ORIGINAL ARTICLE

Links Between Socioeconomic Status, Daily Depressive Affect, Diurnal Cortisol Patterns, and All-Cause Mortality

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ABSTRACT

Objective: Socioeconomic status (SES) remains a robust risk factor for mortality. Various theoretical models postulate that lower SES is associated with higher negative affect, which then initiates a cascade of physiological disturbances that contribute to illness and early mortality. However, few studies have explicitly investigated the interplay between psychological and biological factors in determining SES disparities in mortality. This study examined the role of daily negative affect and cortisol secretion in explaining the SES-mortality link in a large sample of US adults.

Methods: Using data from the Midlife in the United States study (n = 1735, mean [standard deviation] age = 56.40 [12.10] years, 56.4% female), we tested longitudinal associations between SES, daily negative affect, daily cortisol levels, and all-cause mortality 13 years later. Daily negative affect was classified into three clusters reflecting depressive affect, anxiety, and anger.

Results: Higher SES was linked to a lower risk of all-cause mortality (hazard ratio = 0.94, 95% confidence interval = 0.90 to 0.97). Furthermore, there was a sequential link between higher SES and lower mortality through lower daily depressive affect and a steeper ("healthier") diurnal cortisol slope (indirect effect = −0.0007, 95% confidence interval = −0.0014 to −0.0002). Daily anxiety and anger were not associated with cortisol levels or mortality (p values > .05).

Conclusions: These findings suggest that daily negative emotional experiences and the hypothalamic-pituitary-adrenal axis functioning may constitute important psychological and physiological pathways underlying the link between SES and all-cause mortality.

Key words: socioeconomic status, cortisol, daily affect, mortality.

INTRODUCTION

More than four decades ago, a series of pioneering studies provided evidence about the social gradient in health (1,2). These influential data demonstrated that income, education, and wealth shaped health outcomes not only for the very poor but also in a linear fashion across the entire social ladder. Since then, the challenges revealed by these findings have grown. Socioeconomic status (SES)[1] remains a robust risk factor for mortality worldwide (4), and findings suggest that the social gradient has become steeper in recent years, reflecting an even wider gap in longevity across the SES spectrum (4,5). For example, statistics from the United States—one of the countries with the largest SES disparities in health—showed that, over the past 15 years, life expectancy increased by 3 years for the richest 5% but remained stagnant for the poorest 5% of the population (5). A noteworthy fact about the social gradient is that structural and behavioral factors, such as exposure to pollution, limited access to health care, and life-style differences contribute significantly but do not fully explain this phenomenon (6). Other factors, including social and psychological processes, have also been suggested to play important roles in linking SES to mortality (7–9).

Several theoretical models on the role played by psychosocial factors in explaining SES disparities postulate that lower SES is associated with higher psychological stress and negative affect, which then initiate a cascade of physiological alterations that may ultimately contribute to mortality (7,10). Of the proposed biological mechanisms, the activity of the hypothalamic-pituitary-adrenal (HPA) axis has gained significant attention because of the central role of the HPA axis in coordinating biological responses to stress (11). Under stressful conditions, the HPA axis releases cortisol to mobilize energy and resources to facilitate responses to threats (12). In the short term, this response is beneficial to survival. However, sustained HPA axis activation due to repeated exposure to

CAR = cortisol awakening response, **CI** = confidence interval, **HPA** = hypothalamic-pituitary-adrenal, **HR** = hazard ratio, **MIDUS** = Midlife in the United States, **MLM** = multilevel modeling, **NSDE** = National Study of Daily Experiences, **SES** = socioeconomic status

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stressors leads to dysregulated patterns of HPA axis functioning. This is reflected by dysregulated cortisol reactivity to stressors and, at the daily level, an atypical diurnal cortisol rhythm (13).

Cortisol secretion follows a typical circadian tempo, with high levels at awakening, peaking about 30 minutes after wake-up (cortisol awakening response [CAR]), and a gradual decline throughout the day (i.e., diurnal cortisol slope). Various studies suggest that chronic exposure to psychological stress may be linked to ill health primarily through alterations in diurnal cortisol patterns, which is typically indexed by flattened diurnal cortisol slopes (14–16). Less consistent findings have emerged for morning cortisol and CAR (17). For example, flattened cortisol slopes are consistently linked to poor physical health (18), and recent studies also show that a flattened diurnal slope (but not CAR or morning cortisol) reliably predicts elevated risk of early all-cause and cause-specific mortality (15,16).

Low SES, which is typically associated with chronic stress (19), has been linked to alternations in diurnal cortisol rhythm (20–25). Relative to their higher-SES counterparts, lower-SES individuals face more stressors (26), deal with more severe challenges in their daily lives (27), have lower control over their environment (28), and encounter more obstacles in accomplishing their goals (29). Lower-SES individuals also have fewer psychosocial resources to cope with the stressors they encounter (8), putting them at excess vulnerability for negative affect. In this vein, a large body of work has investigated associations between SES and three clusters of emotions believed to be intimately connected to health: depressive affect (characterized by sadness, anhedonia, and low arousal), anxiety (characterized by fear, restlessness, and high arousal), and anger (an approach-oriented state characterized by frustration) (8). The evidence emerging from this research shows that higher SES is consistently linked to depressive affect, both in cross-sectional (30,31) and prospective studies (32–34). Although some work has shown that SES is also negatively linked to anxiety and anger (35), these associations have not been as consistent as in the case of depressive affect (8), making this cluster of emotions a primary candidate for the links between SES, diurnal cortisol patterns, and mortality.

In a similar fashion, decades of work have shown that the HPA axis is particularly sensitive to negative emotional experiences (36–38). Interestingly, although researchers have paid attention to several facets of negative affect (37,39,40), this line of research also reveals that the most consistent links in the literature are between depressive affect and cortisol slope (36,39,41). This observation was supported by a recent meta-analysis on the links between daily cortisol slopes and health, which found significant links between flatter cortisol slopes and higher depressive affect, but only a marginally significant link with anxiety (18). These findings are also in line with laboratory work showing that cortisol reactivity is higher in response to situations that evoke emotions such as worthlessness, shame, and guilt, which are key features of depressive symptoms (42–44).

Together, these series of findings provide strong evidence that negative affect (i.e., distress) and daily cortisol slope may serve as pathways connecting SES to mortality risk (7,8). Furthermore, when looking at the psychological mechanisms that may underlie the links between SES, cortisol slope, and health, the evidence converges on the observation that the type of negative affect may matter, with depressive affect, but not anxiety or anger, showing the most consistent associations. However, this literature needs to be extended in two important directions. First, although researchers have investigated specific components in this hypothesized chain (i.e., links between SES and daily cortisol slope, SES and mortality, and daily cortisol slope and mortality), no study, to our knowledge, has directly tested this full pathway. Second, current research has primarily documented links between chronic negative affect (e.g., symptoms of major depression) and SES (e.g., (32,33)), or chronic negative affect and diurnal cortisol (e.g., (40)), but has not focused on the role of daily affective experiences. This distinction is important, given that daily psychological processes can serve as one of the most proximal mechanisms linking stress to health (45), and findings across several outcomes indicate that aggregated measures of daily psychological states (e.g., (46,47)) are more strongly associated with biological processes than global states. Here, we expanded upon this work and investigated whether daily depressive affect might serve as a proximal psychological mediator in the hypothesized links from SES to diurnal cortisol levels and ultimately mortality. Specifically, we hypothesized that SES and daily depressive affect would be associated with dysregulations in diurnal cortisol rhythm and all-cause mortality. We further hypothesized that the prospective link between socioeconomic disadvantage and all-cause mortality 13 years later would be mediated in a serial fashion by daily depressive affect and diurnal cortisol levels.

Although a large body of research suggests that depressive affect may be uniquely associated with SES and daily cortisol slope (8,18), given previous theoretical and empirical work on SES, anxiety, anger, and health (8), we tested the mediating role of these clusters of negative emotions in an exploratory fashion.

METHODS AND MATERIALS

Participants and Procedure

Data were drawn from the second wave of the National Study of Daily Experiences (NSDE 2; $n = 2022$), a subsample of the second wave of the Midlife Development in the United States (MIDUS 2; n = the main sample of 4963 and the Milwaukee sample of 592), a national longitudinal study on healthy aging among community individuals. The Milwaukee sample was recruited from Milwaukee, Wisconsin, to oversample African Americans in the original MIDUS sample. Participants who completed the MIDUS 2 telephone interview and self-administered questionnaires between 2004 and 2005 were eligible to participate in NSDE 2, conducted between 2004 and 2009. On average, NSDE 2 took place about 22 months (range, 3–56 months) after the MIDUS 2 survey administration. The NSDE 2 is a daily stress project that included daily telephone interviews across 8 consecutive days and an assessment of salivary cortisol across 4 of the 8 days. Among the 2022 participants, 1736 (85.9%) provided saliva samples. Of the 1736 with saliva samples, 1 did not provide valid cortisol data, resulting in a final sample of 1735 adults between the ages of 33 and 84 years (56.4% female, 85.5% White). Mortality status was tracked through June of 2018. The original MIDUS 2 was approved by

[1]Although there are many ways to define SES (e.g., see Ref. (3)), in line with previous work on SES in MIDUS (4), here we adopt the view that SES reflects a multidimensional construct best characterized by a combination of factors that indicate access to material and cultural resources (such as income, education, and financial distress).

the institutional review board at participating institutions, and informed consent was obtained from all participants.

Primary Measures

Socioeconomic Status

In line with approaches that conceptualize SES as a multifaceted construct (48,49), we created an index reflecting social and economic standing by combining information about participants' education, income, and perceptions of financial stress. Specifically, participants reported their education level (1, no school/some grade school; 12, any doctorate/professional degree), household-adjusted income, difficulty in paying monthly bills (1, very difficult; 4, not at all difficult), availability of money to meet needs (recoded as 1, not enough money; 3, more money), and current financial situation (0, the worst; 10, the best) in the MDUS 2 survey. Following previous work (50), each indicator was standardized and then summed into a composite score, with higher scores reflecting higher SES.

Daily Negative Affect

Daily experiences of negative affect were measured over the 8 days of the NSDE 2 using the Affect Scale developed for MIDUS (51,52). Participants rated how accurately each of 14 emotional states described their mood over the day on a 5-point scale of 0 (none of the time) to 4 (all of the time). To examine the potential different effects of various facets of negative affect on health, these emotional states were categorized into three domains: experiences of depressive affect (worthless, sad, hopeless, lonely, ashamed, everything was an effort; Cronbach α was .79), anxiety (restless, afraid, nervous, jittery; Cronbach α was .79), and anger (irritable, upset, angry, frustrated; Cronbach α was .88). A composite score for each domain of daily negative affect was created by averaging the aggregated scores of the corresponding emotional states across the 8 days, with higher scores indicating higher levels in each domain. Confirmatory factor analysis showed that a three-factor model fitted the data acceptably ($\chi^2(74) = 299.94$, comparative fit index = .91, root mean square error of approximation = 0.04).

Diurnal Cortisol

Diurnal cortisol was assessed on 4 of the 8-day sampling in NSDE 2. Saliva samples were collected with Salivettes (Sarstedt, Romelsdorft, Germany) four times a day: immediately upon waking, 30 minutes later, before lunch, and at bedtime. On average, participants provided 15.5 (standard deviation = 1.3) saliva samples of 16 possible samples. Cortisol concentrations were determined with a commercially available luminescence immunoassay (IBL, Hamburg, Germany) with intra-assay and interassay coefficients of variability less than 5%.

Mortality Status

Mortality status (0, not deceased; 1, deceased) was tracked through June 2018 from multiple sources: (1) the National Death Index search by 2016, (2) MIDUS 3 (2013–2015) tracing, mortality closeout interview, and (3) the longitudinal sample maintenance. In the current study, 242 (13.9%) participants were deceased.

Covariate Measures

Covariates were selected based on prior research on mortality (53) and cortisol (54) and included the following: demographic (i.e., sex, race/ethnicity, age), psychological (i.e., history of depressive symptoms indicated by the presence of self-reported symptoms of a major depressive episode(s) over 2 weeks in the past 12 months, daily positive affect), behavioral (i.e., smoking, alcohol use), and health covariates (i.e., medical conditions over the past 12 months, history of heart disease or cancer, self-reported body mass index). Also, we included average wake-up time and medication use across the NSDE 2 days of salivary cortisol sampling as covariates in the models for diurnal cortisol (see the supplementary material, http://links.lww.com/PSYMED/A778 for details).

Statistical Analyses

First, two-level multilevel modeling (MLM) was used to examine the effects of SES and daily negative affect composites on diurnal cortisol using maximum likelihood estimation with robust standard errors in Mplus 7.0 (55). At level 1, time since waking, time since waking-squared, and CAR (1, 30-minute sample; 0, other samples) were included to estimate the diurnal cortisol profile (Equation 1). CAR samples that exceeded the 30-minutes required interval by 10 minutes or more were excluded from analyses (i.e., about 24.9% of total CAR samples). At level 2, SES, daily negative affect composites, and covariates were included as predictors of cortisol parameters. Cortisol intercept, slope (effect of time), and CAR were treated as random effects, whereas time since waking-squared was treated as a fixed effect with no level 2 predictors (Equation 2). The unconditional MLM model was first performed with level 1 predictors to depict the average diurnal cortisol profile (model 1). We then ran conditional MLM models to test the effects of SES and daily negative affect composites on diurnal cortisol (model 2). Raw cortisol values were natural log-transformed to correct for positive skew, and a constant of 1 was added before transformation to ensure that all transformed values were positive. Continuous variables at level 2 were grand-mean centered (56).

Equation 1 (level 1):

$$\text{Cortisol}_{ij} = \pi_{0j} + \pi_{1j}(\text{CAR}_{ij}) + \pi_{2j}\left(\text{time since waking}_{ij}\right) + \pi_{3j}\left(\text{Time since waking}_{ij}{}^{2}\right) + \varepsilon_{ij}$$

Equation 2 (level 2):

$$\pi_{0j} = \beta_{00} + \beta_{0k}\ (\text{personal}-\text{level predictors}) + \mu_{0j}$$

$$\pi_{1j} = \beta_{10} + \beta_{1k}\ (\text{personal}-\text{level predictors}) + \mu_{1j}$$

$$\pi_{2j} = \beta_{20} + \beta_{2k}\ (\text{personal}-\text{level predictors}) + \mu_{2j}$$

$$\pi_{3j} = \beta_{30}$$

Second, Cox regression was used to separately test the effects of SES, daily negative affect composites, and cortisol parameters on all-cause mortality (model 3). Individual differences in cortisol parameters were computed using the MLM model, with wake-up time and medication use included as covariates at level 2. SES was included as a covariate for models concerning daily negative affect composites and cortisol parameters. Continuous variables were standardized so that the hazard ratio (HR) reflected the changes in the ratio of the hazard rate for 1 standard deviation change in continuous variables. Cox regression analyses were conducted in SPSS 26.0 (IBM Corp., Armonk, New York).

TABLE 1. Participants' Characteristics as a Function of Mortality Status

Variables	Overall (n = 1735)	All-Cause Mortality as of June 2018		p
		No (n = 1493)	Yes (n = 242)	
Female, n (%)	979 (56.4)	861 (57.7)	118 (48.8)	.010
Race/ethnicity, n (%)				
White	1483 (85.5)	1272 (85.3)	211 (87.2)	
African American	171 (9.9)	152 (10.2)	19 (7.9)	
Other	80 (4.6)	68 (4.6)	12 (5.0)	.52
Medical conditions (yes), n (%)	1338 (78.9)	1128 (77.4)	210 (88.2)	<.001
Had a history of heart disease or cancer, n (%)	498 (28.8)	374 (25.1)	124 (51.5)	<.001
Smoking (yes), n (%)	213 (12.3)	178 (11.9)	35 (14.5)	.26
Alcohol use (yes), n (%)	293 (16.9)	242 (16.2)	51 (21.1)	.061
Medication use (yes), n (%)	639 (43.2)	555 (43.3)	84 (42.6)	.86
Body mass index, mean (SD), kg/m^2	28.26 (5.92)	28.18 (5.94)	28.70 (5.75)	.23
Age, mean (SD), y	56.40 (12.10)	54.36 (11.09)	69.00 (10.36)	<.001
Average wake-up time, mean (SD), h	6.70 (1.36)	6.68 (1.36)	6.79 (1.34)	.27
History of depressive symptoms, mean (SD)	0.50 (1.61)	0.48 (1.59)	0.56 (1.70)	.49
Daily positive affect, mean (SD)	2.74 (0.70)	2.74 (0.69)	2.70 (0.78)	.38
Socioeconomic status, mean (SD)	0.01 (3.55)	0.13 (3.57)	−0.71 (3.33)	.001
Daily depressive affect, mean (SD)	0.11 (0.24)	0.10 (0.21)	0.16 (0.37)	.020
Daily anxiety, mean (SD)	0.21 (0.29)	0.20 (0.27)	0.27 (0.01)	.18
Daily anger, mean (SD)	0.31 (0.35)	0.31 (0.34)	0.28 (0.36)	.13
Cortisol at awakening[a], mean (SD)	2.62 (0.40)	2.61 (0.39)	2.68 (0.43)	.015
Cortisol awakening response[a], mean (SD)	0.43 (0.07)	0.43 (0.07)	0.42 (0.08)	.011
Diurnal cortisol slope[a], mean (SD)	−0.13 (0.03)	−0.13 (0.03)	−0.11 (0.03)	<.001

SD = standard deviation.

p Values obtained from t tests for continuous variables and χ^2 tests for categorical variables.

[a] Aggregated diurnal cortisol parameters computed from the unconditional multilevel model (model 1).

Third, PROCESS macro for SPSS was used to examine the indirect effect of SES on all-cause mortality through daily negative affect composites and diurnal cortisol parameters (57). The covariates were adjusted for both the mediators (daily negative affect and cortisol) and mortality (model 4). Standard errors were obtained using the bootstrapping method based on 1000 resamples. Indirect effects were tested using the joint significance test, and the 95% bias-corrected bootstrap confidence intervals (CIs) were also obtained for the indirect effects (58). The incidence of missing data was about 3% at level 2, and the expectation-maximization algorithm was used to impute missing data on continuous variables and mode imputation was used for categorical variables. Previous studies suggest that the expectation-maximization approach may obtain less biased estimates than ad hoc methods (e.g., listwise deletion; (59)).

Lastly, sensitivity analyses were conducted to assess the robustness of the results from the primary analyses outlined previously. Mainly, more stringent exclusion criteria were applied for cortisol values, following recent MIDUS articles on cortisol (60). Cortisol values were deleted from data analyses if the values >60 nmol/L or collected on days participants woke before 4:00 AM or after 11:00 PM.

RESULTS

Descriptive Results

Table 1 displays participants' demographic and psychological characteristics and diurnal cortisol patterns by mortality status. Compared with participants who were alive, those who were deceased were more likely to be men and have a history of medical conditions, heart disease, or cancer. Participants who were deceased were also older, had lower SES and higher levels of daily depressive affect, and exhibited higher levels of cortisol at wakening but more blunted CAR and diurnal cortisol slope (p values < .05). Table 2 displays bivariate correlations between the variables.

SES, Daily Negative Affect, and Diurnal Cortisol

SES was positively associated with cortisol at awakening (β_{01} = 0.009, p = .014) and CAR (β_{01} = 0.007, p = .015), but was negatively associated with the diurnal cortisol slope (β_{21} = −0.001, p = .012). In terms of three daily negative affect composites, only daily depressive affect was associated with diurnal cortisol slope (β_{22} = 0.017, p = .001). No other significant associations emerged between daily anxiety and anger and cortisol parameters (p values > .10; Table 3).

SES, Daily Negative Affect, Diurnal Cortisol, and All-Cause Mortality

Cox regression (model 3) showed that higher SES was associated with a lower risk of all-cause mortality (HR = 0.94, 95% CI = 0.90–0.97, p = .001). Of the three daily negative affect composites, none of them were associated with mortality (p values > .05; Table 4). Of the three diurnal cortisol parameters, a blunted diurnal

TABLE 2. Bivariate Correlations Between Study Variables

Variables	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21
1. SES	—																				
2. Daily DA	−0.26***	—																			
3. Daily anxiety	−0.18***	.62***	—																		
4. Daily anger	−0.13***	0.56***	0.64***	—																	
5. CAA [a]	0.15***	−0.06**	−0.04	−0.02	—																
6. CAR[a]	0.09***	−0.07**	−0.03	−0.02	0.32***	—															
7. DCS [a]	−0.18***	0.15***	0.08**	0.02	−0.14***	−0.28***	—														
8. Mortality status	−0.08**	0.08***	0.04	−0.04	0.06**	−0.06*	0.16***	—													
9. Sex	−0.13***	0.05*	0.07**	0.08**	−0.12***	0.07**	0.01	−0.06**	—												
10. African Americans	−0.27***	0.17***	0.07**	0.02	−0.22***	−0.09***	0.28***	−0.03	0.06*	—											
11. Other races	−0.04	0.00	−0.00	−0.00	0.00	0.01	0.04	0.01	−0.02	−0.07**	—										
12. Age	0.02	−0.07**	−0.07**	−0.25***	0.15***	0.04	0.11***	0.42***	−0.03	−0.06*	−0.00	—									
13. Smoking	−0.20***	0.16***	0.13***	0.12***	−0.04	0.01	0.13***	0.03	−0.01	0.02	0.02	−0.14***	—								
14. Alcohol use	0.14***	0.00	0.02	0.02	0.07**	−0.01	0.00	0.04	−0.17***	−0.05*	0.01	0.04	0.02	—							
15. Chronic condition	−0.09***	0.08**	0.13***	0.06*	−0.03	−0.00	0.09**	0.09**	0.07**	0.05	0.02	0.17***	0.00	−0.02	—						
16. Cancer or heart diseases	−0.04	0.05*	0.04	−0.01	0.04	−0.04	0.06*	0.20***	−0.03	−0.04	0.02	0.33***	−0.04	0.02	0.12***	—					
17. History of DS	−0.17***	0.34***	0.27***	0.26***	−0.04	−0.03	0.05*	0.02	0.10***	−0.01	−0.01	−0.11***	0.15***	−0.01	0.10***	0.07**	—				
18. Daily positive affect	0.15***	−0.47***	−0.45***	−0.52***	−0.00	0.02	−0.02	−0.02	−0.01	0.02	−0.02	0.19***	−0.11***	−0.03	−0.11***	−0.01	−0.24***	—			
19. BMI	−0.21***	0.07**	0.03	0.05	−0.13***	−0.02	0.12***	0.03	−0.03	0.20***	0.00	−0.02	−0.03	−0.13***	0.15***	0.01	0.07**	−0.07***	—		
20. Medication use	0.00	0.07**	0.12***	0.11***	−0.04	−0.01	0.04	−0.01	0.22***	−0.12***	−0.01	−0.01	−0.00	−0.01	0.20***	0.08**	0.14***	−0.10***	0.02	—	
21. Average wake —up time	−0.01	0.08**	0.05	0.05	−0.07**	0.01	0.00	0.01	0.05	0.06*	−0.02	−0.06*	0.02	−0.01	0.09**	−0.00	0.09**	−0.11***	0.03	0.10***	—

SES = socioeconomic status; DA = depressive affect; CAA = cortisol at awakening, CAR = cortisol awakening response; DCS = daily cortisol slope; DS = depressive symptoms; BMI = body mass index.

* $p < .05$.

** $p < .01$.

*** $p < .001$.

[a] Aggregated diurnal cortisol parameters computed from the unconditional multilevel model (model 1).

TABLE 3. Results of Multilevel Models Predicting Diurnal Cortisol Parameters

Fixed Effect	Multilevel Model (Model 2) Estimate	SE	*p*
Cortisol at awakening, π_0			
Average cortisol at awakening, β_{00}	2.750	0.029	<.001
Socioeconomic status, β_{01}	0.009	0.004	.014
Daily depressive affect, β_{02}	−0.068	0.078	.38
Daily anxiety, β_{03}	−0.070	0.056	.21
Daily anger, β_{04}	0.087	0.052	.090
Wake-up time, β_{05}	−0.017	0.012	.15
Medication use, β_{06}	−0.037	0.025	.14
Female, β_{07}	−0.101	0.024	<.001
African American (versus White), β_{08}	−0.328	0.043	<.001
Other (versus White), β_{09}	−0.049	0.059	.41
Smoking, β_{010}	−0.057	0.041	.16
Alcohol use, β_{011}	0.022	0.030	.47
Medical conditions, β_{012}	−0.022	0.027	.42
History of heart disease or cancer, β_{013}	−0.001	0.027	.96
Body mass index, β_{014}	−0.007	0.002	.001
Age, β_{015}	0.005	0.001	<.001
History of depressive symptoms, β_{016}	0.000	0.010	.97
Daily positive affect, β_{017}	−0.039	0.020	.056
CAR, π_1			
Average CAR, β_{10}	0.376	0.024	<.001
Socioeconomic status, β_{11}	0.007	0.003	.015
Daily depressive affect, β_{12}	−0.083	0.062	.18
Daily anxiety, β_{13}	0.039	0.054	.47
Daily anger, β_{14}	−0.008	0.041	.84
Wake-up time, β_{15}	0.008	0.009	.40
Medication use, β_{16}	−0.005	0.021	.80
Female, β_{17}	0.088	0.020	<.001
African American (versus White), β_{18}	−0.002	0.043	.97
Other (versus White), β_{19}	0.045	0.046	.33
Smoking, β_{110}	0.086	0.032	.007
Alcohol use, β_{111}	−0.018	0.024	.46
Medical conditions, β_{112}	0.007	0.023	.76
History of heart disease or cancer, β_{113}	−0.039	0.023	.084
Body mass index, β_{114}	0.003	0.002	.089
Age, β_{115}	0.002	0.001	.039
History of depressive symptoms, β_{116}	−0.002	0.006	.80
Daily positive affect, β_{117}	−0.001	0.017	.97
Time since awakening, π_2			
Average diurnal cortisol slope, β_{20}	−0.139	0.004	<.001
Socioeconomic status, β_{21}	−0.001	0.000	.012
Daily depressive affect, β_{22}	0.017	0.005	.001
Daily anxiety, β_{23}	0.001	0.005	.88
Daily anger, β_{24}	−0.004	0.004	.27
Wake-up time, β_{25}	0.000	0.001	.83
Medication use, β_{26}	0.005	0.002	.011
Female, β_{27}	0.000	0.002	.82
African American (versus White), β_{28}	0.040	0.004	<.001
Other (versus White), β_{29}	0.011	0.005	.017
Smoking, β_{210}	0.017	0.003	<.001
Alcohol use, β_{211}	0.001	0.003	.63
Medical conditions, β_{212}	0.003	0.002	.22
History of heart disease or cancer, β_{213}	0.001	0.002	.79
Body mass index, β_{214}	0.000	0.000	.008
Age, β_{215}	0.000	0.000	<.001
History of depressive symptoms, β_{216}	0.000	0.001	.77
Daily positive affect, β_{217}	0.001	0.002	.48
Time since awakening^2, π_3			
Average curvature, β_{30}	0.003	0.000	<.001

SE = standard error; CAR = cortisol awakening response.

slope was associated with an increased risk of mortality (HR = 1.21, 95% CI = 1.06–1.38, $p = .004$), whereas neither cortisol at awakening nor CAR was associated with mortality (HR = 0.93, 95% CI = 0.83–1.04, $p = .19$; HR = 0.90, 95% CI = 0.79–1.02, $p = .11$, respectively). To further interpret the significant associations between SES and diurnal cortisol slope with all-cause mortality, survival curves were plotted using the Kaplan-Meier procedure. The probability of survival was calculated at the mean, and below and above 1 standard deviation of SES (Figure 1) and diurnal cortisol slope (Figure 2).

Given that the results from multilevel models and Cox regression showed no effects of daily anger and anxiety on cortisol parameters or mortality, daily anger and anxiety were not included as mediators in the mediation model. Similarly, cortisol at awakening and CAR were dropped from the mediation model because of their nonsignificant associations with mortality. Therefore, we ran a serial mediation model to test the role of daily depressive affect and diurnal cortisol slope on the association between SES and all-cause mortality (Figure 3).[2] In these analyses, SES was negatively associated with both daily depressive affect ($b = −0.008$, $p < .001$) and diurnal cortisol slope ($b = −0.001$, $p = .011$). Daily depressive affect was associated with a flatter diurnal cortisol slope

[2]Given the significant effect of daily positive affect on mortality (Table 4), we performed post hoc analyses to examine a serial mediation model in which SES predicted all-cause mortality via daily positive affect and diurnal cortisol slope. Controlling for all covariates, SES was associated with daily positive affect ($b = 0.025$, SE = 0.005, $p < .001$). Daily positive affect, in turn, was associated with all-cause mortality ($b = −0.311$, SE = 0.122, $p = .010$), but not diurnal cortisol slope ($b = −0.001$, SE = 0.001, $p = .53$). There was a significant indirect effect from SES to all-cause mortality via daily positive affect (effect = −0.0079, 95% CI = −0.0156 to −0.0008). However, after adjusting for daily negative affect in the mediation model, SES remained associated with daily positive affect ($b = 0.011$, SE = 0.005, $p = .013$), whereas the association between daily positive affect and all-cause mortality became nonsignificant ($b = −0.217$, SE = 0.134, $p = .11$). The indirect effect of SES on all-cause mortality via daily positive affect also was not significant (effect = −0.0025, 95% CI = −0.0073 to 0.0010).

TABLE 4. Results of Cox Regression Predicting All-Cause Mortality

Variables	All-Cause Mortality (Model 3), HR (95% CI)		
Female	0.69 (0.53–0.90)	0.69 (0.53–0.91)	0.71 (0.54–0.93)
African American (versus White)	1.02 (0.62–1.67)	0.92 (0.55–1.52)	0.83 (0.50–1.38)
Other (versus White)	0.93 (0.51–1.67)	0.93 (0.51–1.67)	0.86 (0.47–1.56)
Smoking	2.22 (1.51–3.27)	2.18 (1.48–3.22)	2.14 (1.45–3.16)
Alcohol use	1.31 (0.95–1.81)	1.33 (0.96–1.84)	1.35 (0.98–1.87)
Medical conditions	1.18 (0.78–1.77)	1.17 (0.78–1.76)	1.13 (0.75–1.71)
History of heart disease or cancer	1.33 (1.02–1.73)	1.32 (1.01–1.72)	1.30 (0.99–1.70)
Body mass index	1.19 (1.03–1.37)	1.19 (1.03–1.37)	1.19 (1.03–1.37)
Age	3.96 (3.33–4.70)	3.89 (3.27–4.64)	3.91 (3.27–4.67)
History of depressive symptoms	1.10 (0.97–1.26)	1.06 (0.92–1.21)	1.11 (0.97–1.27)
Daily positive affect	0.83 (0.73–0.95)	0.90 (0.77–1.04)	0.83 (0.73–0.95)
Socioeconomic status	0.94 (0.90–0.97)	0.94 (0.90–0.98)	0.94 (0.90–0.98)
Daily depressive affect		1.15 (0.99–1.33)	
Daily anxiety		1.06 (0.90–1.25)	
Daily anger		0.95 (0.78–1.17)	
Cortisol at awakening[a]			0.93 (0.83–1.04)
Cortisol awakening response[a]			0.90 (0.79–1.02)
Diurnal cortisol slope[a]			1.21 (1.06–1.38)

HR = hazard ratio; CI = confidence interval.

HR reflected the changes in the ratio of the hazard rate for 1 standard deviation changes in continuous variables.

[a] Aggregated diurnal cortisol parameters computed from the multilevel model including wake-up time and medication use as covariates.

(b = 0.009, p = .005), and a flattened cortisol rhythm was positively associated with mortality (b = 9.441, p = .001). Daily depressive affect was not directly associated with mortality (b = 0.598, p = .085), whereas SES remained associated with mortality (b = −0.056, p = .039). Lastly, there was a statistically significant indirect effect linking SES to mortality via daily depressive affect and diurnal cortisol slope (indirect effect = −0.0007, 95% CI = −0.0014 to −0.0002). The indirect effect from SES → diurnal cortisol slope → mortality was also statistically significant (indirect effect = −0.0050, 95% CI = −0.0103 to −0.0009), whereas there were no significant indirect effects from SES → depressive daily affect → mortality (indirect effect = −0.0045, 95% CI = −0.0105 to 0.0012).

Sensitivity Analyses

Sensitivity analyses (n = 1718 [14.0% deceased]) for the multilevel model (i.e., model 2), Cox regression model (i.e., model 3), and the mediation model (i.e., model 4) showed results consistent with what presented previously (see Tables S1 and S2, and Figure

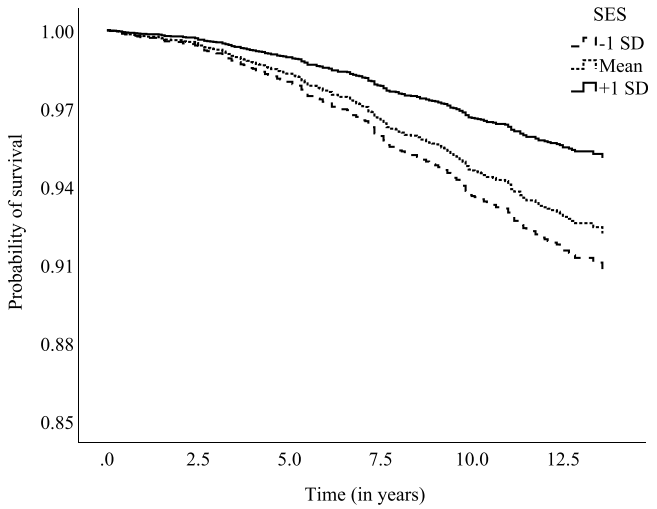

FIGURE 1. Survival curve by socioeconomic status (SES) at mean and below and above 1 standard deviation (SD).

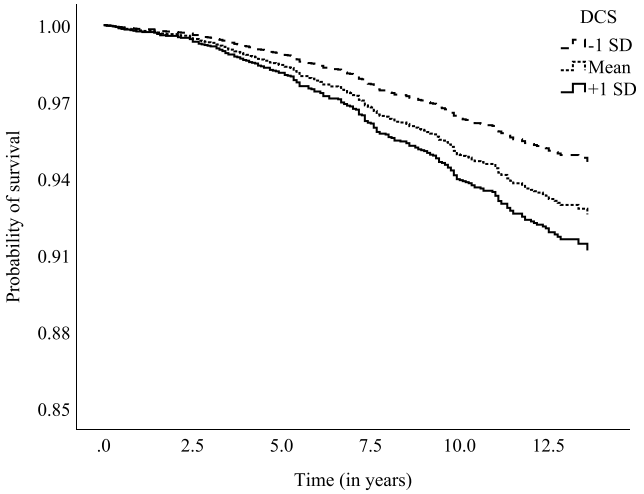

FIGURE 2. Survival curve by diurnal cortisol slope (DCS) at mean and below and above 1 standard deviation (SD).

S1, respectively, in the Supplemental Digital Content, http://links.lww.com/PSYMED/A778).

DISCUSSION

In a large study of middle-aged and older adults, we found that lower SES was associated with higher mortality risk 13 years later through higher daily negative affect and flatter ("unhealthier") diurnal cortisol slopes. These associations emerged for one particular facet of negative emotion, depressive affect, but not for anxiety or anger. These links were robust to the inclusion of a variety of psychological, behavioral, health, and demographic covariates.

Our findings are noteworthy for several reasons. First, our investigation is among the largest in a small number of studies documenting links between diurnal cortisol patterns and mortality (14–16). In line with these previous studies (e.g., (15)), we found a significant association between diurnal cortisol slope and mortality risk, but no associations between morning cortisol or CAR and mortality, providing further support for the clinical relevance of the diurnal cortisol slope as a reliable predictor of health (18). The null effects of cortisol at awakening and CAR on mortality are also somewhat consistent with previous studies documenting nonsignificant associations between cortisol at awakening and CAR and mortality (15), highlighting that more work is needed to evaluate the health relevance of these daily cortisol parameters. Second, although previous work has documented links between lower SES and flatter diurnal cortisol slope (21–24), as well as flatter diurnal cortisol slope and greater mortality risk (14–16), no previous study, to our knowledge, has examined the entire pathway from SES to mortality via diurnal cortisol. This is surprising considering that extensive theoretical work has proposed that SES may be connected to mortality risk through neuroendocrine alterations (7,8). Here, we found support for this hypothesized pathway and expanded previous research by examining the role of daily affective experiences as a psychological mechanism that may link SES to endocrine dysregulation and, ultimately, premature mortality. Our study, therefore, is the first to formally examine all the steps in a hypothesized psychobiological sequence from SES, to daily negative affect, diurnal cortisol patterns, and mortality risk, providing support for theoretical models that have advocated for these connections (8,61). Lastly, our study indicates that the links between SES, daily negative affect, diurnal cortisol, and mortality may be specific to depressive affect, but not anxiety or anger. Previous work on SES and psychological mediators of health has found that SES is more consistently associated with depressive affect (30,32,33) rather than anxiety and anger (8,10,35). Similarly, work on negative affect and diurnal cortisol levels shows that depressive affect, rather than anxiety or anger, is more consistently linked to blunted diurnal cortisol slope (39,41). Our findings support this previous work, showing that only depressive affect was significantly linked to diurnal cortisol parameters. Moreover, our

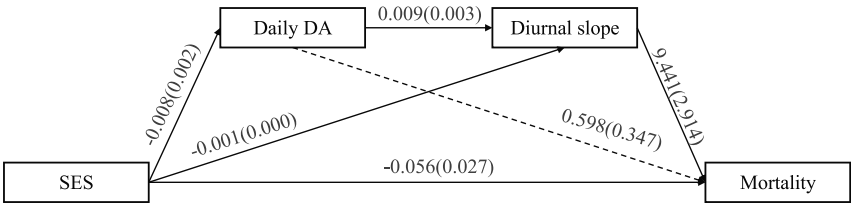

FIGURE 3. Serial mediation model connecting socioeconomic status (SES), daily depressive affect (DA), diurnal cortisol slope, and all-cause mortality. Note. Unstandardized coefficients (standard errors) are presented. Solid paths represent statistical significance at $p < .05$, and dashed lines represent statistically nonsignificant paths. Sex, race/ethnicity, age, smoking, alcohol use, medical conditions, history of heart disease or cancer, body mass index, daily positive affect, and history of depressive symptoms were included as covariates but not displayed for simplification. Diurnal cortisol slope was computed from the multilevel model including wake-up time and medication use as covariates.

study showed that *daily* depressive affect was associated with diurnal cortisol slope after controlling for the presence of self-reported symptoms of a major depressive episode(s), lending further support to perspectives that consider daily affective responses among the most proximal psychological mechanisms connecting stress to health (e.g., (46)). In this regard, our results agree with recent work on affective reactivity and all-cause mortality (62,63). Although affective reactivity refers to differences in emotional responses on stressor days as compared with nonstressor days and therefore may reflect a different mechanistic pathway from the one examined here, this work aligns with our conclusions on the importance of daily affect as a potential mediator of the SES–health link.

It should be noted that the effects documented here between SES, cortisol activity, and mortality risk are small; however, they are comparable with previously documented effects in the psychoneuroendocrinology and health psychology literatures. A recent meta-analysis on momentary emotions and cortisol revealed an association of $r = 0.06$ between negative emotions and cortisol (64). These effects are also comparable with other associations between health-related behaviors and clinical outcomes, such as consumption of fruits and vegetables and coronary heart disease (relative risk = 0.93; (65)), or sedentary behavior and cardiovascular disease (relative risk = 1.15; (66)). Therefore, although the indirect effect from SES, daily negative affect, and diurnal cortisol slope is small, we believe that it is meaningful and may carry the same relevance as other effects similar in magnitude found in the psychoneuroendocrinology and health psychology literatures.

One interesting question raised by our findings is why should this specificity in negative emotional experiences emerge? Previous research has shown that self-evaluative stressors, which typically evoke emotions that characterize depressive affect, such as worthlessness or shame, show stronger links to neuroendocrine activity than other types of stressors (42,43). A recent meta-analysis on diurnal cortisol slope and health further supports this work, showing that a flatter ("unhealthier") cortisol slope was significantly associated with depressive affect, but not with anxiety (18). In fact, higher levels of anxiety were marginally associated with steeper ("healthier") cortisol slopes, although the authors cautioned that this association should be validated in further work. One potential explanation, although speculative, is that anxiety and anger may not always predict adverse health outcomes (67,68). From a motivational perspective, anxiety may facilitate the mobilization of resources in response to threats (69), which may be mitigated if adequate coping mechanisms are in place. For example, research shows that the health effects of anger and neuroticism (a broad personality trait characterized by high levels of anxiety and vigilance) are inconsistent across studies (70) and can depend on various moderating factors that also modulate coping mechanisms, such as cultural context (67) or other personality traits (i.e., conscientiousness; (71)). Another possibility for these findings could be that our results are driven by participants' ability to identify and correctly label their emotional experiences (e.g., emotional clarity). Indeed, some evidence suggests that lower SES is negatively associated with the ability to unambiguously identify and label emotions (i.e., depression versus anxiety; (72)). Unfortunately, we cannot determine whether this is the case here; however, results from the confirmatory factor analysis indicated that the three emotional clusters loaded into three different factors.

Two important limitations of our work should be noted. First, the cross-sectional data of daily negative affect and diurnal cortisol limit our interpretations of the temporal relationships among SES, daily negative affect, and dysregulations in diurnal cortisol rhythm. Prospective designs are needed in the future to examine how the relationships between SES, daily negative affect, and diurnal cortisol rhythm unfold over time. Second, although our work replicates well-established effects of age and sex on mortality (5), our sample was primarily White, highly educated, and more affluent than the general US population (73), which limits the generalization of our findings to more ethnically and economically diverse populations. Racial disparities in health are pervasive in the United States and persist even after taking into account differences in education and income among Whites and African Americans (74,75). In our analyses, SES and race remained significant predictors of cortisol slope when examined simultaneously. Thus, although race and SES are often correlated, they may exert independent effects on health outcomes. Future research including more ethnically diverse samples is needed to better understand the interplay between race and SES on daily affect, cortisol fluctuations, and mortality risk.

Despite these limitations, the present findings represent an important step in the effort to understand the psychological and biological mechanisms that link SES to mortality. Although the structural issues that may give rise to the social gradient are not easy to mitigate, these efforts imply that there are other malleable factors in the pathway from SES to mortality that may be leveraged to reduce SES disparities in health.

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REFERENCES

1. Berkman LF, Syme SL. Social networks, host resistance, and mortality: a nine-year follow-up study of Alameda County residents. Am J Epidemiol 1979;109:186–204.
2. Marmot MG, Smith GD, Stansfeld S, Patel C, North F, Head J, White I, Brunner E, Feeney A. Health inequalities among British civil servants: the Whitehall II study. Lancet 1991;337:1387–93.
3. Kraus MW, Callaghan B, Ondish P. Social class as culture. In: Kitayama S, Cohen D, editors. Handbook of Cultural Psychology. New York, NY: Guilford; 2019:721–47.
4. Stringhini S, Carmeli C, Jokela M, Avendaño M, Muennig P, Guida F, Ricceri F, d'Errico A, Barros H, Bochud M, Chadeau-Hyam M, Clavel-Chapelon F, Costa G, Delpierre C, Fraga S, Goldberg M, Giles GG, Krogh V, Kelly-Irving M, Layte R, Lasserre AM, Marmot MG, Preisig M, Shipley MJ, Vollenweider P, Zins M, Kawachi I, Steptoe A, Mackenbach JP, Vineis P, Kivimäki M, LIFEPATH consortium. Socioeconomic status and the 25 × 25 risk factors as determinants of premature mortality: a multicohort study and meta-analysis of 1.7 million men and women. Lancet 2017;389:1229–37.
5. Chetty R, Stepner M, Abraham S, Lin S, Scuderi B, Turner N, Bergeron A, Cutler D. The association between income and life expectancy in the United States, 2001–2014. JAMA 2016;315:1750–66.
6. Pampel FC, Krueger PM, Denney JT. Socioeconomic disparities in health behaviors. Annu Rev Sociol 2010;36:349–70.
7. Chen E, Miller GE. Socioeconomic status and health: mediating and moderating factors. Annu Rev Clin Psychol 2013;9:723–49.
8. Gallo LC, Matthews KA. Understanding the association between socioeconomic status and physical health: do negative emotions play a role? Psychol Bull 2003;129:10–51.
9. Zilioli S, Imami L, Slatcher RB. Socioeconomic status and health-related biology: links between socioeconomic disadvantage, psychological factors, and HPA

activity in MIDUS. In: Ryff CD, Krueger RF, editors. The Oxford Handbook of Integrative Health Science. Oxford: Oxford University Press; 2018:431–42.
10. Matthews KA, Gallo LC, Taylor SE. Are psychosocial factors mediators of socioeconomic status and health connections? A progress report and blueprint for the future. Ann N Y Acad Sci 2010;1186:146–73.
11. Tsigos C, Chrousos GP. Hypothalamic-pituitary-adrenal axis, neuroendocrine factors and stress. J Psychosom Res 2002;53:865–71.
12. Susman EJ. Psychobiology of persistent antisocial behavior: stress, early vulnerabilities and the attenuation hypothesis. Neurosci Biobehav Rev 2006;30:376–89.
13. Miller GE, Chen E, Zhou ES. If it goes up, must it come down? Chronic stress and the hypothalamic-pituitary-adrenocortical axis in humans. Psychol Bull 2007;133:25–45.
14. Sephton SE, Lush E, Dedert EA, Floyd AR, Rebholz WN, Dhabhar FS, Spiegel D, Salmon P. Diurnal cortisol rhythm as a predictor of lung cancer survival. Brain Behav Immun 2013;30:S163–70.
15. Kumari M, Shipley M, Stafford M, Kivimaki M. Association of diurnal patterns in salivary cortisol with all-cause and cardiovascular mortality: findings from the Whitehall II study. J Clin Endocrinol Metab 2011;96:1478–85.
16. Ronaldson A, Kidd T, Poole L, Leigh E, Jahangiri M, Steptoe A. Diurnal cortisol rhythm is associated with adverse cardiac events and mortality in coronary artery bypass patients. J Clin Endocrinol Metab 2015;100:3676–82.
17. Chida Y, Steptoe A. Cortisol awakening response and psychosocial factors: a systematic review and meta-analysis. Biol Psychol 2009;80:265–78.
18. Adam EK, Quinn ME, Tavernier R, McQuillan MT, Dahlke KA, Gilbert KE. Diurnal cortisol slopes and mental and physical health outcomes: a systematic review and meta-analysis. Psychoneuroendocrinology 2017;83:25–41.
19. Baum A, Garofalo JP, Yali AM. Socioeconomic status and chronic stress. Does stress account for SES effects on health? Ann N Y Acad Sci 1999;896:131–44.
20. Cohen S, Schwartz JE, Epel E, Kirschbaum C, Sidney S, Seeman T. Socioeconomic status, race, and diurnal cortisol decline in the Coronary Artery Risk Development in Young Adults (CARDIA) study. Psychosom Med 2006;68:41–50.
21. Hajat A, Diez-Roux A, Franklin TG, Seeman T, Shrager S, Ranjit N, Castro C, Watson K, Sanchez B, Kirschbaum C. Socioeconomic and race/ethnic differences in daily salivary cortisol profiles: the multi-ethnic study of atherosclerosis. Psychoneuroendocrinology 2010;35:932–43.
22. Zilioli S, Imami L, Slatcher RB. Socioeconomic status, perceived control, diurnal cortisol, and physical symptoms: a moderated mediation model. Psychoneuroendocrinology 2017;75:36–43.
23. Agbedia OO, Varma VR, Seplaki CL, Seeman TE, Fried LP, Li L, Harris GC, Rebok GW, Xue QL, Tan EJ, Tanner E, Parisi JM, McGill S, Carlson MC. Blunted diurnal decline of cortisol among older adults with low socioeconomic status. Ann N Y Acad Sci 2011;1231:56–64.
24. Karlamangla AS, Friedman EM, Seeman TE, Stawksi RS, Almeida DM. Daytime trajectories of cortisol: demographic and socioeconomic differences—findings from the National Study of Daily Experiences. Psychoneuroendocrinology 2013; 38:2585–97.
25. Kumari M, Badrick E, Chandola T, Adler NE, Epel E, Seeman T, Kirschbaum C, Marmot MG. Measures of social position and cortisol secretion in an aging population: findings from the Whitehall II study. Psychosom Med 2010;72:27–34.
26. Businelle MS, Mills BA, Chartier KG, Kendzor DE, Reingle JM, Shuval K. Do stressful events account for the link between socioeconomic status and mental health? J Public Health (Oxf) 2014;36:205–12.
27. Grzywacz JG, Almeida DM, Neupert SD, Ettner SL. Socioeconomic status and health: a micro-level analysis o exposure and vulnerability to daily stressors. J Health Soc Behav 2004;45:1–16.
28. Lachman ME, Weaver SL. The sense of control as a moderator of social class differences in health and well-being. J Pers Soc Psychol 1998;74:763–73.
29. Laurin K, Engstrom HR, Alic A. Motivational accounts of the vicious cycle of social status: an integrative framework using the United States as a case study. Perspect Psychol Sci 2019;14:107–37.
30. Lorant V, Deliège D, Eaton W, Robert A, Philippot P, Ansseau M. Socioeconomic inequalities in depression: a meta-analysis. Am J Epidemiol 2003;157:98–112.
31. Stansfeld SA, Head J, Marmot MG. Explaining social class differences in depression and well-being. Soc Psychiatry Psychiatr Epidemiol 1998;33:1–9.
32. Koster A, Bosma H, Kempen GI, Penninx BW, Beekman AT, Deeg DJ, van Eijk JT. Socioeconomic differences in incident depression in older adults: the role of psychosocial factors, physical health status, and behavioral factors. J Psychosom Res 2006;61:619–27.
33. Melchior M, Chastang J-F, Head J, Goldberg M, Zins M, Nabi H, Younès N. Socioeconomic position predicts long-term depression trajectory: a 13-year follow-up of the GAZEL cohort study. Mol Psychiatry 2013;18:112–21.
34. Wang JL, Schmitz N, Dewa CS. Socioeconomic status and the risk of major depression: the Canadian National Population Health Survey. J Epidemiol Commun Health 2010;64:447–52.
35. Lund C, Breen A, Flisher AJ, Kakuma R, Corrigall J, Joska JA, Swartz L, Patel V. Poverty and common mental disorders in low and middle income countries: a systematic review. Soc Sci Med 2010;71:517–28.
36. Adam EK, Hawkley LC, Kudielka BM, Cacioppo JT. Day-to-day dynamics of experience–cortisol associations in a population-based sample of older adults. Proc Natl Acad Sci 2006;103:17058–63.
37. Polk DE, Cohen S, Doyle WJ, Skoner DP, Kirschbaum C. State and trait affect as predictors of salivary cortisol in healthy adults. Psychoneuroendocrinology 2005; 30:261–72.
38. Van Eck M, Berkhof H, Nicolson N, Sulon J. The effects of perceived stress, traits, mood states, and stressful daily events on salivary cortisol. Psychosom Med 1996;58:447–58.
39. Doane LD, Mineka S, Zinbarg RE, Craske M, Griffith JW, Adam EK. Are flatter diurnal cortisol rhythms associated with major depression and anxiety disorders in late adolescence? The role of life stress and daily negative emotion. Dev Psychopathol 2013;25:629–42.
40. Wardenaar KJ, Vreeburg SA, van Veen T, Giltay EJ, Veen G, Penninx BW, Zitman FG. Dimensions of depression and anxiety and the hypothalamo-pituitary-adrenal axis. Biol Psychiatry 2011;69:366–73.
41. Hsiao FH, Yang TT, Ho RT, Jow GM, Ng SM, Chan CL, Lai YM, Chen YT, Wang KC. The self-perceived symptom distress and health-related conditions associated with morning to evening diurnal cortisol patterns in outpatients with major depressive disorder. Psychoneuroendocrinology 2010;35:503–15.
42. Dickerson SS, Kemeny ME. Acute stressors and cortisol responses: a theoretical integration and synthesis of laboratory research. Psychol Bull 2004;130:355–91.
43. Gruenewald TL, Kemeny ME, Aziz N, Fahey JL. Acute threat to the social self: shame, social self-esteem, and cortisol activity. Psychosom Med 2004;66:915–24.
44. Dickerson SS, Mycek PJ, Zaldivar F. Negative social evaluation, but not mere social presence, elicits cortisol responses to a laboratory stressor task. Health Psychol 2008;27:116–21.
45. Birditt KS, Fingerman KL, Almeida DM. Age differences in exposure and reactions to interpersonal tensions: a daily diary study. Psychol Aging 2005;20:330–40.
46. Graham-Engeland JE, Sin NL, Smyth JM, Jones DR, Knight EL, Sliwinski MJ, Almeida DM, Katz MJ, Lipton RB, Engeland CG. Negative and positive affect as predictors of inflammation: timing matters. Brain Behav Immun 2018;74: 222–30.
47. Koffer R, Drewelies J, Almeida DM, Conroy DE, Pincus AL, Gerstorf D, Ram N. The role of general and daily control beliefs for affective stressor-reactivity across adulthood and old age. J Gerontol B Psychol Sci Soc Sci 2019;74:242–53.
48. 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.
49. Kraus MW, Piff PK, Keltner D. Social class, sense of control, and social explanation. J Pers Soc Psychol 2009;97:992–1004.
50. Gruenewald TL, Karlamangla AS, Hu P, Stein-Merkin S, Crandall C, Koretz B, Seeman TE. History of socioeconomic disadvantage and allostatic load in later life. Soc Sci Med 2012;74:75–83.
51. Kessler RC, Andrews G, Colpe LJ, Hiripi E, Mroczek DK, Normand SL, Walters EE, Zaslavsky AM. Short screening scales to monitor population prevalences and trends in non-specific psychological distress. Psychol Med 2002;32:959–76.
52. Mroczek DK, Kolarz CM. The effect of age on positive and negative affect: a developmental perspective on happiness. J Pers Soc Psychol 1998;75:1333–49.
53. Chen E, Turiano NA, Mroczek DK, Miller GE. Association of reports of childhood abuse and all-cause mortality rates in women. JAMA Psychiat 2016;73:920–7.
54. Adam EK, Kumari M. Assessing salivary cortisol in large-scale, epidemiological research. Psychoneuroendocrinology 2009;34:1423–36.
55. Muthén L, Muthén B. Mplus Statistical Modeling Software: Release 7.0. Los Angeles, CA: Muthén & Muthén; 2012.
56. Enders CK, Tofighi D. Centering predictor variables in cross-sectional multilevel models: a new look at an old issue. Psychol Methods 2007;12:121–38.
57. Hayes AF. Introduction to Mediation, Moderation, and Conditional Process Analysis: A Regression-Based Approach. New York, NY: Guilford Publications; 2017.
58. MacKinnon DP, Lockwood CM, Williams J. Confidence limits for the indirect effect: distribution of the product and resampling methods. Multivariate Behav Res 2004;39:99.
59. Schafer JL, Graham JW. Missing data: our view of the state of the art. Psychol Methods 2002;7:147–77.
60. Karlamangla AS, Merkin SS, Almeida DM, Friedman EM, Mogle JA, Seeman TE. Early-life adversity and dysregulation of adult diurnal cortisol rhythm. J Gerontol B Psychol Sci Soc Sci 2019;74:160–9.
61. 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.
62. Chiang JJ, Turiano NA, Mroczek DK, Miller GE. Affective reactivity to daily stress and 20-year mortality risk in adults with chronic illness: findings from the National Study of Daily Experiences. Health Psychol 2018;37:170–8.
63. Stanton SCE, Selcuk E, Farrell AK, Slatcher RB, Ong AD. Perceived partner responsiveness, daily negative affect reactivity, and all-cause mortality: a 20-year longitudinal study. Psychosom Med 2019;81:7–15.
64. Joseph NT, Jiang Y, Zilioli S. Momentary emotions and salivary cortisol: a systematic review and meta-analysis of ecological momentary assessment studies. Neurosci Biobehav Rev 2021;125:365–79.
65. He FJ, Nowson CA, Lucas M, MacGregor GA. Increased consumption of fruit and vegetables is related to a reduced risk of coronary heart disease: meta-analysis of cohort studies. J Hum Hypertens 2007;21:717–28.

66. Grøntved A, Hu FB. Television viewing and risk of type 2 diabetes, cardiovascular disease, and all-cause mortality: a meta-analysis. JAMA 2011;305:2448–55.
67. Kitayama S, Park J, Miyamoto Y, Date H, Boylan JM, Markus HR, Karasawa M, Kawakami N, Coe CL, Love GD, Ryff CD. Behavioral adjustment moderates the link between neuroticism and biological health risk: a U.S.-Japan comparison study. Pers Soc Psychol Bull 2018;44:809–22.
68. Park J, Kitayama S, Markus HR, Coe CL, Miyamoto Y, Karasawa M, Curhan KB, Love GD, Kawakami N, Boylan JM, Ryff CD. Social status and anger expression: the cultural moderation hypothesis. Emotion 2013;13:1122–31.
69. Halfmann E, Bredehöft J, Häusser JA. Replicating roaches: a preregistered direct replication of Zajonc, Heingartner, and Herman's (1969) social-facilitation study. Psychol Sci 2020;31:332–7.
70. Luchetti M, Barkley JM, Stephan Y, Terracciano A, Sutin AR. Five-factor model personality traits and inflammatory markers: new data and a meta-analysis. Psychoneuroendocrinology 2014;50:181–93.
71. Turiano NA, Mroczek DK, Moynihan J, Chapman BP. Big 5 personality traits and interleukin-6: evidence for "healthy neuroticism" in a US population sample. Brain Behav Immun 2013;28:83–9.
72. Mankus AM, Boden MT, Thompson RJ. Sources of variation in emotional awareness: age, gender, and socioeconomic status. Personal Individ Differ 2016;89:28–33.
73. Dienberg Love G, Seeman TE, Weinstein M, Ryff CD. Bioindicators in the MIDUS national study: protocol, measures, sample, and comparative context. J Aging Health 2010;22:1059–80.
74. Fuller-Rowell TE, Curtis DS, Doan SN, Coe CL. Racial disparities in the health benefits of educational attainment: a study of inflammatory trajectories among African American and White adults. Psychosom Med 2015;77:33–40.
75. Curtis DS, Fuller-Rowell TE, El-Sheikh M, Carnethon MR, Ryff CD. Habitual sleep as a contributor to racial differences in cardiometabolic risk. Proc Natl Acad Sci 2017;114:8889–94.