



Socioeconomic status, perceived control, diurnal cortisol, and physical symptoms: A moderated mediation model



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ABSTRACT

Social class is a robust predictor of health, with risk for disease and mortality increasing towards the lower end of the socioeconomic (SES) spectrum. While certain psychological characteristics, such as high sense of control, can protect low-SES individuals from adverse health outcomes, very few studies have investigated the biological mechanisms underlying these relationships. In this study, we tested whether sense of control mitigated the associations between SES and cortisol activity, and SES and physical health in daily life (i.e., number and severity of physical symptoms). Next, we tested whether individual differences in cortisol secretion would act as a mechanism by which SES and perceived control influenced physical health. In a large national sample from the Midlife in the United States (MIDUS) survey, we found that SES interacted with perceived control in predicting morning cortisol levels, cortisol slopes, number of physical symptoms, and severity of physical symptoms. Specifically, SES disparities in these health outcomes were more pronounced among individuals reporting low levels of perceived control than among individuals endorsing high levels of perceived control. Further, we found that a flatter cortisol slope mediated the link between lower SES and greater number and severity of physical symptoms for those individuals who reported lower levels of perceived control, but not for individuals reporting higher levels of perceived control. These findings suggest that perception of greater control may act as a buffer against the effect of low SES on health-related physiological processes.

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1. Introduction

Social class differences are robust predictors of health, with risk for disease and mortality increasing towards the lower end of the socioeconomic spectrum (Adler et al., 1994). Accumulating evidence suggests that biopsychosocial factors may constitute important mechanisms through which socioeconomic status (SES) is linked to health (Miller et al., 2011). One consistent finding within this line of research points to physiological disturbances associated with stress, such as alteration in the circadian rhythm of cortisol, as one of the pathways through which low SES leads to deleterious health outcomes (Gustafsson et al., 2010).

The steroid hormone cortisol is the end product of the hypothalamic-pituitary adrenal (HPA) axis, a neuroendocrine system responsible for coordinating biological responses to stress

(Tsigos and Chrousos, 2002). Cortisol release follows a diurnal pattern, with high levels upon wake-up that steadily decline throughout the day. Exposure to chronic stress modulates the circadian activity of cortisol, being associated with lower waking levels and a flatter cortisol decline throughout the day (Miller et al., 2007). Both of these cortisol parameters have been associated with poor health outcomes (Gunnar and Vazquez, 2001), with flatter cortisol slopes, in particular, showing links to cardiovascular disease (Matthews et al., 2006), Type II diabetes (Schoorlemmer et al., 2009), and increased mortality risk (Gustafsson et al., 2010; Kumari et al., 2011). Because low-SES individuals experience many stressors that are likely to accumulate over the life-course, it is not surprising that lower SES has been associated with both lower cortisol waking levels and flatter daily slopes (Hajat et al., 2010; Zilioli et al., 2015).

The physiological costs of low SES are thought to develop alongside psychological characteristics that are also associated with health problems, such as lower sense of control (Miller et al., 2011). Sense of control refers to the belief that one's actions can directly influence one's life outcomes (Pearlin and Schooler, 1978). These beliefs are based on perceptions of constraints and limitations

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present in the surrounding environment as well as on perceptions of individuals' abilities to control and change their life circumstances. Similar to SES, sense of control is a consistent predictor of physical health (Fauth et al., 2007; Infurna et al., 2011) and mortality (Surtees et al., 2010). Studies have shown that lower levels of perceived control are associated with reports of greater functional limitations (Lachman and Agrigoroaei, 2010), prolonged recovery from illness (Chipperfield et al., 2004), and increased likelihood of mortality due to cardiovascular disease, even among low cardiovascular disease risk individuals (Surtees et al., 2010). Despite this empirical evidence, the neuroendocrine pathways through which sense of control is related to health remain largely unexplored.

Because socioeconomically disadvantaged individuals tend to report greater constraints in their environments and lower self-efficacy in accomplishing desired goals, sense of control has often been conceptualized as a mediating pathway linking SES and health (Lachman and Weaver, 1998). In other words, accumulating SES-related challenges contribute to the development of low control beliefs over time (Gallo et al., 2005), which, in turn, account for part of the effects of SES on various health outcomes (Bosma et al., 1999; Schnittker, 2004). For example, in one prospective study involving a large sample of older adults, Bosma et al. (2005) found that self-efficacy and mastery beliefs reported at baseline explained the association between SES and incidence of heart disease five years later. In another longitudinal study, perceived mastery explained part of the link between SES and prevalence of self-reported chronic conditions one year later (Schnittker, 2004). Taken together, these findings suggest that perceived control may serve as an important proximal mediating mechanism through which low SES increases vulnerability towards health problems.

However, studies have also shown that not all low-SES individuals endorse a low sense of control, and those with a higher sense of control may be protected against the effects of socioeconomic disadvantage on health (Turiano et al., 2014). For example, Lachman and Weaver (1998) found that low-SES individuals with higher levels of perceived control report better health and lower number of acute physical symptoms than low-SES individuals with lower levels of perceived control. Most notably, this study revealed that the health outcomes for low-SES individuals with higher levels of control were quite similar to those of their high-SES counterparts. Recently, Turiano et al. (2014) showed that greater control beliefs can buffer the effects of low-SES on mortality such that higher control beliefs protected low—but not high—SES individuals from mortality risk assessed 14 years later. These findings provide empirical support for the idea that high perceived control may confer health benefits among the most socioeconomically disadvantaged and offer an alternative explanation for the relationship between low SES and perceived control, with sense of control serving as a moderating rather than mediating factor.

The current study investigated the relationship between low SES, perceived control, HPA activity, and physical health in daily life in a large national sample of U.S. adults. First we tested whether control beliefs would be associated with daily cortisol secretion. Second, in order to clarify the relationship between SES, perceived control, and HPA activity, we tested whether sense of control would mediate or moderate the association between SES and daily cortisol. Third, in the attempt to replicate previous work showing an association between lower perceived control and greater functional limitations (i.e., number and severity of physical symptoms, Lachman and Agrigoroaei, 2010; Lachman and Weaver, 1998), we also investigated whether sense of control would mediate or moderate the association between SES and self-reported physical symptoms. Lastly, we tested whether individual differences in cortisol secretion would act as a mechanism by which SES and per-

ceived control influenced physical health (i.e., number and severity of physical symptoms).

2. Method

The current analyses were based on data from the National Study of Daily Experiences (NSDE II; $N=2022$), a subsample of the second wave of the Midlife in the United States (MIDUS II) study, a large panel survey of adults between the ages of 25 and 74. The NSDE II included assessments of salivary cortisol over four days and daily phone interviews over eight days (see, Almeida et al., 2009 for further details). Eligibility criteria for the current study required that participants did not have any missing values for the variables of interest during MIDUS II, and cortisol data collection for NSDE II. The final sample consisted of 1322 adults (54.5% female, 95.8% White/Caucasian; age: $M=55.6$ years, $SD=11.66$ years). Although the average SES for this sample was higher compared to the rest of the larger MIDUS II sample ($p=0.024$), no differences between the two subsamples emerged for perceived control ($p=0.180$) or depressed affect ($p=0.189$).

2.1. Measures

2.1.1. Socioeconomic status (SES)

In line with previous studies (Gruenewald et al., 2012), we used participants' reports from the following sources in order to compute an index of SES: education level (ranging from 1 = no school/some grade school to 12 = any type of doctorate), evaluation of current financial situation (11-point Likert scale), difficulty in paying bills (where 1 = very difficult, 2 = somewhat difficult, 3 = not very difficult, 4 = not at all difficult), annual wage from the last calendar year (ranging from 1 = less than \$0 to 46 = \$500,000–\$999,999), and availability of money to meet basic needs (1 = not enough, 2 = just enough, 3 = more than enough). Scores from these scales (average $r=0.305$; range $r=0.09–0.67$, highest $p=0.001$) were standardized and summed into a composite, with higher scores indicating higher SES. SES was measured during MIDUS II.

2.1.2. Perceived control

The perceived control measure included two subscales: 1) personal mastery, assessing participants' self-efficacy in accomplishing goals, and 2) perceived constraints, assessing participants' perceptions of environmental constraints or inevitable obstacles in reaching their goals (Lachman and Weaver, 1998). Personal mastery was assessed with four items (e.g., "I can do just about anything I really set my mind to"), while perceived constraints were assessed with eight items (e.g., "I sometimes feel I am being pushed around in my life"). For both scales, participants provided responses on a scale from 1 (*strongly disagree*) to 7 (*strongly agree*). The perceived control scale was constructed by calculating the mean of the 12 items, after reverse-coding items from the personal mastery scale, so that higher values indicate greater perceived control ($M=5.61$, $SD=0.97$, $\alpha=0.87$). Perceived control was measured during MIDUS II.

2.1.3. Salivary cortisol

Salivary cortisol was collected using Salivettes (Sarstedt, Rommelsdorf, Germany). On average, saliva collection during NSDE II occurred 20.54 months ($SD=13.57$) after the MIDUS II survey administration. Participants provided saliva samples four times a day on days 2–5 from the 8-day NSDE assessment window. The daily collection time points occurred immediately upon waking, 30 min later, before lunch, and at bedtime. Cortisol concentrations were quantified with a commercially available luminescence immunoassay (IBL, Hamburg, Germany) with intra-assay and inter-assay coefficients of variability resulting below 5%. Collection

compliance was monitored with nightly phone interviews and paper-and-pencil logs included in the collection kits. A logarithmic transformation was performed on all the cortisol values in order to correct for positive skew in the distribution (Adam and Kumari, 2009). To ensure that all transformed scores were positive, a constant of 1 was added before the transformation.

2.1.4. Daily physical symptoms and symptom severity

Physical symptoms were assessed during the eight days of the NSDE II period through phone interviews with a modified version of the Larsen and Kasimatis (1991) symptom checklist. Participants were asked to report the occurrence of 28 physical symptoms including upper respiratory symptoms (e.g., sore throat, runny nose), aches (e.g., headache, muscle soreness, backache), gastrointestinal symptoms (e.g., stomach problems, diarrhea), and any other symptoms not included in the list ($M=1.73$, $SD=1.71$). The severity of each reported symptom was rated on a scale from 1 “Very Mild” to 10 “Very Severe” ($M=2.49$, $SD=1.28$).

2.1.5. Demographic covariates

Several standard covariates in diurnal cortisol studies (Adam and Kumari, 2009) were included in the analyses. Demographic covariates included age, gender (male=0, female=1), race/ethnicity (0=white, 1=non-white), average sleep duration across the NSDE II, and average wake time across the days of salivary cortisol sampling.

2.1.6. Depressed affect

Following recent work by Turiano et al. (2014) on perceived control and mortality, depressed affect was included as a covariate in the analyses. Specifically, participants reported whether they experienced depressive symptoms during 2 weeks in the past 12 months (e.g., “Did you lose interest in most things?”; “Did you have more trouble falling asleep than usual?”; “Did you think a lot about death?”). Responses on each item (0=no, 1=yes) were added to derive a continuous measure of depressed affect, so that higher scores indicate higher depressed affect. Depressed affect was measured during MIDUS II.

2.2. Data analysis

Hierarchical Linear Modeling (HLM) was employed in order to properly account for the strong diurnal rhythm of cortisol. HLM allows for the simultaneous estimation of more than one cortisol parameter (e.g., wakeup values, CAR, and slope) along with the prediction of individual differences in diurnal cortisol profiles. Based on previous diurnal cortisol research (Adam and Kumari, 2009), Time Since Waking, Time Since Waking-squared, and CAR (dummy coded 0 and 1) were modeled at Level 1 in order to estimate each participants’ diurnal cortisol profile. CAR cortisol values that deviated by 10 min or more from the requested 30-min interval were dropped from the analyses (i.e., treated as missing values at Level-1 in our multilevel models). At Level 2 (person-level), we tested the effect of SES while controlling for perceived control, and then we included the SES X perceived control interaction term. Analyses were conducted both with (reported in the tables) and without controlling for covariates (reported in the text). Cortisol intercept, slope (effect of time), and CAR were all allowed to vary randomly at Level-2, while Time Since Waking-squared was considered as a fixed effect with no Level-2 predictors. Continuous person-level variables were all standardized. All significance tests were 2-tailed with robust standard errors.

Multiple regression was employed to test whether SES, perceived control, and their interaction predicted the number of daily physical symptoms and their severity. As in the case of cortisol,

analyses were conducted with and without controlling for covariates. Continuous person-level variables were all standardized. Simple-slope analyses, controlling for covariates, were conducted to interpret significant interactions.

Lastly, PROCESS (Hayes, 2013) was used to test whether individual differences in cortisol secretion would act as a mechanism by which SES and perceived control influence physical health (i.e., number and severity of physical symptoms). Individual differences in cortisol parameters were measured using empirical Bayes residuals³ obtained from an HLM model predicting cortisol with all Level 1 variables included (Time Since Waking, Time Since Waking-squared, and CAR) and wake-up time as the only variable at Level-2 (more details about these statistical analyses are reported in the Results subsections, *Simple Mediation Analyses* and *Moderated Mediation Analyses*).

3. Results

Descriptive statistics are reported in Table 1. As reported in Table 2 (Model 1), high SES individuals had elevated morning cortisol levels ($\beta_01=0.053$, $SE=0.014$, $p<0.001$, without controlling for covariates) and a steeper diurnal slope throughout the day ($\beta_21=-0.0006$, $SE=0.001$, $p<0.001$, without controlling for covariates) compared to low SES individuals. Although SES and perceived control were correlated ($r=0.324$, $p<0.001$), the latter was not associated with either cortisol levels at awakening ($\beta_02=0.004$, $SE=0.016$, $p=0.781$, without controlling for covariates) or cortisol diurnal rhythm ($\beta_22=0.000$, $SE=0.001$, $p=0.938$, without controlling for covariates). Neither SES ($\beta_11=0.001$, $SE=0.010$, $p=0.893$, without controlling for covariates) nor perceived control ($\beta_12=0.013$, $SE=0.011$, $p=0.250$, without controlling for covariates) predicted CAR. Because perceived control was not related to any of the cortisol parameters, we can conclude that it did not mediate the relationship between SES and HPA activity.

As shown in Table 3 (Model 1), low SES individuals reported a greater number ($b=-0.329$, $SE=0.047$, $p<0.001$, without controlling for covariates) and more severe physical symptoms ($b=-0.263$, $SE=0.036$, $p<0.001$, without controlling for covariates). Similarly, individuals endorsing low levels of perceived control reported a greater number ($b=-0.325$, $SE=0.047$, $p<0.001$, without controlling for covariates) and more severe physical symptoms ($b=-0.187$, $SE=0.036$, $p<0.001$, without controlling for covariates).

3.1. Moderation analyses

Next, we tested our moderation hypothesis by adding the interaction between SES and perceived control as an additional predictor of cortisol secretion. These analyses revealed that perceived control moderated the relationship between SES and both cortisol slope ($\beta_23=0.002$, $SE=0.001$, $p=0.030$, without controlling for covariates) and morning cortisol ($\beta_03=-0.034$, $SE=0.015$, $p=0.027$, without controlling for covariates) (Table 2, Model 2). In particular, the effect of SES on cortisol slope was weaker in those individuals who reported high levels of perceived control compared to those individuals who reported low levels of perceived control. Similarly, among individuals who scored high on perceived control, SES was less strongly associated with variation in morning cortisol than among individuals who reported low levels of perceived control. Panel A of Fig. 1 depicts the association between SES

³ Bivariate correlations revealed that morning cortisol residuals positively correlated with CAR residuals ($r=0.723$, $p<0.001$) and negatively with cortisol slope residuals ($r=-0.254$, $p<0.001$), while CAR residuals and cortisol slope residuals were negatively associated ($r=-0.525$, $p<0.001$).

Table 1
Descriptive Statistics.

Descriptive variables	%	Mean or Median	SD
Female	54.46%	–	–
Non-White	4.24%	–	–
Age	–	55.60	11.66
Current Financial Situation	–	6.65	2.02
Money for Basic Needs	–	–	–
not enough	15.0%	–	–
enough	54.1%	–	–
more than enough	30.9%	–	–
Difficulty Paying Bills	–	–	–
very/somewhat difficult	23.2%	–	–
not very difficult	37.4%	–	–
not at all difficult	39.4%	–	–
Education	–	–	–
high school/GED or less	27.5%	–	–
some college/associate degree	29.5%	–	–
bachelor's degree or higher	43.0%	–	–
Individual Income, median	–	\$22,500–\$24,999	–
Average Sleep Duration	–	7.20	0.91
Average Wake-up Time	–	6.69	1.32
Perceived Control	–	5.61	0.97
Depressed Affect	–	0.46	1.55
Average Daily Physical Symptoms	–	1.73	1.71
Average Severity of Daily Physical Symptoms	–	2.49	1.28

Note: GED = General Educational Development.

Table 2
HLM Models of Diurnal Cortisol Parameters.

Fixed effect (independent variable)	Model 1			Model 2		
	Estimate	SE	P	Estimate	SE	P
Morning cortisol, π_0						
Average Morning Cortisol (Intercept), β_{00}	2.7210	0.0203	<0.001	2.7342	0.0206	<0.001
SES, β_{01}	0.0355	0.0149	0.017	0.0329	0.0147	0.026
Perceived Control, β_{02}	0.0015	0.0156	0.924	–0.0063	0.0150	0.672
SES \times Perceived Control, β_{03}	–	–	–	–0.0355	0.0151	0.019
Female, β_{04}	–0.1092	0.0252	<0.001	–0.1125	0.0250	<0.001
Age, β_{05}	0.0324	0.0128	0.011	0.0310	0.0127	0.014
Non-white, β_{06}	–0.1106	0.0554	0.046	–0.1059	0.0561	0.059
Average Waketime, β_{07}	–0.0265	0.0169	0.117	–0.0263	0.0170	0.121
Depressed Affect, β_{08}	–0.0070	0.0162	0.664	–0.0017	0.0159	0.913
Sleep Duration, β_{09}	0.0597	0.0147	<0.001	0.0621	0.0147	<0.001
Cortisol Awakening Response, π_1						
Average CAR, β_{10}	0.3775	0.0149	<0.001	0.3776	0.0154	<0.001
SES, β_{11}	0.0138	0.0107	0.196	0.0139	0.0107	0.195
Perceived Control, β_{12}	0.0120	0.0111	0.281	0.0119	0.0112	0.287
SES \times Perceived Control, β_{13}	–	–	–	–0.0007	0.0089	0.936
Female, β_{14}	0.0927	0.0212	<0.001	0.0927	0.0213	<0.001
Age, β_{15}	0.0150	0.0110	0.173	0.0150	0.0110	0.173
Non-white, β_{16}	0.0265	0.0602	0.661	0.0272	0.0602	0.652
Average Waketime, β_{17}	0.0012	0.0118	0.920	0.0014	0.0118	0.908
Depressed Affect, β_{18}	0.0026	0.0107	0.806	0.0027	0.0109	0.803
Sleep Duration, β_{19}	–0.0203	0.0118	0.087	–0.0204	0.0118	0.085
Time Since Waking, π_2						
Average Linear Slope, β_{20}	–0.1324	0.0037	<0.001	–0.1334	0.0037	<0.001
SES, β_{21}	–0.0040	0.0012	0.001	–0.0038	0.0012	0.001
Perceived Control, β_{22}	–0.0001	0.0012	0.904	0.0004	0.0012	0.717
SES \times Perceived Control, β_{23}	–	–	–	0.0027	0.0011	0.013
Female, β_{24}	0.0013	0.0023	0.558	0.0016	0.0022	0.482
Age, β_{25}	0.0046	0.0011	<0.001	0.0047	0.0011	<0.001
Non-white, β_{26}	0.0208	0.0056	<0.001	0.0204	0.0056	<0.001
Average Waketime, β_{27}	–0.0006	0.0015	0.658	–0.0007	0.0015	0.654
Depressed Affect, β_{28}	0.0017	0.0012	0.132	0.0013	0.0011	0.231
Sleep Duration, β_{29}	–0.0074	0.0013	<0.001	–0.0076	0.0013	<0.001
Time Since Waking ² , π_3						
Average Curvature, β_{30}	0.0025	0.0002	<0.001	0.0025	0.0002	<0.001

Note. Intercepts indicate average cortisol values at wakeup; average slopes of time since waking indicate change in cortisol per 1-h change in time; average slopes of time since waking² indicate change in cortisol per 1-h change in time². CAR = Cortisol Awakening Response; SES = Socioeconomic Status.

and diurnal cortisol among individuals who reported low levels of perceived control, while Panel B of Fig. 1 depicts the association

between SES and diurnal cortisol among individuals who reported high levels of perceived control.

Table 3
Multiple Regression Models of Number and Severity of Daily Symptoms.

	Model 1			Model 2		
	Estimate	SE	P	Estimate	SE	P
Average Number Daily Symptoms						
SES	−0.2560	0.0480	<0.001	−0.2490	0.0480	<0.001
Perceived Control	−0.2910	0.0470	<0.001	−0.2710	0.0480	<0.001
SES × Perceived Control	–	–	–	0.0880	0.0420	0.034
Female	0.3190	0.0910	<0.001	0.3280	0.0910	<0.001
Age	0.0860	0.0450	0.054	0.0890	0.0450	0.045
Non-white	0.0150	0.2210	0.945	0.0040	0.2210	0.986
Depressed Affect	0.2400	0.0460	<0.001	0.2270	0.0460	<0.001
Sleep Duration	−0.0860	0.0450	0.058	−0.0920	0.0450	0.042
Average Severity Daily Symptoms						
SES	−0.1980	0.0360	<0.001	−0.1920	0.0360	<0.001
Perceived Control	−0.1740	0.0350	<0.001	−0.1560	0.0360	<0.001
SES × Perceived Control	–	–	–	0.0810	0.0310	0.010
Female	0.3910	0.0680	<0.001	0.3990	0.0680	<0.001
Age	0.0990	0.0330	0.003	0.1030	0.0330	0.002
Non-white	−0.1720	0.1660	0.299	−0.1820	0.1650	0.270
Depressed Affect	0.0900	0.0340	0.009	0.0780	0.0340	0.023
Sleep Duration	−0.1330	0.0340	<0.001	−0.1390	0.0340	<0.001

Note. SES = Socioeconomic Status.

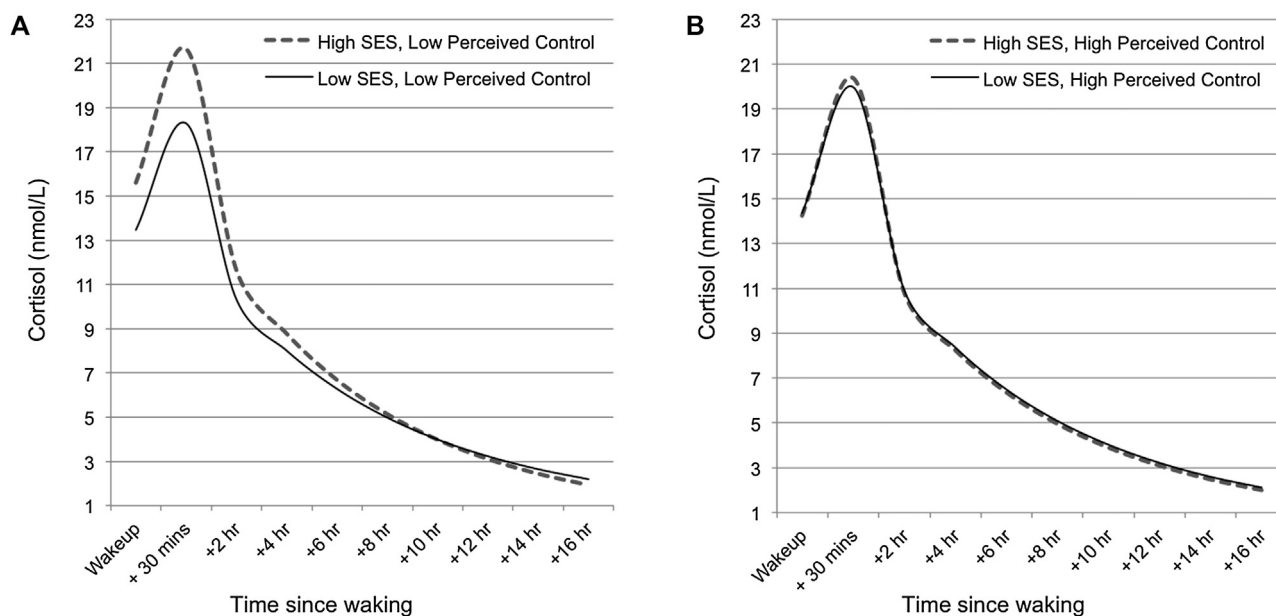


Fig. 1. Effects of SES and perceived control on diurnal cortisol levels. Cortisol level (nmol/L) is graphed as a function of time since waking for (A) participants who reported low levels of perceived control at high and low SES (i.e., levels 1 SD above and below the mean) and (B) participants who reported high levels of perceived control at high and low SES (i.e., levels 1 SD above and below the mean).

The interaction between SES and perceived control was significant when predicting both the average number of daily symptoms ($b = 0.104$, $SE = 0.042$, $p = 0.013$; without controlling for covariates) and the average severity of daily symptoms ($b = 0.074$, $SE = 0.031$, $p = 0.019$; without controlling for covariates) (Table 3, Model 2). Simple slope analyses revealed that SES had a significant negative effect on number of daily symptoms ($b = -0.160$, $SE = 0.066$, $p = 0.015$) and severity of daily symptoms ($b = -0.111$, $SE = 0.049$, $p = 0.024$) among high perceived control individuals, but the magnitude of this effect was diminished relative to low perceived control individuals ($b = -0.337$, $SE = 0.061$, $p < 0.001$, for number of symptoms; $b = -0.272$, $SE = 0.046$, $p < 0.001$, for symptoms severity) (Fig. 2). We also conducted region of significance analyses using the Johnson–Neyman technique and found that SES was not associated with number of daily symptoms for standardized values of perceived control above 1.214 ($b = -0.142$, $SE = 0.072$). Similarly, we

found that SES was not associated with severity of daily symptoms for standardized values of perceived control above 1.118 ($b = -0.102$, $SE = 0.052$).

3.2. Simple mediation analyses

Simple mediation analyses were run to test whether the association between SES and physical symptoms was mediated by individual differences in cortisol parameters (i.e., morning cortisol and cortisol slope). These analyses were run controlling for all covariates, except for wake up time. Models were run separately for number of daily symptoms and severity of daily symptoms. Bootstrapping (20,000 repetitions) was used to derive 95% confidence interval (CI) for all indirect effects. We found evidence for an indirect effect linking lower SES to greater number of physical symptoms via flatter cortisol slopes (95% CI [-0.0510 ; -0.0038]),

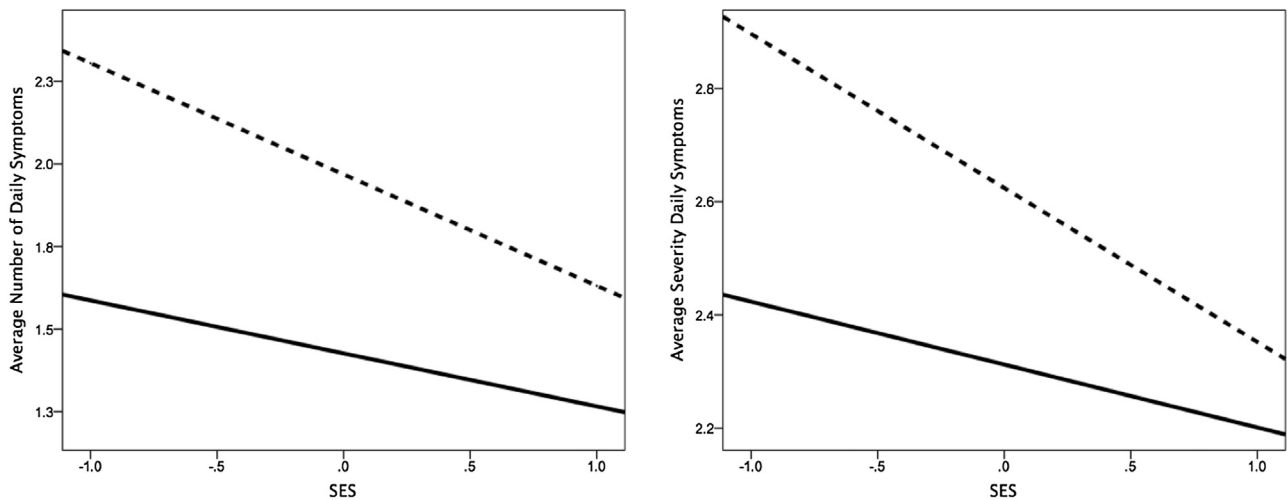


Fig. 2. Effects of SES and perceived control on average number of daily symptoms (A) and average severity of daily symptoms (B). Dotted lines represent individuals reporting low levels of perceived control (1 SD below the mean), while solid lines represent individuals reporting high levels of perceived control (1 SD above the mean).

but not via higher morning cortisol levels (95% CI [−0.0188; 0.0099]). Similar results emerged for physical symptoms severity, with cortisol slope –but not morning cortisol (95% CI [−0.0171; −0.0012])– being a significant mediator of the negative association between SES and severity of daily symptoms (95% CI [−0.0327; −0.0056]).

3.3. Moderated mediation analyses

To summarize, we found that SES interacted with perceived control in predicting cortisol slope, number of physical symptoms, and severity of physical symptoms. Further, we found evidence for an indirect effect linking SES to physical health (i.e., number and severity of daily symptoms) via cortisol slope. Based on these results we ran a moderated mediation model (for a graphical representation, see Fig. 2) to test whether the indirect effect of SES on physical health through cortisol slope was moderated by perceived control. Interestingly, we found a conditional indirect effect of SES on number of physical symptoms via cortisol slope, with the indirect effect being significant at low (95% CI [−0.0693; −0.0039]) but not at high levels of perceived control (95% CI [−0.0302; 0.0001]). This finding suggests that flatter cortisol slope mediated the link between lower SES and greater number of physical symptoms for those individuals who reported lower levels of perceived control, but not for individuals reporting higher levels of perceived control. This was confirmed by the positive and significant index of moderated mediation ($b = 0.0110$, $SE = 0.0067$, 95% CI [0.0015; 0.0291]), which represents the slope of the line for the association between the moderator (i.e., perceived control) and the indirect effect (Hayes, 2015). Similarly, the association between SES and symptoms severity was partially accounted for by cortisol slope, conditional upon individual differences in perceived control (index of moderated mediation, $b = 0.0082$, $SE = 0.0040$, 95% CI [0.0020; 0.0181]). In other words, at low levels of perceived control –but not at high levels of perceived control (95% CI [−0.0188; 0.0000001])– flatter cortisol slopes mediated the association between lower SES and higher symptoms severity (95% CI [−0.0435; −0.0064]) (Fig. 3).

4. Discussion

The current findings show that perceived control moderates the association between SES and diurnal cortisol secretion, such that SES disparities in cortisol slopes (i.e., lower SES/flatter cortisol slope) were weaker in those individuals who reported high levels

of perceived control compared to those individuals who reported low levels of perceived control. Similarly, among individuals who scored high on perceived control, SES was less strongly associated with variation in morning cortisol than among individuals who reported low levels of perceived control. A similar pattern was also found for the number and severity of daily physical symptoms, a finding that extends prior research on SES, perceived control, and functional limitations. Further, we found that flatter cortisol slope mediated the link between lower SES and greater number and severity of physical symptoms for those who reported lower levels of perceived control, but not for individuals reporting higher levels of perceived control. Notably, these effects remained significant after controlling for relevant demographic characteristics and individual differences that can bias perceptions of perceived control, such as depressive affect.

Socioeconomic disadvantage brings about a multitude of challenges that increase the risk for detrimental health outcomes over the life-course (Miller et al., 2011). Accumulating evidence, however, shows that low-SES individuals that are able to maintain resilient psychological characteristics may also be able to resist the negative health consequences evoked by disadvantage (Lachman and Weaver, 1998; Chen et al., 2012). One assumption of this line of work is that positive psychological features serve as resources that help low-SES individuals to successfully manage the stressors embedded in their environments (Gallo and Matthews, 2003). Possessing high levels of perceived control, for example, may alter the perception of stressors or facilitate adaptive coping mechanisms, which in turn, decrease maladaptive physiological responses that promote vulnerability towards disease. Our findings are consistent with this framework as they indicate that higher levels of perceived control among low-SES individuals exert beneficial effects on daily cortisol activity, resulting in cortisol profiles comparable to those of their high-SES counterparts.

Previous studies have shown that high control beliefs may reduce mortality risk (Turiano et al., 2014), functional limitations, and experiences of acute physical symptoms among low-SES individuals (Lachman and Weaver, 1998). Perhaps one of the most important implications of the current findings is that HPA activity may serve as one of the biological intermediaries through which psychological resources manifest their protective effects on health in the context of socioeconomic disadvantage. This finding complements evidence from research investigating the relationship between inflammatory markers and other psychological constructs related to resilience, such as meaning in life and “shift and per-

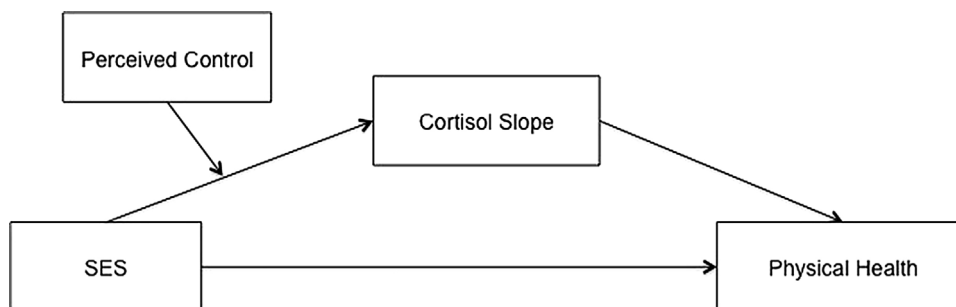


Fig. 3. Moderated mediation model depicting the indirect effect of SES on physical health (i.e., number and severity of physical symptoms) through cortisol slope and conditional upon perceived control.

sist” strategies (i.e., the ability to disassociate oneself from stressors while remaining optimistic about the future; [Morozink et al., 2010](#); [Chen et al., 2011](#)). For instance, in these previous studies, low-SES individuals that maintained a resilient psychological profile displayed more adaptive inflammatory responses, comparable to those of high-SES individuals ([Morozink et al., 2010](#); [Chen et al., 2011](#)). Our findings extend this line of work by showing similar results for control beliefs and by suggesting that alterations of the HPA axis may constitute another biological pathway through which these associations take place.

While perceived control has been treated both as a mediating and a moderating factor of the relationship between SES and health, the current findings align with the latter in showing that sense of control acts as a buffer rather than mediator of the SES–health relationship. One implication of these findings is that sense of control can be conceptualized as an individual difference that exists at varying degrees across all levels of the SES spectrum. Considering that research suggests that control beliefs are modifiable rather than immutable ([Lachman et al., 2011](#)), these findings support the idea of employing interventions in order to increase control beliefs among low-SES individuals as a way of promoting better health. Thus, future research should focus on understanding the psychosocial factors behind the development of control beliefs throughout the lifespan, so as to design effective interventions that might offset the consequences of low-SES on health.

While our findings extend prior work in suggesting that perceived control may influence physical health outcomes through neuroendocrine mechanisms, the cross-sectional design of our study does not allow us to pinpoint causality in our findings. Prospective designs tracking changes in SES status and the development of control beliefs over time are needed in order to clarify causality in the current associations. Another useful direction for future research would be to test the prospective association between SES, perceived control, cortisol, and objective clinical endpoints in order to determine if cortisol dysregulation due to low SES is associated with disease end-points over time and whether perceived control continues to moderate these associations. Our findings provide an initial step towards these future investigations by offering further evidence regarding the biopsychosocial pathways that connect SES to physical health outcomes.

Author contributions

SZ, LI and RBS: study conceptualization and writing. SZ: data analyses. SZ and LI contributed equally to the present manuscript.

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Conflict of interest

None declared.

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