Socioeconomic Status, Race, and Diurnal Cortisol Decline in the Coronary Artery Risk Development in Young Adults (CARDIA) Study

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Objectives: The objectives of this study were to assess whether socioeconomic status (SES) is associated with dysregulation of the cortisol diurnal rhythm and whether this association is independent of race and occurs equally in whites and blacks; and to determine if an association between SES and cortisol can be explained (is mediated) by behavioral, social, and emotional differences across the SES gradient. **Methods:** Seven hundred eighty-one subjects from a multisite sample representing both whites and blacks provided six saliva cortisol samples over the course of the day: at awakening, 45 minutes, 2.5 hours, 8 hours, and 12 hours after awakening, and at bedtime. **Results:** Both lower SES (education and income) and being black were associated with higher evening levels of cortisol. These relationships were independent of one another and SES associations with cortisol were similar across racial categories. The evidence was consistent with poorer health practices (primarily smoking), higher levels of depressive symptoms, poorer social networks and supports, and feelings of helplessness (low mastery) mediating the link between SES and cortisol. However, we found no evidence for psychosocial or behavioral mediation of the association between race and cortisol response. **Conclusions:** Lower SES was associated in a graded fashion with flatter diurnal rhythms as a result of less of a decline during the evening. This association occurred independent of race and the data were consistent with mediation by health practices, emotional and social factors. Blacks also showed a flatter rhythm at the end of the day. This association was independent of SES and could not be explained by behavioral, social, or emotional mediators. **Key words:** socioeconomic status, blacks, cortisol, health behavior, social support, social networks.

CARDIA = Coronary Artery Risk Development in Young Adults Study; **SES** = socioeconomic status; **HPA** = hypothalamic–pituitary adrenocortical; **BMI** = body mass index; **AUC** = area under the curve; **CES-D** = Center for Epidemiologic Studies Depression scale; **MIDUS** = Midlife in the U.S. Survey; **PAH** = Physical Activities History questionnaire.

INTRODUCTION

Increasing socioeconomic status (SES), whether measured in terms of income, education, or occupation, has been associated with decreasing rates of mortality and morbidity from almost every disease condition (1). This relationship exists across countries with and without universal access to health care, suggesting that access to care is not the primary mechanism behind this effect. One hypothesized explanation for this association has been that SES is a marker of exposure to both environmental and psychological stressors (1,2). Because persons with lower levels of SES are embedded in environments characterized by higher levels of stressor exposure, they are thought to be subject to the stress-elicited dysregulation of key behavioral and biologic systems that increase risk for disease (1,3).

One biologic system thought to be central in linking stressor exposure to disease is the hypothalamic–pituitary adrenocortical (HPA) axis. In particular, the possible role of chronic stressors in cortisol response has received considerable attention. Stressors such as caregiving and work strain have been associated with elevated cortisol (e.g., [4,5]), although not in all cases (6,7). Moreover, the diurnal rhythm of cortisol, which characteristically peaks shortly after wakening and then falls throughout the day, may be altered by chronically stressful situations (8,9). Cortisol levels that are either higher or lower than normal for any given time of day may set the stage for pathogenic processes that predispose to illness (3).

Several studies have examined the relationships between indices of SES and diurnal cortisol using ambulatory, repeated saliva collections. These studies differ in populations, sample size, timing, number of cortisol samples, and how they measured SES, and unfortunately show few consistencies in results. For example, higher SES as indicated by job grade in the Whitehall Study was associated with lower average working day cortisol levels in men but *higher* working day levels in women (10). Increasing SES was associated with *higher* morning cortisol levels in 35- to 65-year-old men and women (11). In contrast, studies of 17- to 49-year-old men (12) and 45- to 58-year-old men and women (10) found no association of SES and morning cortisol. None of these studies have found SES differences in late and end-of-the-day cortisol levels (10–12).

Because race and SES are correlated, the potential association of race and cortisol response becomes an important issue in interpreting this literature. This association is not well studied in representative healthy populations, but there is a single study suggesting that pregnant black women have higher evening and lower morning cortisol levels (13). Because of evidence that SES may play a bigger role in the

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psychologic and physical health of blacks than whites (14), it is also possible that SES interacts with race in predicting cortisol in a similar manner.

Finally, there is the question of the pathways through which SES may affect the regulation of cortisol. SES may influence cortisol levels through its effects on emotions and behaviors (1). For example, greater cortisol responses to wakeup have been related to greater perceived stress (15), depressive symptoms (16) and negative affect (17), and greater average daily cortisol levels to weak social networks and supports (18) and to the loss of sleep (19). Although these factors have also been associated with lower SES (20), none have been tested as potential mediators of relations between SES and cortisol.

The analyses reported here examine the association between SES and cortisol in a cohort of middle-aged adults—the Coronary Artery Risk Development in Young Adults Study (CARDIA). CARDIA has several strengths that make it a better model for testing this association than earlier studies. It has a large multisite sample representing both men and women. It includes measures of both education and income, allowing an examination of potentially different associations of cortisol with these two SES markers. It also includes adequate subsamples of both whites and blacks, enabling us to more clearly interpret whether observed associations are attributable to race or SES and whether SES has a greater impact on cortisol activity in blacks than in whites. Cortisol samples were collected over the course of the day so that we would be able to examine whether there were differences between groups in waking-day rhythm and, if so, when during the day the differences occurred. Finally, it includes a range of potential mediating variables, including depression, chronic burden, mastery, perceived discrimination, social network diversity, emotional support, and health practices (smoking, alcohol consumption, sleep, physical activity) that might account for associations of SES or race with cortisol.

METHODS

Participants

In 1985 to 1986, 5115 black and white men and women, aged 18 to 30 years, were recruited into CARDIA at four sites. Data reported here are based on a substudy conducted at the year-15 follow up at the Chicago, Illinois, and Oakland, California, sites. Chicago participants were randomly selected and recruited at study entry by telephone from census tracts and Oakland participants from lists of the Kaiser-Permanente Health Plan membership. Participants were chosen to achieve a balance at each site by race (black, white), sex, education (high school degree or less, more than high school), and age (18–24 years, 25–30 years) (21). They were excluded if they were blind, deaf, mute, mentally retarded, unable to walk on a treadmill, or pregnant (22). Participants were examined at study entry and years 2, 5, 7, 10, and 15. The 78.5% of CARDIA (entry) subjects who participated at year 15 were more likely to be black, younger, less educated, and smokers than those who did not (23). Site institutional review committee approval and informed consent (separately for participation in the CARDIA and cortisol collection) were obtained. This article has been approved by the CARDIA steering committee.

At the year-15 examination, subjects seen at the Chicago and Oakland sites who lived within 50 miles of the clinic ($N = 615$ and 721, respectively) were asked to participate in a substudy of SES and the development of biologic risk, including assessments of salivary cortisol. Of the 1336 subjects who were eligible for the substudy, 836 (62.6%) agreed to participate. Of these, 806 returned salivettes containing saliva and indicating the time each sample was collected. We excluded 25 participants who woke up after 11 AM (between 11:15 AM and 11:00 PM) because they had a diurnal pattern of cortisol that was noticeably different from the rest of the sample. The final analysis was based on the remaining 781 participants. Those who participated in the substudy tended to have lower education and income and higher body mass index (BMI) and diastolic and systolic blood pressure than those who did not.

Cortisol

Salivary cortisol levels closely reflect plasma free cortisol (24) and are reliable across sampling days (17). Participants were given materials and instructions regarding the collection of the salivary cortisol samples at the conclusion of the year-15 CARDIA clinic visit. Samples were collected on a single weekday, in most cases the Monday after a Friday or Saturday clinic visit. Participants were instructed not to eat, brush their teeth, or drink liquids for at least 15 minutes before taking a sample. They provided six cortisol samples over the course of the day: at awakening ("when your eyes open and you are ready to get up"), 45 minutes, 2.5 hours, 8 hours, and 12 hours after awakening, and at bedtime ("right before getting into bed"). Participants were provided with alarm watches (preset to their regular wakeup time) to remind them to collect samples and a chart that allowed them to simply readjust sample times if they woke up at a different time than anticipated. They were also instructed to record the time they woke up and (on the tube label) the time each sample was collected.

To provide a sample, participants placed a roll of cotton in their mouths, chewed on it for approximately 30 seconds or until it became saturated, and placed it in a tube called a salivette (Sarstedt, Rommelsdorft, Germany). They then filled in the time of the sample on the tube label. Samples were stored in baggies at room temperature in participants' homes and were returned to the clinic the next day (24). The salivettes were then frozen until they were assayed. Cortisol level was determined by time-resolved immunoassay with fluorometric end point detection (25). Nine samples with levels below the minimum detectable level (0.7 nmol/L) for this assay were assigned values of 0.5 nmol/L. Intra- and interassay variabilities were each less than 12%.

Most analyses were performed separately on each of the six cortisol samples. However, we were also interested in various measures that are commonly used to summarize aspects of the diurnal pattern of cortisol. The following measures were calculated:

Morning Rise

The difference between the wakeup sample and the sample collected 45 minutes later (the log of ratio of the two samples).

Area Under the Curve (AUC)

AUC was calculated using log-transformed values (to approximately normalize the distribution) and adjusting (residual from regression equation) for total waking time. (Results did not differ when waking time adjustment was not used.)

Diurnal Slope

Heuristically, we calculated the diurnal slope by separately fitting a linear regression line for each participant that predicted the log-transformed cortisol values from time (hours since awakening); in practice, our analyses of diurnal slope treated the slope and intercept as random coefficients (latent variables) in a multilevel, repeated-measures model. To avoid any effect of the morning rise on the diurnal slope, the second cortisol sample (wakeup +45 minutes) was excluded from the estimation/analysis of slopes.

Not every cortisol sample was taken at the precise time we intended. For each of the six targeted times, we determined a window within which there was little if any relationship between time since awakening and cortisol level. This window was narrowest for the morning samples as a result of the general pattern of a rapid increase in cortisol during the first 30 to 60 minutes after awakening (the "morning rise") followed by a more gradual decrease during

TABLE 1. Windows of Acceptable Times for Each Cortisol Sample

	Targeted Time	Window of Accepted Times	No. Outside Window
1	Wakeup	Wakeup–wakeup+15 min	29
2	+45 min	+15–+90 min	9
3	+2.5 hr	+2–+3.5 hr	31
4	+8 hr	+7–+9 hr	37
5	+12 hr	+11–+13 hr	41
6	Bedtime	+12–+20 hr	12

the rest of the day. The windows are listed in Table 1. The sixth sample was not targeted to a specific time. However, we observed that those samples collected after people had been awake for more than 20 hours were quite atypical. A sample provided outside the acceptable window was excluded from analyses of samples for that targeted time (see Table 1); it was, however, used in the analysis of the diurnal slope. The AUC measures were computed only for those who had data for all six samples and for whom the first, second, and last samples were within the respective windows of acceptable times. Finally, because the estimate of morning rise is very dependent on the first sample being taken at wakeup, it was only computed if the first sample was collected within 10 minutes of the time they reported waking up.

Socioeconomic Status

For SES, we used both total years of education completed as of the year-15 examination and current household income (recoded to the midpoint of each of nine categories and then cube root-transformed to reduce skewness). When graphing average diurnal patterns by SES, education was classified into three categories (completed high school [HS], completed HS and college, and at least some postcollege education) and income was classified into approximate tertiles (<$50,000, $50,000 to <$99,999, and ≥$100,000).

Potential Mediating Pathways

Depression

Depressive symptoms were measured with the 20-item Center for Epidemiologic Studies Depression scale (CES-D [26]).

Discrimination Questionnaire

The Discrimination Questionnaire is a measure of self-reported experienced frequency of discrimination based on sex, race, and/or SES as well as coping responses to perceived unfair treatment (adapted from [27]). For each of the three domains above (sex, race, SES), participants were asked to indicate whether (yes, no) and how often (rarely, sometimes, often) they had experienced discrimination in seven situational or physical settings (e.g., at school, getting a job, getting housing). Total discrimination scores in each domain were calculated by assigning values for the reported frequency of experienced discrimination in each of the seven settings (from 0 = no to 5 = often) and averaging across the responses.

Personal Control/Mastery Questionnaire

Pearlin and Schooler's (28) Personal Mastery Scale was used to assess feelings of personal control over life circumstances and outcomes versus feelings of helplessness.

Social Network Diversity

We created a network diversity index by summing three dichotomous measures: any close friends, any close relatives, any group memberships. Scores could range from zero to three (see [29]).

Emotional Support and Demands/Criticisms

Emotional support was assessed with a four-item scale (alpha = 0.83) measuring how much family members care and provide support to the respondent (30). Demands/criticisms were assessed by a four-item scale (alpha = 0.73) measuring how often family members criticize and make demands on the respondent (30). In both cases, participants responded to each item on a four-point scale (1 = not at all to 4 = a lot).

Chronic Burden

Ongoing (i.e., lasting for 6 months or longer) strains were assessed in four domains: health of close others, work, finances, and relationships (31). Respondents were asked to indicate whether (yes, no) they were experiencing an ongoing problem in each of these domains, and if so, to indicate how stressful the ongoing problem was. Ratings were made on a four-point scale (0 = no, 2 = yes, not very, 3 = yes, moderately, and 4 = yes, very stressful). We summed the number of domains for which the respondent reported experiencing moderately stressful or very stressful ongoing problems (range, 0–4).

Health Practices

Health practice measures included smoking status, alcohol consumption, sleep quality and duration, and physical activity. The health practice questionnaires can be found on the CARDIA web site (32).

Smoking status was ascertained by an interviewer-administered questionnaire. Participants were classified as current smokers if they reported smoking at least five cigarettes per week almost every week.

Alcohol consumption was assessed by separate questions regarding how many drinks of wine, beer, and liquor they usually consume in a week. Amount of alcohol consumed was determined by transforming total number of drinks (wine [17.0 mL/drink] + beer [16.7 mL/drink] + liquor [19.1 mL/drink]) into milliliters alcohol consumed in a typical week.

Physical activity was assessed by the CARDIA Physical Activities History (PAH) interviewer-administered questionnaire (33). Activity reports were based on the year before the examination. Data from the PAH were converted to exercise units for heavy and moderate intensity scores. These scores were analyzed separately and also summed to estimate total exercise units.

Sleep quality and usual number of hours of sleep are based on responses to single items from a brief sleep questionnaire. Sleep quality for the past month was rated on a five-point scale ranging from very good to very bad. Usual number of hours of actual sleep (per night) during the past month was answered in an open-ended format and recorded to the nearest half hour. Sleep duration the night before the cortisol assessments was calculated from diary reports of the time the participant reported going to sleep and waking up.

Waist was measured at the minimum girth of the abdomen. Two measurements were taken and the average used in analyses.

Control Variables

We included a series of variables as covariates in all of the analyses. These were sex (male, female), age (years), BMI (weight divided by height squared; kg/m^2), and time woke up on the day that saliva was collected. In the analysis of individual cortisol samples, the time interval between the sample collection time and the time the participant woke up is also controlled.

Statistical Analyses

The means and standard deviations of all measures are reported in Table 2. Multiple regression was used to estimate and test the association of the cortisol measures (each individual sample, the morning rise, and area under the curve) with the two SES measures (years of education and transformed income), race, sex, and the control variables (covariates). Race and sex were also controlled in analyses in which they were not a focal variable. When addressing the role of race, a separate analysis that added income and education as controls was conducted. Rather than present relatively uninterpretable regression coefficients, we present partial correlations that are an easily interpreted measure of the strength of association (effect size).

To assess the association of the diurnal slope of cortisol with SES, race, and sex, we estimated a multilevel model that treats the intercept and slope from these separate within-person regressions as latent variables (or random

TABLE 2. Sample Size, Means, and Standard Deviations (SDs) for Variables Used in Analyses

Variable	*n*	Mean	SD
Salivary cortisol measures			
Sample 1 (nmol/L)	769	20.30	16.35
Sample 2 (nmol/L)	769	25.89	15.50
Sample 3 (nmol/L)	778	15.12	12.21
Sample 4 (nmol/L)	773	10.73	12.35
Sample 5 (nmol/L)	769	6.95	8.52
Sample 6 (nmol/L)	750	7.76	11.89
Morning rise	709	0.31	0.66
Area under the curve	683	12.18	9.04
Controls (covariates)			
Sex (1 = female)	781	58%	
Race (1 = white)	781	46%	
Age (years)	781	39.95	3.64
Body mass index (kg/m^2)	778	29.32	7.40
Time woke up (hrs)	781	6.33	1.20
Socioeconomic status			
Year of education	781	14.88	2.44
Income ($1000)	777	77.80	48.33
Potential mediating pathways			
CES-D depression score	771	9.41	7.79
Sex discrimination	779	0.85	1.03
Race discrimination	781	0.81	1.04
SES discrimination	780	0.50	0.90
Personal control/mastery	781	3.12	0.62
Network diversity	781	2.74	0.44
Emotional support	781	2.48	0.59
Demands/criticisms	781	1.13	0.65
Chronic burden	781	1.20	1.21
Health practices			
Past smokers	780	17%	
Current smokers	780	19%	
Any alcoholic beverage in past yr	777	80%	
Milliliters alcohol consumed/day	776	9.85	20.36
Physical exertion—moderate	780	135.85	108.41
Physical exertion—heavy	780	215.06	228.21
Physical exertion—total	780	350.91	295.23
Sleep quality rating	781	2.51	1.00
Average hours sleep (past month)	775	6.45	1.28

CES-D = Center for Epidemiologic Studies Depression scale; SES = socioeconomic status.

coefficients) that are in turn regressed on an SES measure and the covariates. For each analysis, we estimated one model that regressed both the intercept and diurnal slope on SES and the covariates and a second model that removed the SES measure from the prediction of the diurnal slope. The partial correlation of the SES measure with the diurnal slope of cortisol is calculated as the square root of the proportional decrease in the estimated variance of the diurnal slopes in the first model relative to the second. Like in ordinary multiple regression analysis, the statistical significance of this partial correlation corresponds to that of the regression coefficient for SES (predicting diurnal slope).

RESULTS

The geometric means of each of the six cortisol samples within approximate tertiles of education and income are shown in Figures 1 and 2. Education and income exhibit similar patterns with a clear gradient emerging for the afternoon and, especially, the two evening samples; those with more education or more income have lower cortisol levels later in the day. Table 3 presents the partial correlations of education and income with the six individual cortisol samples and with morning rise, AUC, and diurnal slope controlling for race, sex, age, BMI, time woke up, and time since woke up. The statistics are consistent with what is depicted in the figures. Both income and education are associated with steeper declines over the day in diurnal slope. For both education and income, the partial correlations with the six individual cortisol samples progress from being small and nonsignificant in the morning to become more negative and increasingly statistically significant as the day progresses. Partial correlations for the AUC measure indicate a lower AUC with increasing income but no association between AUC and education. Neither income nor education was associated with morning rise.

In additional analyses (also Table 3), we added income as a control in the education analysis and education as a control in the income analysis. These analyses suggest the extent to which income and education have independent effects on the cortisol outcomes. Because income and education are moderately correlated ($r = 0.44$, $p < .001$), we expected that this analysis would result in smaller effects. As is apparent from

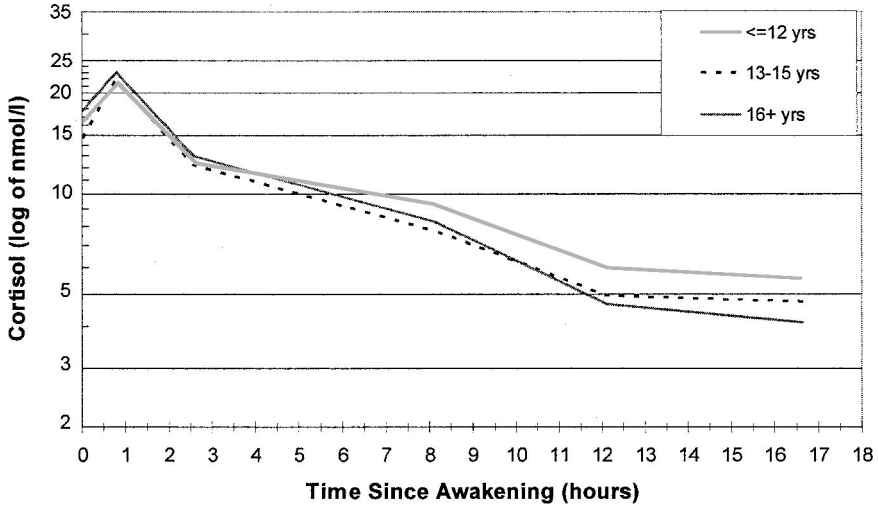

Figure 1. Mean level of six salivary cortisol samples as a function of time since awakening, by tertiles of education.

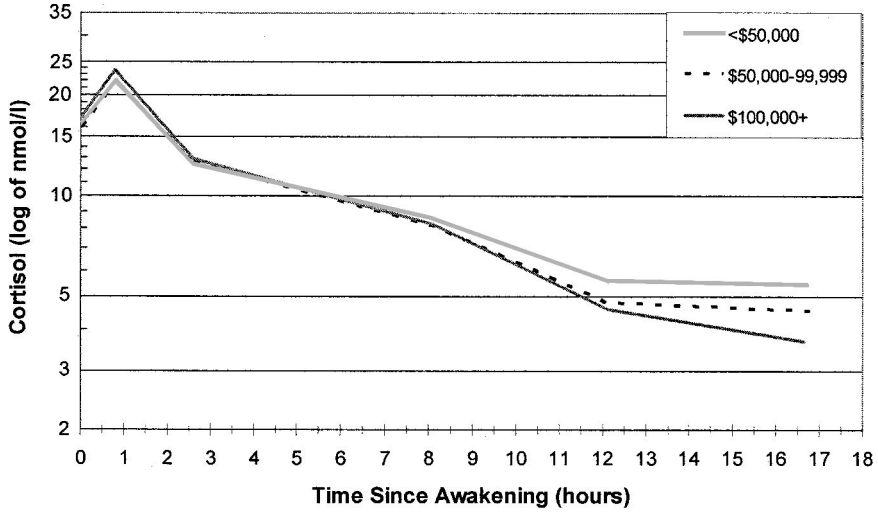

Figure 2. Mean level of six salivary cortisol samples as a function of time since awakening, by tertiles of income.

the table, income continued to predict in most cases with smaller effect sizes (drops of 25% or less). Education still predicted sample five and the slope, but the effect sizes were substantially reduced (generally by 33% or more), and in the case of sample six, the effect size dropped below significance. The reduction in the education effect when income is added to the equation suggests that some of the education effect may be proximally mediated by income.

The means for the four sex-by-race subgroups are shown in Figure 3. For race the partial correlations in Table 3 control for sex, age, BMI, time woke up, and time since woke up and for both education and income. The pattern for race is similar to that found for SES, except that the correlations progressively decrease from being positive and statistically significant in the morning to becoming significantly negative at bedtime. Black men and women have lower cortisol levels when they wake up and higher cortisol levels at the end of the day than white men and women. As a result, whites exhibit a steeper decline in cortisol during the day ($p < .001$). In general, men and women of the same race have similar cortisol levels, except for the second cortisol sample (45 minutes after awakening). For this one sample, women have higher average cortisol levels than men of the same race ($p < .01$; see [34]), although there is no sex difference in morning rise (difference between the first and second samples). The partial correlations of race with morning rise and with AUC measures are not significant.

TABLE 3. Partial Correlations (Controlling for Sex, Age, Body Mass Index, Time Woke Up, Time Since Woke Up) Predicting Cortisol Outcomes (nmol/L) From Education, Income, and Race

	Sample 1	Sample 2	Sample 3	Sample 4	Sample 5	Sample 6	Morning Rise	AUC-Log (Time-Adjusted)	Diurnal Slope
Education (controlling for race)									
Education (years)	0.03	0.02	−0.01	−0.06	−0.11**	−0.08*	−0.01	−0.07	−0.18***
n	740	760	747	736	728	738	709	684	684
Education (controlling income)	0.04	0.02	0.01	−0.04	−0.08*	−0.03	−0.03	−0.05	−0.11*
n	737	756	743	732	724	734	706	681	681
Income (cube root)									
Income (cube root)	−0.03	0.01	−0.05	−0.08*	−0.12**	−0.13***	0.05	−0.09*	−0.20***
n	737	756	743	732	724	734	706	681	681
Income (controlling education)	−0.04	0.00	−0.05	−0.06	−0.09*	0.11**	0.06	0.07	−0.15*
n	737	756	743	732	724	734	706	681	681
Race									
Race (1 = white)	0.12**	0.08*	0.04	0.00	−0.05	−0.18****	−0.05	−0.02	−0.36****
n	740	760	747	736	728	738	709	684	684
Race (continuing education + income)	0.11**	0.07*	0.05	0.03	0.01	−0.12***	−0.05	0.02	−0.26****
n	737	756	743	732	724	734	706	681	681

*$p < .05$; **$p < .01$; ***$p < .001$; ****$p < .0001$.
AUC = area under the curve.

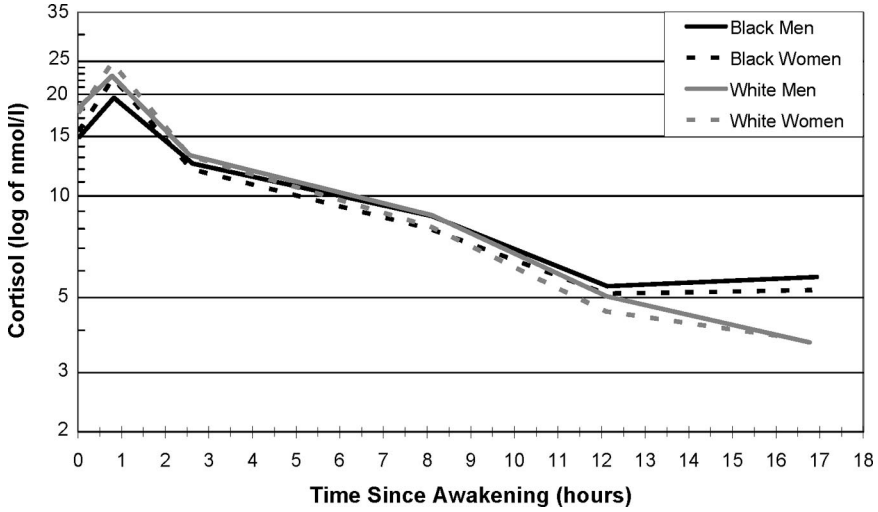

Figure 3. Mean level of six salivary cortisol samples as a function of time since awakening, by race and sex.

None of the results were substantially influenced by adding income and education to the regression suggesting that the race effect is independent of SES.

We also examined SES-by-race, SES-by-sex, SES-by-race-by-sex interactions as well as SES-by-site interactions. Overall, less than 6% of the interactions examined reached $p < .05$, a rate that is consistent with chance expectations.

Mediational Analysis

Having determined that education and income were both associated with the fifth and sixth cortisol samples and the diurnal slope, and income with AUC, we proceeded to evaluate whether these associations might be mediated by behavioral and psychosocial factors. First, we determined which of the hypothesized mediators were correlated (entering control variables as covariates) with the relevant cortisol outcomes. These data are presented in Table 4. (Table 4 is based on the maximum available sample size for each analysis.)

Then, we estimated the percent reduction in associations between SES and cortisol when we controlled for the hypothesized mediators. To avoid multicollinearity, we used education and income coefficients from the regression that did not control for the other SES marker. In each case, we estimated the percent reduction in these regression coefficients attributable to each potential mediator by adding the mediators one at a time to the equation (see percent reduction in Table 5). The greater the reduction of the effect size when proposed mediators were added, the stronger the evidence for mediation. We then estimated total mediation in two ways: by adding all the potential mediators to the equation (all potential mediators in Table 5) and by using a backward elimination procedure so that only the mediators making significant unique contributions to the outcome are included in the final equation (final parsed in Table 5). When all significant behavioral/psychosocial factors and education were simultaneously entered into a model, the estimated "total" effects of education for cortisol samples five and six and the diurnal slope were reduced by 48%, 83%, and 50%, respectively. The corresponding effects of income were reduced by 41%, 33%, and 44%; there was also a 50% reduction for AUC.

As apparent from Table 5, smoking is responsible for the greatest percent reduction in the effect of both income and

TABLE 4. Partial Correlations (Controlling for Sex, Race, Age, Body Mass Index, Time Woke Up, Time Since Woke Up) Between Proposed Psychosocial Mediators and Cortisol (nmol/L)[a]

	Sample 4	Sample 5	Sample 6	AUC-Log (Time-Adjusted)	Diurnal Slope
SES discriminatory average	−0.07*	0.03	−0.04	−0.04	0.04
CES-D	0.05	0.11**	0.10*	0.07	0.15**
Emotional support	0.01	−0.05	−0.07	−0.01	−0.12*
Network diversity	−0.05	−0.12**	−0.15***	−0.12**	−0.21***
Mastery	0.00	−0.06	−0.07	−0.01	−0.12*
Smoker	0.06	0.16***	0.13***	0.15***	0.26***
Milliliters alcohol	0.06	0.08*	0.08*	0.09*	0.15**
Sleep quality	0.00	0.14***	0.07	0.08*	0.12*
Sleep duration	−0.10**	−0.08*	0.02	−0.09*	−0.04
n	727–736	719–728	728–738	675–684	770–780

[a]Proposed mediators are limited to those associated with income or education and with at least one of the cortisol outcomes.
*$p < .05$; **$p < .01$; ***$p < .001$.
AUC = area under the curve; SES = socioeconomic status; CES-D = Center for Epidemiologic Studies Depression scale.

TABLE 5. The Percent Reduction in Coefficients Predicting Cortisol Outcomes (nmol/L) From SES Markers After Hypothesized Mediators Are Entered Into the Equation

Variable	Model 1 B	Model 1 *p*	Percent Reduction in SES Coefficient When Individual Mediator Added	Model 2 (Final Parsed) B	Model 2 *p*	Model 3 (All Potential Mediators) B	Model 3 *p*
Cortisol 4 and income							
Income	−0.05	.05		−0.05	.06	−0.05	.06
Sleep duration			7%	−0.05	.01	−0.05	.01
Percent reduction in SES coefficient				7%		7%	
n	730			730		730	
Cortisol 5 and education							
Education	−0.04	.01		−0.02	.10	−0.02	.16
Network Diversity			17%	−0.13	.04	−0.13	.04
Sleep quality			5%	0.09	.01	0.08	.01
CES-D			15%			0.00	.58
Smoking			31%	0.12	.01	0.11	.01
Milliliters alcohol			6%			0.00	.48
Sleep duration			−3%	−0.05	.04	−0.04	.06
Percent reduction in SES coefficient				43%		48%	
n	725			725		711	
Cortisol 5 and income							
Income	−0.10	.01		−0.07	.03	−0.05	.10
Negative support			8%			0.03	.56
Network diversity			9%	−0.13	.04	−0.14	.03
Sleep quality			3%	0.09	.01	0.09	.01
CES-D			18%			0.00	.83
Smoking			24%	0.12	.01	0.11	.01
Milliliters alcohol			3%			0.00	.42
Sleep duration			3%			−0.04	.09
Percent reduction in SES coefficient				30%		41%	
n	723			723		707	
Cortisol 6 and education							
Education	−0.03	.04		−0.01	.51	0.00	.75
Network diversity			36%	−0.27	.01	−0.28	.01
CES-D			19%			0.01	.15
Smoking			38%	0.12	.01	0.10	.03
Milliliters alcohol			10%			0.00	.49
Percent reduction in SES coefficient				67%		83%	
n	737			737		722	
Cortisol 6 and income							
Income	−0.13	.01		−0.09	.02	−0.08	.03
Network diversity			13%	−0.25	.01	−0.26	.01
CES-D			12%			0.00	.31
Smoking			17%	0.11	.02	0.08	.07
Milliliters alcohol			2%			0.00	.43
Percent reduction in SES coefficient				27%		33%	
n	733			733		718	
Time-adjusted AUC-log and income							
Income	−0.05	.02		−0.03	.21	−0.02	.29
Network diversity			10%	−0.12	.01	−0.11	.01
Smoking			29%	0.08	.01	0.07	.01
Milliliters alcohol			6%			0.00	.22
Sleep quality			5%			0.02	.21
Sleep duration			7%	−0.04	.02	−0.03	.02
Percent reduction in SES coefficient				45%		50%	
n	679			679		674	
Diurnal slope and education							
Education	0.00	.01		0.00	.07	0.00	.11
CES-D			12%			0.00	.77
Emotional support			4%			0.00	.55
Network diversity			17%	−0.01	.01	−0.01	.01

(Continued)

TABLE 5. (Continued)

Variable	Model 1 B	Model 1 p	Percent Reduction in SES Coefficient When Individual Mediator Added	Model 2 (Final Parsed) B	Model 2 p	Model 3 (All Potential Mediators) B	Model 3 p
Mastery			5%			0.00	.38
Smoking			30%	0.01	.01	0.01	.01
Milliliters alcohol			7%			0.00	.30
Sleep quality			3%			0.00	.26
Percent reduction in SES coefficient				44%		50%	
n	779			779		764	
Diurnal slope and income							
Income	−0.01	.01		0.00	.02	0.00	.08
CES-D			15%			0.00	.72
Emotional support			9%			0.00	.76
Network diversity			11%	−0.01	.01	−0.01	.01
Mastery			10%			0.00	.49
Smoking			24%	0.01	.01	0.01	.01
Milliliters alcohol			3%			0.00	.26
Sleep quality			2%			0.00	.25
Percent reduction in SES coefficient				32%		44%	
n	775			775		760	

SES = socioeconomic status; CES-D = Center for Epidemiologic Studies Depression scale; AUC = area under the curve.

education on samples five and six and in the diurnal slope and for income on AUC. Other contributing factors include network diversity and depression followed by smaller contributions of all of the remaining factors entered in the equation. Sleep duration is the only potential mediator in the analysis of the association of education and sample four.

We did similar analyses for potential mediation of the race effects. In this case, we used both the race coefficients from the analysis controlling for income and then from the analysis controlling for education. We do not present the statistics for race because the behavioral/psychosocial factors account for very little of the race effect (typically a 5% reduction in the coefficient).

Analysis of Nonsmokers

Given the sizable effect of smoking status on the various cortisol measures, we wondered whether the pattern of findings described here would hold if the analysis was restricted to the 64% of the sample that had never smoked. Although not statistically significant in this reduced sample ($B = -0.0016$, $p < .12$), the relationship between education and the diurnal slope is very similar to the earlier analysis of the entire sample when smoking was statistically controlled. However, education is not related to cortisol samples five or six in the nonsmokers. A somewhat different pattern emerged for income. For cortisol sample six ($B = -0.1369$, $p < .01$) and the diurnal slope ($B = -0.0069$, $p < .02$), the effects of income are *larger* (and statistically significant) in nonsmokers than in the full sample when smoking was statistically controlled. The analysis of cortisol sample five in nonsmokers results in a coefficient for income that is approximately three fourths the magnitude of the coefficient when smoking was statistically controlled ($B = 0.0556$, $p < .16$), whereas AUC was not related to income in the nonsmokers.

DISCUSSION

We found that SES, whether defined as income or education, was associated with diurnal cortisol response with decreasing levels of SES associated with relatively higher levels of cortisol during the evening and at bedtime but not at other times of the day. The higher evening and nighttime levels have some similarity to the rhythm that has been found among depressed persons (35).

The graded effects of income and education on cortisol response are striking in that both the mean education and income of this sample are much higher than that of the general population. Hence, these associations represent a gradient that occurs far above the poverty level. Surprisingly, there are independent effects of both income and education. There is some indication, however, that at least part of the education effect is mediated through income. Moreover, the reduction in either when the other is added to the equation suggests there is substantial (25% minimum) overlap in their prediction of cortisol.

With regard to race, blacks had higher levels of cortisol during the evening and this association was similar for men and women. The SES and race associations with cortisol were independent of one another, and there were no SES-by-race interactions. These results exclude an alternative explanation that the SES associations we found could be attributed to differences in racial distributions across SES groups. Moreover, they indicate that race is also associated with cortisol response and that this association cannot be explained by differences in SES across racial groups. Finally, we found no

support for the hypothesis that blacks pay a higher price when they are low in SES than do whites.

How do the effect sizes of SES and race compare with those of other predictors of the cortisol diurnal slope? The effects of income (−0.20) and education (−0.18) on the slope, controlling for race and other covariates, were somewhat smaller than the best mediator—smoking (−0.26). The effect size for race, controlling for education and other covariates, was the largest (−0.30).

Why are the differences we found apparent in the evening but not the morning? One possibility is that there is a residual (and aggregate) effect of the daily challenges involved in lower SES or minority racial status that accumulate over the course of the day. Evening levels, when cortisol should be at its nadir, may be a particularly sensitive marker of chronic stress (35). Another is that the association actually occurs across the day, but because the diurnal slope is very steep from morning to early afternoon, relatively small errors in the timing of samples (difficult to avoid in naturalistic studies) result in large errors in cortisol measurement.

How could SES influence cortisol regulation? The mediational analyses for the fifth (12 hour) and bedtime samples and for the cortisol slope indicated the potential role of a range of psychosocial and behavioral factors. In general, the increase in smoking rates as SES decreases can explain the greatest part (17–37%) of the effects, but additional variance is contributed by depression, social relationships (social network diversity, emotional support), feelings of mastery, and the remaining health practices (alcohol consumption and sleep quality). Together these variables account for between one third and two thirds of the associations. Interestingly, when we remove smokers from the analysis, the associations between income and cortisol remain and in some cases are even stronger. However, the associations between education and cortisol are attenuated. This suggests that smoking is playing a primary mediating role for the effects of education but is less important in the case of income.

In contrast, none of the psychosocial or health behavior measures could account for any of the relationship between race and cortisol response. This includes discrimination. This finding provides further support for the independence of SES and race associations with cortisol response. It also leaves us with little concrete evidence of what might account for the race–cortisol association. We can only speculate on possible pathways. First, cortisol response is partly heritable (36), and it is possible (although no evidence exists at this point) that allelic variation in the glucocorticoid receptor gene is associated with race. Second, the association between race and cortisol levels occurs at the same time of day when elevated cortisol levels have been associated with stress and depression. This suggests the possibility that negative affect does play a role, but we have not adequately tapped affective responses in this group. Finally, it is possible that factors not assessed here might account for the link between race and cortisol.

There are, of course, limitations to the study. Because our analyses are cross-sectional, causal inference is not possible. We controlled for the more obvious third-factor (spurious) variables (sex, age, BMI, wakeup time) that might be associated with both increases in cortisol and decreases in SES. Another possible spurious factor is serious illness causing both lower SES and higher cortisol. This, however, is unlikely given the sample is quite young (33–45 years old) with very low rates of morbidity. Reverse causation is also logically possible. However, it seems unlikely that late-day cortisol levels as assessed in adulthood influence social economic disparities, and they certainly do not influence racial differences. Inferences about potential mediation are at a similar disadvantage. Clearly, longitudinal evidence for earlier differences in SES, race, and mediators predicting later changes in cortisol would contribute to our confidence in the causal model central to our hypotheses.

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