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## Socioeconomic Status, Race/Ethnicity, and Diurnal Cortisol Trajectories in Middle-Aged and Older Adults

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### Abstract

**Objectives:** Slow afternoon cortisol decline may be a marker of aging. We hypothesize that lower socioeconomic status (SES) and African American race are associated with lower waking cortisol and slower afternoon decline.

**Method:** Six salivary cortisol samples, collected within a 24-hr period from 566 cohort participants aged 56–78 years, were examined in random-effects models. SES measures included socioeconomic vulnerability (household income and assets <500% of poverty) and education ( $\geq$ college, some college, and  $\leq$ high school). African Americans were compared with all others.

**Results:** Adjusting for age and sex, intermediate, but not low, education was associated with approximately 17% lower average waking cortisol and 1% slower decline, compared with high education. Socioeconomic vulnerability was not associated with waking cortisol or linear decline. Accounting for African American race/ethnicity, socioeconomic vulnerability was associated with a 3% faster decline, and education was not associated with cortisol. African Americans had 26% lower average waking cortisol and 1% slower decline than others.

**Discussion:** African American race/ethnicity, but not lower SES, was associated with lower waking cortisol and slower afternoon decline in middle-aged and older adults. This pattern is likely a marker of earlier biological aging in vulnerable groups. Race/ethnicity may compete with SES as a measure of cumulative vulnerability.

**Keywords:** Earlier aging—Education—Hypothalamic pituitary adrenal axis—Wealth

Individuals facing social adversity, including those of low socioeconomic status (SES) or racial minorities, often experience earlier biological aging than their peers (Crimmins, Kim, & Seeman, 2009; Seeman et al., 2004). This increases their risk of cognitive impairment, disability, and chronic diseases associated with the aging process (Almeida, Neupert, Banks, & Serido, 2005; Karlamangla,

Singer, McEwen, Rowe, & Seeman, 2002; Lee et al., 2007; McEwen, 1998; Sabbah, Watt, Sheiham, & Tsakos, 2008; Seeman, McEwen, Rowe, & Singer, 2001). Biological aging may also partially account for vast disparities in mortality (Seeman et al., 2004). This effect is believed to accumulate over time, producing the greatest disparities in older adults (Dannefer, 2003).

Differential exposure to stressful experiences is believed to contribute to earlier biological aging in low SES and racial minority groups (Kessler, 1979; Pearlin, 1989). There is good evidence that individuals of low SES and African Americans are more frequently exposed to stressful experiences than their peers (Almeida et al., 2005; Turner & Avison, 2003). Repeated stressful experiences may accelerate biological aging in the hypothalamic pituitary adrenal (HPA) axis (McEwen, 1998). It has been hypothesized that flattened diurnal cortisol trajectories and high late-day cortisol nadir are HPA axis markers of aging (Piazza, Almeida, Dmitrieva, & Klein, 2010) because late-day nadir increases with age (Deuschle et al., 1997; Ferrari et al., 2001; Karlamangla, Friedman, Seeman, Stawski, & Almeida, 2013; Kern, Dodt, Born, & Fehm, 1996) and flattened trajectories predict mortality (Kumari, Shipley, Stafford, & Kivimaki, 2011). High cortisol nadir likely represents loss of homeostatic regulation (Chrousos, 2009), since it appears to be due to decreased HPA axis inhibitory feedback (Wilkinson, Peskind, & Raskind, 1997). Therefore, individuals of low SES or African Americans may demonstrate slower late-day diurnal cortisol decline, compared with their peers, a marker of biological aging in the HPA axis.

Several studies have tested associations between SES, race, and late-day cortisol patterns, when HPA axis inhibition should be the most pronounced. Results are more consistent for race than SES. Middle-aged and older African Americans show slower afternoon and evening cortisol decline and higher late-day nadir than whites (Hajat et al., 2010; Karlamangla et al., 2013). However, the literature provides less insight into socioeconomic differences in late-day cortisol patterns. In the Coronary Artery Risk Development in Young Adult Study, young adults provided up to three of six samples after noon; low education, low income, and African American race were each associated with slower diurnal decline and a higher cortisol nadir (Cohen et al., 2006). These differences should be more pronounced in older adults, who have a longer duration of exposure to SES (Dannefer, 2003), but cortisol nadir has not been found to differ according to level of occupation (Gustafsson, Janlert, Theorell, & Hammarström, 2010; Steptoe et al., 2003) or financial strain (Steptoe, Brydon, & Kunz-Ebrecht, 2005). This evidence is based on data from racially homogeneous samples (e.g., Whitehall). Results may differ in racially diverse samples, where minority status itself may be a surrogate for unmeasured features of SES as well as race-associated adversity and stress (Geronimus, Hicken, Keene, & Bound, 2006; Kaufman & Cooper, 1999; LaVeist, 2005). In such settings, both low SES and minority race may contribute to HPA axis dysfunction in an additive fashion. In racially diverse samples of middle-aged and older adults (e.g., Multi-Ethnic Study of Atherosclerosis and Midlife in the United States Study), low education (Karlamangla et al., 2013) and low wealth (Hajat et al., 2010) have been associated with lower waking cortisol

and slower decline in the morning hours but not afternoon and evening hours, after adjusting for race (Hajat et al., 2010; Karlamangla et al., 2013). These studies may have been under-powered to detect difference in late-day cortisol patterns by collecting only one or two samples after noon. Therefore, there is a gap in testing associations between SES and late-day cortisol decline in racially diverse samples of middle-aged and older adults, and evaluating whether SES or race is more relevant to disparities in HPA axis aging.

One critical consideration of such research is that standard SES indicators may be prone to misclassification in older adults (Grundty & Glaser, 2000). After retirement, income and occupation may not reflect SES as well as household wealth, which is not captured in most studies. Education, although generally fixed throughout adulthood, may not capture current SES. Few studies have examined a fuller range of multidimensional measures to evaluate how each may be differentially sensitive to exposure to psychosocial hazards.

The purpose of this study was to evaluate associations between SES and African American race and diurnal cortisol trajectories in middle-aged and older adults. Since race associations have been previously documented, this study focuses on SES associations. In this study, five of the six salivary cortisol samples were collected after noon, facilitating precise modeling of cortisol trajectories during the nadir phase. We hypothesized that low SES and African American race would be associated with lower waking cortisol and slower afternoon diurnal decline as a result of less robust responsiveness of the HPA axis.

## Methods

### Study Design and Sample

The Baltimore Memory Study (BMS) is a cohort study that enrolled 1,140 adults aged 50–70 years residing in 65 contiguous Baltimore City neighborhoods in 2001 and 2002 using random household phone sampling methods, representing a 48% response rate among those deemed eligible (Schwartz et al., 2004). A detailed examination of salivary cortisol was undertaken during the fourth study visit, which took place between February 2009 and May 2010. At that time, 624 participants were re-consented and came to a clinic visit. The study was approved by the Committee for Human Research of the Johns Hopkins Bloomberg School of Public Health. Participants provided written, informed consent.

### Data Collection

All data collection was performed by trained research assistants. Baseline data used in these analyses include SES, age, sex, and race/ethnicity (analyzed as African American vs. all others). The remainder of the data used in these analyses was collected during the fourth study visit. Participants provided detailed medication data, including current use of

exogenous hormones or corticosteroids, and recorded the number of alcoholic drinks consumed and the number of cigarettes smoked (0, 1–5, 6–10, 10–15, 16–20, and >20 cigarettes) in the 48 hr prior to the clinic visit. Participants also indicated whether they had experienced any of 25 potentially stressful life events in the past week, and the severity of the perceived distress associated with those events, which was summed to create a stressful life event index. Awakening time for the day of the clinic visit was estimated based on reported time going to bed and number of hours slept. Body mass index (BMI) was computed based on measured height and weight. A medical condition summary score assigned one point for each of 15 different chronic diseases or medical events.

### Saliva Collection and Cortisol Measurement

Each participant collected two saliva samples at home prior to the clinic visit. One was collected at bedtime the night before the visit and the second was collected immediately upon awakening the morning of the visit. Participants were asked to chew lightly on a salivette swab (Sarstedt, Inc., Newton, NC) or place it under their tongue for 45 s. Participants were instructed to document the date and time for both samples and to refrigerate them in their original vials until their clinic visit. Four additional saliva samples were obtained during the study visit using the same procedure over a span of about 150 min, before, during and after cognitive testing and at visit completion. In-clinic salivary collection protocols were initiated between 12 p.m. and 6 p.m. Bedtime saliva was collected the night before the study visit; here we assume that measure to be a surrogate for the cortisol value on the day of the study visit. Other studies have found little day-to-day variation in unprovoked cortisol values (Hajat et al., 2010; Karlamangla et al., 2013). To evaluate the sensitivity of our findings to the bedtime cortisol value, we conducted a planned sensitivity analysis excluding the bedtime measure. Salivary cortisol was measured by the core laboratory of the General Clinical Research Center at the Johns Hopkins Bayview Medical Center campus, Baltimore, using a standard radioimmunoassay (Diagnostic Systems Laboratories, Inc., Webster, TX). The lower and upper limits of detection were 2.76 and 276 nmol/L, respectively, for the assay. The inter-assay coefficient of variation was 3.2%. Due to severe skew, cortisol values were *ln*-transformed prior to analyses.

### Socioeconomic Status

Household wealth and educational achievement were measured at baseline using a 110-item questionnaire developed for this study and described in detail elsewhere (Schwartz et al., 2004). A key feature of this instrument is that it assessed several sources of income (e.g., salaries, bonuses, extra income), transfers (e.g., social security, welfare, supplemental security income) and assets (e.g., homes, businesses,

vehicles, and retirement assets) for both the respondent and a spouse/partner. Also, the instrument used bracketed value ranges to reduce missing data on participants who would not report exact dollar amounts. Locally weighted scatterplot smoothing (LOWESS) graphs (Cleveland & McGill, 1985) were used to explore cortisol trajectories across SES groups and collapse SES variables in a manner that ensured comparable trajectories within SES and race groups (see Supplementary Figure 1 which examines alternative classifications). Socioeconomic vulnerability was defined as annual income <500% of the US poverty threshold and insufficient assets to remain above that threshold for more than a year, based on prior work (Caner & Wolff, 2004). A lower, more policy-relevant threshold of 250% was considered, but there were insufficient numbers of whites below this threshold to enable race-adjusted analyses. The poverty threshold for either one- or two-person households was used, depending on whether the participant reported a spouse/partner (\$8,860 and \$11,940, respectively). Educational attainment was measured by combining self-reported years of education completed with credentials and certifications acquired (e.g., degrees, certificates, trade school). Educational status was categorized as low (high school, GED, or less than high school), intermediate (trade certificate, some college) or high (4 year college degree or higher).

### Statistical Analyses

The purpose of this analysis was to evaluate associations between SES, race, and cortisol trajectories over the day. Descriptive analyses of variable distributions were conducted using standard graphing techniques and summary statistics. Bivariate associations with cortisol levels were examined using *t*-tests and ANOVA, as appropriate, and examined with scatterplots. To test hypotheses, random-effects models were estimated in a sequence of models from simple to more complex, adding suspected confounders. Random-effects models allow for robust hypothesis testing of factors thought to alter trajectories of change across a range of temporal specifications while correctly accounting for the serial correlation of repeated observations within subject (Singer & Willett, 2003; Willett, 1997). First, the average diurnal trajectory over the six cortisol values was modeled in an unrestricted time model by adding fixed, and then random effects for time and time polynomials in succession, evaluating fit using nested likelihood ratio tests. Time was calculated as an elapsed count of 30-min intervals centered on the average reported waking time (7:53 a.m.) across the sample (Time 0), allowing the intercept to be interpreted as the average waking cortisol. A random intercept for each subject was included in all models. The final model included fixed and random effects for time and time<sup>2</sup> and a fixed effect for time<sup>3</sup>. An unstructured covariance matrix was used for the random effects and an autoregressive error structure was used to address heteroscedasticity in Level 1 residuals.

For hypothesis testing, each SES measure was tested in a separate model. To test the hypothesis that average cortisol trajectories differed by SES, we added main effects and any statistically significant interaction terms with time and time polynomials to the baseline model, adjusting for the influence of age (years, grand mean centered) and sex, entered as both main effects and as all statistically significant interaction terms with time and time polynomials (Model 1). Model 2 additionally adjusted for race/ethnicity (African American vs. all others). Model 3 additionally adjusted for the stressful life event index, cigarette use, awakening time, BMI, and medical conditions.

**Results**

Subjects lost to follow-up between baseline and the fourth study visit were similar in age to those who completed the fourth visit. However, they were less wealthy (median income and assets of \$37,648 and \$60,000, respectively, vs. \$53,000 and \$101,500), and less likely to have a college degree (35% vs. 48%), or be women (62% vs. 69%). Eleven participants were excluded from analyses due to current oral corticosteroid use. Another 46 were excluded who reported alcohol intake exceeding recommended limits within 48 hr prior to cortisol sampling (>4 servings if men, >2 if women). One person was missing SES data, resulting in an analytic sample of 566. Participants contributed, on average, 5.9 cortisol observations (range 3–6), reflecting very little missing cortisol data. Also, the within-person *ln*-cortisol correlations for the four in-clinic samples

was high (partial correlation  $\rho$  ranging from .541 to .854), suggesting minimal within-person variability in response to the in-clinic cognitive testing. African Americans had lower waking and higher bedtime cortisol than others (Table 1). Also, an intermediate, but not a low level of education was associated with lower waking cortisol. African Americans were more likely than whites to be women, have socioeconomic vulnerability, and lower levels of education.

Since cortisol was *ln*-transformed, regression coefficients represent the proportion of expected change in cortisol. In all models, cortisol declined linearly after waking (Tables 2 and 3). The coefficient for time<sup>2</sup> was also negative in all models suggesting that the decline in cortisol accelerated across the day. The coefficient for time<sup>3</sup> was positive, reflecting a trajectory that flattened as the diurnal pattern approaches its evening nadir. Unadjusted diurnal cortisol trajectories within SES and race strata are depicted in Figure 1.

Table 2 presents results of models examining socioeconomic vulnerability. After adjusting for the fixed effects of age and sex and their associations with cortisol decline (Model 1), socioeconomic vulnerability was not associated with waking cortisol (main effect  $\beta = -.02, p > .05$ ) or the linear change in diurnal cortisol decline ( $\beta = -.02, p > .05$ ) (Table 2). However, socioeconomic vulnerability was associated with slower acceleration in average diurnal cortisol decline (indicated by time<sup>2</sup> interaction  $\beta = .002, p < .05$ ) and a slightly less pronounced late-day flattening of the diurnal trajectory (indicated by time<sup>3</sup> interaction  $\beta = -.0001, p < .05$ ). Also, each additional year of age was associated

**Table 1.** Selected Sample Characteristics, Baltimore Memory Study, 2001–2002 and 2009–2010

	Mean <i>ln</i> -cortisol, nmol/L (SD)		Sample characteristics by race	
	Waking	Bedtime	White (%) ( <i>n</i> = 336)	African American (%) ( <i>n</i> = 230)
Race/ethnicity				
White	3.22 (0.73)*	1.84 (0.79)*		
African American	3.00 (0.72)*	2.05 (0.79)*		
Other	3.17 (0.75)*	1.80 (0.72)*		
Socioeconomically vulnerable <sup>a</sup>				
No	3.13 (0.69)	1.89 (0.79)	296 (88)*	142 (62)*
Yes	3.15 (0.86)	2.01 (0.81)	40 (12)*	88 (38)*
Education				
High	3.20 (0.71)*	1.89 (0.80)	208 (62)*	56 (24)*
Intermediate	3.00 (0.65)*	1.94 (0.69)	58 (17)*	97 (42)*
Low	3.14 (0.83)*	1.95 (0.89)	70 (21)*	77 (33)*
Age				
Mean (SD)			66.76 (5.74)	67.14 (6.08)
56–66 years (%)	3.09 (0.76)	1.92 (0.84)	188 (56)	119 (52)
67–78 years (%)	3.18 (0.68)	1.93 (0.75)	148 (44)	111 (48)
Sex				
Men	3.19 (0.82)	1.95 (0.78)	120 (36)*	58 (25)*
Women	3.10 (0.69)	1.91 (0.80)	216 (64)*	172 (75)*

<sup>a</sup>Defined as household annual income <500% US poverty threshold and insufficient assets to remain above that threshold for more than 1 year.

\*Subgroup differences  $p < .05$ .

**Table 2.** Associations Between Socioeconomic Vulnerability and Both Waking *Ln*-Cortisol and 24-hr *Ln*-Salivary Cortisol Trajectories From Random Effects Models, Baltimore Memory Study

	Model 1 <sup>a</sup> ( <i>n</i> = 566)		Model 2 <sup>b</sup> ( <i>n</i> = 566)		Model 3 <sup>c</sup> ( <i>n</i> = 549)	
	Coefficient	<i>p</i> Value	Coefficient	<i>p</i> Value	Coefficient	<i>p</i> Value
Time	-.02	.012*	-.02	.003*	-.03	<.001*
Time <sup>2</sup>	-.00	<.001*	-.00	<.001*	-.00	<.001*
Time <sup>3</sup>	.00	<.001*	.00	<.001*	.00	<.001*
Socioeconomically vulnerable <sup>d</sup>	-.02	.738	.07	.320	.07	.322
Vulnerable × time	-.02	.073	-.02	.022*	-.03	.015*
Vulnerable × time <sup>2</sup>	.00	.013*	.00	.014*	.00	.010*
Vulnerable × time <sup>3</sup>	-.00	.029*	-.00	.033*	-.00	.022*
AA race			-.26	<.001*	-.24	<.001*
AA race × time			.01	<.001*	.01	<.001*

Notes: All models included random intercept, fixed, and random effects for time and time<sup>2</sup> and fixed effect for time<sup>3</sup> and used an unstructured covariance matrix and a first-order autoregressive error structure.

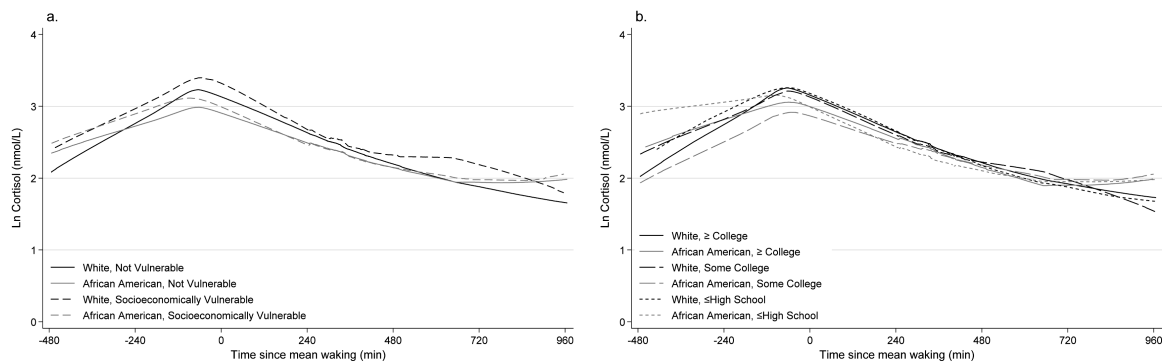
<sup>a</sup>Model adjusted for the fixed effects socioeconomic vulnerability, mean-centered age, sex, and their statistically significant interactions with time and time polynomials.

<sup>b</sup>Model additionally adjusted for the fixed effects of race/ethnicity and its interaction with time.

<sup>c</sup>Model additional adjusted for the fixed effects of the stressful life event index, cigarette use, awakening time, body mass index and its interaction with time, and medical conditions.

<sup>d</sup>Defined as household annual income and assets <500% US poverty threshold.

\**p* < .05.



**Figure 1.** Unadjusted locally weighted scatterplot smoothing graphs (LOWESS) of *ln*-transformed salivary cortisol trajectories, stratified by race/ethnicity, categorized as African Americans and all others and (a) socioeconomic vulnerability, defined as annual household income <500% US poverty threshold and insufficient assets to remain above that threshold for more than 1 year, and (b) education, categorized as high (referent, ≥college), intermediate (some college or trade school), or low (≤high school). Time was centered at sample mean waking time (7:53 a.m.).

with an almost 1% higher average waking cortisol level ( $p < .05$ ), but there were no age-based differences in the rate of diurnal cortisol decline (see Supplementary Tables 1 and 2 for complete regression model results). There were no sex differences in waking cortisol, but women had more rapid decline compared with men ( $\beta = -.02, p < .05$ ) (Supplementary Tables 1 and 2).

In Model 2, African Americans had approximately 26% lower average waking cortisol levels compared with the others and a 1% slower diurnal decline (African American race by time interaction). After adjusting for the fixed effect of African American race/ethnicity, socioeconomic vulnerability was associated with a 2% faster diurnal decline in addition to a slower acceleration in decline (time<sup>2</sup> interaction  $\beta = .002, p < .05$ ) and a slightly less pronounced

late-day flattening (time<sup>3</sup> interaction  $\beta = -.00005, p < .05$ ) (Model 2). Further adjustment by the stressful life event index, cigarette use, awakening time, BMI, and medical conditions did not alter inferences (Model 3).

Table 3 presents results examining three levels of educational attainment. After accounting for age and sex (Model 1), intermediate education (some college or trade school) was associated with approximately 17% lower average waking cortisol ( $p < .05$ ) and a 1% slower diurnal decline ( $p < .05$  for intermediate education × time), compared with high education (college or more) (Table 3). Low educational attainment was not associated with waking cortisol or rate of decline. After adjustment for African American race/ethnicity (Model 2) educational attainment was not associated with cortisol trajectories.

**Table 3.** Associations Between Educational Achievement and Both Waking *Ln*-Cortisol and 24-hr *Ln*-Salivary Cortisol Trajectories From Random Effects Models, Baltimore Memory Study

	Model 1 <sup>a</sup> (n = 566)		Model 2 <sup>b</sup> (n = 566)		Model 3 <sup>c</sup> (n = 549)	
	Coefficient	p Value	Coefficient	p Value	Coefficient	p Value
Time (30 min)	-.02	<.001*	-.02	<.001*	-.04	<.001*
Time <sup>2</sup>	-.00	<.001*	-.00	<.001*	-.00	<.001*
Time <sup>3</sup>	.00	<.001*	.00	<.001*	.00	<.001*
Intermediate education vs. high <sup>d</sup>	-.17	.009*	-.09	.214	-.05	.438
Low education vs. high <sup>d</sup>	-.11	.089	-.05	.452	-.04	.571
Intermediate education × time	.01	.016*	.00	.439	.00	.832
Low education × time	.00	.310	-.00	.920	-.00	.551
AA race/ethnicity			-.21	<.001*	-.21	.001*
AA race/ethnicity × time			.01	<.001*	.01	<.001*

Notes: All models included random intercept, and fixed and random effects for time and time<sup>2</sup> and fixed effect for time<sup>3</sup> and used an unstructured covariance matrix and a first-order autoregressive error structure.

\*p < .05.

<sup>a</sup>Model adjusted for the fixed effects of educational level, mean-centered age, sex, and their statistically significant interactions with time and time polynomials.

<sup>b</sup>Model additionally adjusted for the fixed effects of race/ethnicity and its interaction with time.

<sup>c</sup>Model additionally adjusted for the fixed effects of the stressful life event index, cigarette use, awakening time, body mass index and its interaction with time, and medical conditions.

<sup>d</sup>Categorized as high (≥college), intermediate (some college), or low (≤high school).

Finally, we conducted a planned sensitivity analyses excluding bedtime cortisol sample values (i.e., using only the waking and four in-clinic samples) to assess the impact of our assumption that previous bedtime cortisol could be used as a surrogate for bedtime cortisol on the day of the clinic visit. In those models, not presented here, inferences were largely unchanged regarding associations for waking cortisol and rate of afternoon decline for education models. For socioeconomic vulnerability models, coefficients were in the expected direction, but were not statistically significant. Separately, we adjusted for the study visit time, prior diagnosis of anxiety or depression, and both anti-anxiety or anti-depression medications, but these were trimmed from the final models because they did not alter inferences.

## Discussion

This study examined associations of SES and African American race with cortisol trajectories measured by six salivary samples in a cohort of community-dwelling middle- and older-aged adults. Based on our conceptual framework, we hypothesized that low SES and African American race contributed to greater stress exposures, resulting in earlier biological aging in these groups. In multilevel regression models of change in *ln*-transformed cortisol, African American race/ethnicity was associated with approximately 24% lower waking cortisol and 1% slower rate of afternoon cortisol decline in fully adjusted models. However, socioeconomic vulnerability was associated with 3% faster diurnal decline after accounting for race/ethnicity. These results suggest that race, more than SES, is associated with diminished late-day cortisol inhibition. As reviewed earlier,

prior work suggests this cortisol trajectory may be a marker for earlier aging in vulnerable groups.

Our results failed to corroborate other studies showing that low SES is associated with lower waking cortisol. After adjusting for age and sex, socioeconomic vulnerability was not associated with waking cortisol or afternoon cortisol decline, and intermediate, but not the lowest level of education, was associated with lower waking cortisol and slower afternoon cortisol decline. After further adjustment for race/ethnicity, socioeconomic vulnerability was associated with faster afternoon decline and no associations were noted between education and cortisol. Results for socioeconomic vulnerability are contrary to our hypothesis of a slower afternoon decline. These results suggest a convex pattern to the diurnal cortisol trajectory in socioeconomically vulnerable individuals (rather than the typical concave pattern), with faster decline and slower acceleration, as depicted in Figure 1. These results add to the growing literature suggesting complex associations between SES and diurnal cortisol trajectories, as reviewed earlier and summarized by Dowd (2009). These results also suggest that low household wealth is associated with faster, rather than slower, diurnal cortisol decline. Although faster decline may also be a marker for HPA axis dysfunction (McEwen, 1998), this pattern has not been linked with earlier aging.

Results from this study suggest a more consistent link between African American race/ethnicity and diminished late-day cortisol inhibition in middle-aged and older adults, corroborating other findings. Associations between SES and cortisol were altered after adding African American race/ethnicity to models. This adds to an ongoing discussion regarding the complex interplay of race/ethnicity and SES (Glymour, Weuve, & Chen, 2008; Kaufman, Cooper,

& McGee, 1997; Vanderweele & Robinson, 2014). Causal associations between SES, race/ethnicity and cortisol patterns are ambiguous in observational studies. To the extent that African American race/ethnicity determines individual SES, it may act as a powerful confounder of this association and should be adjusted for. On the other hand, race/ethnicity itself may act as a surrogate for unmeasured aspects of SES not well captured in our data. Self-reported race/ethnicity may capture long-term exposure to stressful experiences. This implies that race/ethnicity (Vanderweele & Robinson, 2014) and SES (Oakes & Rossi, 2003) are not separate causal processes, but are describing different aspects of cumulative adversity and vulnerability (Kawachi, Daniels, & Robinson, 2005). In this scenario, race/ethnicity is a competing measure of the exposure of interest or a surrogate for the effect of causal forces that lie between SES and HPA axis dysregulation. Under this scenario, adjustment for race/ethnicity increases rather than reduces bias. It is possible that our specification is incorrect and that Model 1 represents a better characterization of the causal effect of SES. As seen in Table 1, race and SES are highly correlated and race has been associated with salivary cortisol patterns in this study and others, as reviewed earlier. We followed previous studies that treat race as a confounder. However, questions have been raised about what exactly is being measured by self-reported race/ethnicity, because it represents the joint effect of physical phenotype, psychosocial exposures, and cultural and historical factors such as residential segregation (Vanderweele & Robinson, 2014). Some argue that race/ethnicity cannot be considered a confounder, or a causal factor at all, since it is not subject to plausible counterfactual manipulation (Kaufman & Cooper, 1999; Kaufman et al., 1997).

In addition, adjustment for race/ethnicity may produce biased estimates of the SES associations due to differential and dependent measurement error of SES by race/ethnicity. Measures of household income, wealth, and education may not perform equally well in blacks and whites (Caner & Wolff, 2004). Race/ethnicity may also be linked to measurement error of salivary cortisol. Physiologic factors, such as amount of binding proteins and medication use, can influence salivary cortisol measurement (Kudielka, Gierens, Hellhammer, Wüst, & Schlotz, 2012) and may be associated with race/ethnicity due to racial disparities in chronic diseases. However, our inferences remained unchanged after adjusting for BMI and medical conditions. Also, this study utilized a salivary cortisol assay with a very low coefficient of variation, and a wide-ranging limit of detection, providing evidence to support measurement reliability.

Salivary cortisol was measured as a biomarker for HPA axis dysregulation. However, the stress response system is complex. HPA axis dysregulation may be better captured with additional biomarkers (McEwen & Gianaros, 2010) or with additional cortisol samples, such as the 30–45 min post-awakening sample to capture cortisol awakening response. Some (Stephoe et al., 2005;

Wright & Steptoe, 2005), but not all (Hajat et al., 2010; Karlamangla et al., 2013) prior studies have found that low SES is associated with heightened cortisol awakening response, and this may account for steeper declines in the socioeconomically vulnerable group. Also, salivary and serum cortisol have a nonlinear correlation, so changes in salivary cortisol may not be proportional to changes in bioactive cortisol (Hellhammer, Wüst, & Kudielka, 2009). Cortisol samples were collected during a study visit that also included a battery of cognitive testing, which may not reflect basal cortisol patterns, although we found no evidence of an HPA axis response to cognitive testing. Also, SES was measured during the first study visit, and may not reflect changes in income at time of cortisol sampling, although household assets and education are likely stable over time. However, this study was strengthened by use of a population-based sample, multidimensional measures of SES with little missing data, and six repeated measures of cortisol, including five during the afternoon and evening hours.

In conclusion, in this study of middle aged and older adults, socioeconomic vulnerability was associated with faster diurnal decline in models accounting for age, sex, and African American race/ethnicity. African Americans had lower waking cortisol and slower afternoon decline, compared with others. This may contribute to health declines in African Americans, since lack of afternoon decline is considered to be a biological aging marker. Race/ethnicity may be a competing measure of cumulative vulnerability in this sample.

## Supplementary Material

Please visit the article online at <http://gerontologist.oxfordjournals.org/> to view supplementary material.

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L. J. S. conceived the research question, analyzed the data and led manuscript writing. T. A. G. and D. L. R. assisted with analyses and T. A. G. supervised the analyses. B. S. S. and T. A. G. coordinated the parent study, including planning instrumentation. All authors contributed to manuscript writing.

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