AL, Flory J, Votruba-Drzal E, Muldoon MF, Manuck SB. Community socioeconomic status is associated with circulating interleukin-6 and C-reactive protein. Psychosom Med 2008;70:646–52.
25. Nathan C, Ding A. Nonresolving inflammation. Cell 2010; 140:871–82.
26. Chung HY, Cesari M, Anton S, Marzetti E, Giovannini S, Seo AY, Carter C, Yu BP, Leeuwenburgh C. Molecular inflammation: underpinnings of aging and age-related diseases. Ageing Res Rev 2009;8:18–30.
27. Libby P, Ridker PM, Maseri A. Inflammation and atherosclerosis. Circulation 2002;105:1135–43.
28. Ridker PM, Hennekens CH, Buring JE, Rifai N. C-reactive protein and other markers of inflammation in the prediction of cardiovascular disease in women. N Engl J Med 2000; 342: 836–43.
29. Chen E, Strunk RC, Bacharier LB, Chan M, Miller GE. Socioeconomic status associated with exhaled nitric oxide responses to acute stress in children with asthma. Brain Behav Immun 2010;24:444–50.
30. Chen E, Miller GE, Walker HA, Arevalo JM, Sung CY, Cole SW. Genome-wide transcriptional profiling linked to social class in asthma. Thorax 2009;64:38–43.
31. Chen E, Cohen S, Miller GE. How low socioeconomic status affects 2-year hormonal trajectories in children. Psychol Sci 2010;21:31–7.
32. Chen E, Lee WK, Cavey L, Ho A. Role models and the psychological characteristics that buffer low–socioeconomic-status youth from cardiovascular risk. Child Dev 2013;84:1241–52.
33. Wrosch C, Heckhausen J, Lachman ME. Primary and secondary control strategies for managing health and financial stress across adulthood. Psychol Aging 2000;15:387–99.
34. Crumbaugh JC, Maholick LT. An experimental study in existentialism: the psychometric approach to Frankl's concept of noogenic neurosis. J Clin Psychol 1964;20:200–7.
35. Wrosch C, Scheier MF, Miller GE, Schulz R, Carver CS. Adaptive self-regulation of unattainable goals: goal disengagement, goal reengagement, and subjective well-being. Pers Soc Psychol Bull 2003;29:1494–508.
36. Miller GE, Wrosch C. You've gotta know when to fold 'em: goal disengagement and systemic inflammation in adolescence. Psychol Sci 2007;18:773–7.
37. Gross JJ, John OP. Individual differences in two emotion regulation processes: implications for affect, relationships, and well-being. J Pers Soc Psychol 2003;85:348–62.
38. Oberle E, Schonert-Reichl KA, Zumbo BD. Life satisfaction in early adolescence: personal, neighborhood, school, family, and peer influences. J Youth Adolesc 2011;40:889–901.
39. Scheier MF, Carver CS, Bridges MW. Distinguishing optimism from neuroticism (and trait anxiety, self-mastery, and self-esteem): a reevaluation of the Life Orientation Test. J Pers Soc Psychol 1994;67:1063–78.
40. Strathman A, Gleicher F, Boninger DS, Edwards CS. The consideration of future consequences: weighing immediate and distant outcomes of behavior. J Pers Soc Psychol 1994; 66:742–52.
41. Robbins M, Francis LJ. Religion, personality and well-being: the relationship between church attendance and purpose in life. J Res Christian Educ 2000;9:223–38.
42. Rubin Z, Peplau LA. Who believes in a just world? J Soc Issues 1975;31:65–89.
43. Kawachi I, Kennedy BP, Lochner K, Prothrow-Stith D. Social capital, income inequality, and mortality. Am J Public Health 1997;87:1491–8.
44. McLean KC, Pratt MW. Life's little (and big) lessons: identity statuses and meaning-making in the turning point narratives of emerging adults. Dev Psychol 2006;42:714–22.
45. Aiken LS, West SG. Multiple Regression: Testing and Interpreting Interactions, London, Sage Publications; 1991.
46. Saraiva M, O'Garra A. The regulation of IL-10 production by immune cells. Nat Rev Immunol 2010;10:170–81.
47. Raison CL, Miller AH, Halaris A, Leonard BE. Role of inflammation in depression: implications for phenomenology, pathophysiology, and treatment. Mod Trends Pharmacopsychiatry 2013;28:33–48.
48. Miller GE, Cohen S, Ritchey AK. Chronic psychological stress and the regulation of pro-inflammatory cytokines: a glucocorticoid resistance model. Health Psychol 2002;21: 531–41.
49. Cole SW, Hawkley LC, Arevalo JM, Sung CY, Rose RM, Cacioppo JT. Social regulation of gene expression in human leukocytes. Genome Biol 2007;8:R189.
50. Miller GE, Chen E, Sze J, Marin T, Arevalo JM, Doll R, Ma R, Cole SW. A functional genomic fingerprint of chronic stress in humans: blunted glucocorticoid and increased NF-kappaB signaling. Biol Psychiatry 2008;64:266–72.
51. Chen E, Miller GE, Kobor MS, Cole SW. Maternal warmth buffers the effects of low early-life socioeconomic status on pro-inflammatory signaling in adulthood. Molec Psychiatry 2011;16:729–37.
52. Dowd JB, Aiello AE. Socioeconomic differentials in immune response. Epidemiology 2009;20:902–8.
53. Chipperfield JG, Perry RP, Menec VH. Primary and secondary control-enhancing strategies: implications for health in later life. J Aging Health 1999;11:517–39.
54. Wrosch C, Heckhausen J. Control processes before and after passing a developmental deadline: activation and deactivation of intimate relationship goals. J Pers Soc Psychol 1999;77: 415–27.
55. James SA, Keenan NL, Strogatz DS, Browning SR, Garrett JM. Socioeconomic status, John Henryism, and blood pressure in black adults. The Pitt County Study. Am J Epidemiol 1992;135:59–67.
56. James SA, Strogatz DS, Wing SB, Ramsey DL. Socioeconomic status, John Henryism, and hypertension in blacks and whites. Am J Epidemiol 1987;126:664–73.
57. Chen Y, McAnally HM, Wang Q, Reese E. The coherence of critical event narratives and adolescents' psychological functioning. Memory 2012;20:667–81.
58. McLean KC, Breen AV. Processes and content of narrative identity development in adolescence: gender and well-being. Dev Psychol 2009;45:702–10.
59. Yang Y, Green SB. Coefficient alpha: a reliability coefficient for the 21st century? J Psychoeduc Assess 2011;29: 377–92.

Copyright © 2015 by the American Psychosomatic Society. Unauthorized reproduction of this article is prohibited.

Appendix A

Shift-and-Persist Questionnaire.

1 = not at all
2 = a little
3 = some
4 = a lot

Please rate how well the following statements describe you:
1. I feel my life has a sense of purpose
2. My life feels worthwhile
3. I believe that there is a larger reason or purpose for my life
4. I feel my life is going nowhere

Next you will see a list of things that people sometimes do, think, or feel when something stressful happens. Everybody deals with problems in their own way. Please rate how much you do each of the following things when something stressful happens in your life.

When something stressful happens in my life…
5. I think about what I can learn from the situation
6. I work to change or fix the problem
7. I try not to think about it, to forget about it
8. I think about the positive aspects, or the good that can come from the situation
9. I start to act without thinking

In life, things don't always go the way that we want. Everyone has different preferences for how they deal with situations in which something doesn't turn out the way that they want, and they are not able to change it. Please rate how much you do each of the following.

When something doesn't turn out the way that I want…
10. Little things upset me easily
11. I think about what good things could come from the situation
12. I find it hard to stop thinking about what happened
13. I start working on other new goals
14. I think about what I can learn from the situation

Items 6, 7, 9, 10, 12, and 13 are distractors. Items 5, 8, 11, and 14 are summed for a shift score. Scores 1 to 4 are summed for a persist score, with 4 reverse scored.

Copyright © 2015 by the American Psychosomatic Society. Unauthorized reproduction of this article is prohibited.