Check for updates

Original Article

Cortisol and Racial Health Disparities Affecting Black Men in Later Life: Evidence From MIDUS II

American Journal of Men's Health July-August 2019: 1–13 © The Author(s) 2019 Article reuse guidelines: sagepub.com/journals-permissions DOI: 10.1177/1557988319870969 journals.sagepub.com/home/jmh

\$SAGE

Julie Ober Allen, PhD, MPH D, Daphne C. Watkins, PhD², Linda Chatters, PhD^{2,3}, Arline T. Geronimus, ScD^{1,3}, and Vicki Johnson-Lawrence, MS, PhD⁴

Abstract

In the United States, Black men have poorer overall health and shorter life spans than most other racial/ethnic groups of men, largely attributable to chronic health conditions. Dysregulated patterns of daily cortisol, an indicator of hypothalamic–pituitary–adrenal (HPA) axis stress–response functioning, are linked to poor health outcomes. Questions remain regarding whether and how cortisol contributes to Black–White differences in men's health. This exploratory study compared early day changes in cortisol levels (diurnal cortisol slopes from peak to pre-lunch levels) and their associations with medical morbidity (number of chronic medical conditions) and psychological distress (Negative Affect Scale) among 695 Black and White male participants in the National Survey of Midlife in the United States (MIDUS II, 2004–2009). Black men exhibited blunted cortisol slopes relative to White men (-.15 vs. -.21, t = -2.97, p = .004). Cortisol slopes were associated with medical morbidity among Black men (b = .050, t = 3.85, p < .001), but not White men, and were unrelated to psychological distress in both groups. Findings indicate cortisol may contribute to racial health disparities among men through two pathways, including the novel finding that Black men may be more vulnerable to some negative health outcomes linked to cortisol. Further, results suggest that while cortisol may be a mechanism of physical health outcomes and disparities among older men, it may be less important for their emotional health. This study increases understanding of how race and male sex intersect to affect not only men's lived experiences but also their biological processes to contribute to racial health disparities among men in later life.

Keywords

men's health, stress, cortisol, multimorbidity, weathering, health disparities, African Americans

Received April 8, 2019; revised July 15, 2019; accepted July 27, 2019

Despite increased attention on health disparities in the United States during the 21st century, Black men continue to have shorter average life spans than most other racial/ethnic groups of men and poor health overall (Gilbert et al., 2016). The average life expectancy for Black men continues to lag behind that of White and Latino men (71.5 years compared to 76.1 and 79.1 years, respectively; National Center for Health Statistics [NCHS], 2018). Although Black men share many of the same leading causes of death as other groups of men, there are notable differences in patterns of morbidity and mortality at the intersection of race/ethnicity and age (Gilbert et al., 2016). For example, Black men's chronic conditions are, on average, more severe, poorly controlled, and likely to result in premature death

(Gilbert et al., 2016). Black men also tend to develop chronic conditions earlier in life than most other groups (Geronimus, Bound, Keene, & Hicken, 2007; Olives,

¹Population Studies Center, Institute for Social Research, University of Michigan, Ann Arbor, MI, USA

²School of Social Work, University of Michigan, Ann Arbor, MI, USA ³School of Public Health, University of Michigan, Ann Arbor, MI, USA ⁴Department of Family Medicine/Division of Public Health, College of Human Medicine, Michigan State University, Ann Arbor, MI, USA

Corresponding Author:

Julie Ober Allen, Population Studies Center, Institute for Social Research, University of Michigan, 426 Thompson Street (2411 ISR), Ann Arbor, MI 48106-1248, USA. Email: joallen@umich.edu

Creative Commons Non Commercial CC BY-NC: This article is distributed under the terms of the Creative Commons Attribution-NonCommercial 4.0 License (http://www.creativecommons.org/licenses/by-nc/4.0/) which permits non-commercial use, reproduction and distribution of the work without further permission provided the original work is attributed as specified on the SAGE and Open Access pages (https://us.sagepub.com/en-us/nam/open-access-at-sage).

Myerson, Mokdad, Murray, & Lim, 2013). Although previous studies have chronicled physical and mental health differences among subgroups of men at the intersection of race and age (Geronimus et al., 2007; Johnson-Lawrence, Griffith, & Watkins, 2013; Watkins & Johnson, 2018), few studies have investigated how these socially defined characteristics interact with biological mechanisms to affect men's health. A deeper dive into within-group differences among men is necessary to understand the patterning of health and health disparities among men. The aim of the current study was to investigate biological pathways, specifically diurnal cortisol slopes, of physical and emotional health outcomes and disparities among midlife and older U.S. Black and White men.

Biological Mechanisms of Stress and Health Disparities Affecting Black Men

A robust literature links health disparities to political, economic, historic, and social inequities (Braveman, Egerter, & Williams, 2011; Geronimus & Thompson, 2004; Williams & Mohammed, 2013). Several researchers suggest that differential exposure to sources of stress (i.e., stressors) rooted in pervasive social inequities and constrained coping opportunities cause allostatic load, premature aging, and more rapid physiological deterioration and weathering among socially marginalized groups (Braveman et al., 2011; Das, 2013; Geronimus, 1992; Geronimus, Hicken, Keene, & Bound, 2006; Geronimus et al., 2015; Juster, McEwen, & Lupien, 2010; Thorpe & Kelley-Moore, 2012). As a consequence, marginalized groups develop aging-related health conditions earlier in life than their more privileged counterparts, thereby generating health disparities that widen with increasing age (Das, 2013; Geronimus, 1992; Juster et al., 2010; Paradies, 2006; Thorpe & Kelley-Moore, 2012).

Exposure to stressors is patterned according to intersections of key socially defined and meaningful characteristics such as race and sex. The authors consider "Black" and "White" to be complex, socially constructed racial categories that reflect an array of historic, geographic, cultural, political, and economic factors that coalesce to shape individuals' lived experiences and health (Williams & Sternthal, 2010). Although racial groups are heterogeneous, collectively, U.S. Blacks experience more interpersonal and institutional racism and social marginalization than Whites, who benefit from privileges associated with affiliation with the dominant racial group that possesses disproportionate political, economic, and social power. As such, racial categories serve as proxies for shared experiences of discrimination or privilege (Geronimus & Thompson, 2004). Race and sex are also inextricably intertwined, such that they intersect to shape the number and types of stressors men experience, their psychological and biobehavioral responses, and their resultant health outcomes (Griffith, 2012; Griffith et al., 2013).

Although many stressors experienced by Black men are also experienced by other groups, research suggests that the type and intensity of stressors Black men face are specific to the combined influence of their race and sex (Ellis, Griffith, Allen, Thorpe, & Bruce, 2015). For example, Black men may experience more intense discrimination than Black women and other racial/ethnic groups, because Black men tend to be treated according to particularly harmful gendered racism, such as stereotypes that Black men are dangerous (Smith, Allen, & Danley, 2007). Psychosocial stressors linked to poor health outcomes for Black men include: (a) negative, narrow stereotypes of Black males; (b) interpersonal and structural discrimination; (c) disproportionate poverty, unemployment, and underemployment; (d) residential segregation in investment-poor, underserved, and decaying neighborhoods; (e) inadequate health care; (f) negative interactions with the criminal justice system; and (g) barriers to fulfilling valued masculine roles (Chae et al., 2014; Gilbert et al., 2016; Griffith et al., 2013; Griffith, Gunter, & Allen, 2011; Williams & Mohammed, 2013). Exposure to the substantial stressors many Black men face may accelerate biological aging and physiological deterioration, resulting in the disproportionate burden of morbidity and early mortality documented among Black men. Assuming an approach that considers the combined influences of race and male sex may facilitate identification of specific mechanisms contributing to health disparities over the life course that differentiate Black men from other racial/ethnic groups of men.

As an indicator of premature aging and physiological weathering implicated in the etiology of many different chronic conditions as well as early mortality, diurnal cortisol slopes may be a valuable tool for investigating pathways contributing to the unique patterning of health among Black men (Juster et al., 2010; Samuel, Roth, Schwartz, Thorpe, & Glass, 2018; van den Beld et al., 2018). A growing body of research indicates that diurnal cortisol slopes, which capture one component of the daily cortisol rhythm, are sensitive to exposure to psychosocial and environmental stressors (Adam et al., 2015; Dowd, Simanek, & Aiello, 2009; Karb, Elliott, Dowd, & Morenoff, 2012; Lam, Shields, Trainor, Slavich, & Yonelinas, 2018). When the biological systems involved in the stress response are overtaxed due to prolonged exposure to toxic stressors, the physiological stress response becomes dysregulated. Under these conditions, the hypothalamic-pituitary-adrenal (HPA) axis fails to generate robust surges of cortisol and other stress hormones when necessary or as part of naturally occurring daily fluctuations. Daily cortisol rhythms become blunted. Other biological systems deteriorate, leading to

accelerated aging, chronic disease, and premature mortality (Adam et al., 2017; Juster et al., 2010; McEwen & Gianaros, 2010). A meta-analysis by Adam et al. (2017) linked blunted cortisol slopes to a variety of poor physical and mental health outcomes in diverse samples, though few studies examined findings specifically for men and none compared men of different racial/ethnic groups.

There are reasons to believe diurnal cortisol slopes differ for Black men compared to other groups and that these slopes change with age. Researchers have consistently documented more blunted cortisol slopes among Blacks relative to Whites in mixed sex samples, though males have been underrepresented in these studies (25%-48%; Cohen et al., 2006; DeSantis et al 2007; DeSantis, Adam, Hawkley, Kudielka, & Cacioppo, 2015; Hajat et al., 2010; Karlamangla, Friedman, Seeman, Stawksi, & Almeida, 2013; Samuel et al., 2018). These findings remained robust even after accounting for confounding factors such as socioeconomic status and health behaviors (Cohen et al., 2006; Hajat et al., 2010; Karlamangla et al., 2013; Samuel et al., 2018). Some studies have observed blunted cortisol slopes among men when compared to women (DeSantis et al., 2007; Dmitrieva, Almeida, Dmitrieva, Loken, & Pieper, 2013), though sex differences have been less consistently reported. Researchers have observed that cortisol slopes are generally more blunted among older adults relative to younger adults (Gaffey, Bergeman, Clark, & Wirth, 2016). Few studies have examined cortisol slopes exclusively among men, and the authors are not aware of any studies that have compared cortisol slopes at the intersection of race, male sex, and age (Adam et al., 2017). Furthermore, questions remain regarding whether cortisol slopes are indicators of comparable health risks for all, or whether race moderates these relationships, thereby indicating that some subgroups are at a higher risk for poor health outcomes associated with blunted cortisol slopes than others. The purpose of the current exploratory study was to assess whether diurnal cortisol slopes provide insight into the biological pathways underlying the disproportionate burden of poor health outcomes among Black men.

The Current Study

This study explored racial differences in diurnal cortisol slopes and their relationships to health among midlife and older U.S. men in order to better understand possible biological pathways contributing to the disproportionate burden of poor health among Black men. First, the authors characterized and compared diurnal cortisol slopes among Black and White men, and then among Black and White men in different age groups. It was hypothesized that Black men would exhibit more blunted diurnal cortisol slopes than same-aged White men, consistent with the racial disparities in diurnal cortisol slopes documented in

mixed sex and female samples. Next, whether race moderated relationships between diurnal cortisol slopes and two general indicators of physical and emotional health (i.e., medical morbidity and psychological distress) was examined to determine if Black men were also more vulnerable to the negative health outcomes associated with blunted diurnal cortisol slopes. It was anticipated that diurnal cortisol slopes would be more strongly associated with medical morbidity and psychological distress among Black men when compared to their White counterparts.

Methods

This study used linked participant data from the second wave of the National Survey of Midlife in the United States (MIDUS II) and the National Study of Daily Experiences (NSDE II), which is a MIDUS supplemental project. MIDUS II (2004–2006) consisted of an interview and self-administered questionnaire that assessed a range of sociodemographic, psychosocial, and health topics. NSDE II (2004–2009) was an in-depth study of daily stressors, cortisol biomarkers collected from saliva, and health indicators (Almeida, McGonagle, & King, 2009). NSDE II data were collected an average of 21 months after participants provided data for MIDUS II.

MIDUS originated with a stratified probability sampling design. MIDUS II included 5,555 English-speaking, community-residing adults from the contiguous U.S. who were between 35 and 85 years old (Radler & Ryff, 2010). A random subsample of MIDUS II participants was recruited for NSDE II. A total of 2,022 participants provided data for NSDE II (78% response rate), and 1,735 (85.8%) of these provided saliva samples (Almeida et al., 2009). Institutional review board (IRB) review and participant informed consent were obtained for all study components. For the current study, the analytic sample was limited to the 59 Black and 636 White men who completed the MIDUS II interview and questionnaire and provided sufficient NSDE II salivary cortisol data for calculating diurnal cortisol slopes. This sample was determined to be sufficient for detecting racial differences in men's diurnal cortisol slopes with effect sizes as small as .012, using linear multiple regression with 80% power, a .05 Type I error, and 10 predictor variables; for reference, studies with mixed sex samples have documented effect sizes of race on similar cortisol measures at or above .040 (Karlamangla et al., 2013). When compared to the analytic sample, the 130 excluded men (i.e., 38 nonparticipants in MIDUS II questionnaire, 104 nonparticipants in saliva collection, 2 with insufficient salivary cortisol data) were more likely to be Black (8.5% vs. 16.9%, p =.006) but otherwise comparable to the sample in age, socioeconomic status, medical morbidity, psychological distress, and other characteristics.

Cortisol Data Collection

Cortisol was collected in saliva. Participants used Salivette kits (Sarstedt, Rommelsdorft, Germany) to collect four saliva samples each day for 4 consecutive days. Participants collected samples upon waking (T1), 30 min after waking (T2), before lunch (T3), and at bedtime (T4). Salivary cortisol samples were collected using standard procedures and protocols used in large-scale epidemiological research (Adam & Kumari, 2009); details are described elsewhere (Almeida et al., 2009). Participants recorded sample collection times in written logs and nightly telephone interviews (correlation > .90; Almeida et al., 2009). Luminescence immunoassays measured raw salivary cortisol concentrations in the samples; intra and interassay coefficients were <5%.

Measures

Diurnal cortisol slopes. The diurnal cortisol slopes measure captured the early-day rapid declining cortisol slope from 30 min after waking (T2), when cortisol levels typically peak, to before lunch (T3). Diurnal cortisol slopes have demonstrated good predictive validity for a broad range of mental and physical health outcomes (Adam et al., 2017), and early-day declining slopes demonstrate significant Black-White disparities (Hajat et al., 2010). Although cortisol levels and patterns fluctuate daily, diurnal cortisol slopes are more reliable than other commonly used cortisol measures, especially when data from several days are averaged to increase stability (Wang et al., 2014). Accordingly, slopes from the 4 days cortisol were collected and were averaged for the current study. Raw cortisol values were natural log transformed to adjust for skew, consistent with previous cortisol research, prior to slope calculations (Adam & Kumari, 2009). More blunted (i.e., horizontal, closer to 0) diurnal cortisol slopes reflect less robust dynamic range, which is an indicator of HPAaxis dysregulation (Adam et al., 2017).

Health indicators. Two health variables theoretically and empirically linked to diurnal cortisol slopes were examined: (a) medical morbidity, which reflected number of chronic physical health conditions representing major sources of morbidity, mortality, and racial health disparities among older U.S. men (Gilbert et al., 2016; National Center for Health Statistics [NCHS], 2017; Sturmberg, Bennett, Martin, & Picard, 2017); and (b) self-reported psychological distress (Adam et al., 2017; Paradies, 2006). Medical morbidity indicated how many conditions participants reported from a list of seven: hypertension/high blood pressure, heart disease/heart attack, a stroke, cancer, diabetes, chronic lower respiratory diseases including asthma, and arthritis/bone/joint diseases. Psychological

distress was assessed with the 14-item Negative Affect Scale, which captures self-reported, nonspecific psychological distress (Mroczek, & Kolarz, 1998; Piazza, Charles, Stawski, & Almeida, 2013). The scale assessed how often participants felt a range of moods in the past 30 days, including moods theorized to capture expressions of distress specifically among men (Martin, Neighbors, & Griffith, 2013). Moods assessed were nervous, restless or fidgety, hopeless, that everything was an effort, worthless, so sad nothing could cheer you up, afraid, jittery, irritable, ashamed, upset, lonely, angry, and upset. Responses were reverse coded and averaged, so higher scores indicated more psychological distress. The scale has demonstrated good internal validity and reliability in other studies (Piazza et al., 2013) and good reliability for the Black and White men in this sample (Cronbach's α .95 and .90, respectively).

Other variables. Models included variables for age, selfreported race, and highest level of education attainedthree sociodemographic characteristics consistently associated with diurnal cortisol slopes (Gaffey et al., 2016; Samuel et al., 2018). Four variables previously identified as affecting cortisol were also included—smoking, atypical sleep schedule, medications, and not adhering to salivary cortisol collection protocols (Adam & Kumari, 2009; Direk, Newson, Hofman, Kirschbaum, & Tiemeier, 2011; Granger, Hibel, Fortunato, & Kapelewski, 2009; Kumari et al., 2009). There were no missing data on these variables. Smoker indicated reported cigarette smoking during NSDE II. Atypical sleep schedule indicated waking before 4 a.m. or after 11 a.m. and/or being awake >20 hrs or <12 hrs during NSDE II salivary cortisol collection (Dmitrieva et al., 2013). Medications indicated use of medications that can affect cortisol (i.e., containing steroids or hormones; treatments for allergies, depression, or anxiety) during NSDE II. Protocol nonadherent indicated T3 cortisol values >10 nanomoles/liter higher than corresponding T2 values, signaling protocol nonadherence (i.e., eating prior to T3 sample collection).

Data Analysis

Exploratory analyses were conducted to identify confounding variables; examine missing data; explore variable distributions and conduct transformations, as needed; and ensure that the most psychometrically sound scales were used. Two-tailed t, chi-squared, and Mann—Whitney U tests were used for basic descriptive statistics and to compare Black and White men in the sample.

Since cortisol data were collected in NSDE II, after the health indicator measures were collected in MIDUS II, diurnal cortisol slopes were the dependent measure in all multivariate analyses. Findings were interpreted as

Table 1. Participant Sociodemographic and Health Characteristics, by Race.

	Black	men	Wh	ite men	
Characteristic	%	M (SD)	%	M (SD)	Þ
Sociodemographic					
Age (years)		54.3 (10.00)		57.2 (12.06)	.040
Married/cohabitating	62.7		83.0		<.001
Educational attainment					
No high school diploma/GED	13.6		4.7		.011
High school diploma/GED	66.1		46.9		.006
4-year college degree	10.2		30.6		<.001
Graduate/professional degree	10.2		17.7		.152
Employed	57.6		68.4		.109
Supervisory role, current/last job	31.6		51.9		.004
Decision autonomy, current/last job ^a		19.4 (6.27)		23.0 (4.86)	.001
Annual household income, median		\$39,000		\$65,250	<.001
Health					
# medical conditions		1.46 (1.15)		1.15 (1.18)	.058
Psychological distress ^b		1.73 (.81)		1.51 (.44)	.039
Health insurance	83.3	. ,	94.4	• •	.083
Total N	59 (8.5%)			636 (91.5%)	

Note. GED = General Educational Development.

^aadapted Decision Authority Subscale from Karasek & Theorell, (1990). Scores range from 5 (none) to 30 (a great deal). ^boriginal Negative Affect Scale from Mroczek & Kolarz (1998). Scores range from 1 (none of the time) to 5 (all of the time).

cross-sectional rather than prospective, however, because measures captured constructs expected to be stable over the time period between data collections.

Ordinary least squares hierarchical regression was used to examine racial similarities and differences in factors related to men's diurnal cortisol slopes. Diurnal cortisol slopes were regressed on the health indicators, with other variables added to the model in successive blocks. The moderating role of race was then tested by adding interaction terms created by taking the product of the race indicator variable and health indicators significant at the α < .05 level. Significant interactions were graphed, and simple slopes tests were used to identify which groups of men exhibited significant relationships, by conducting t tests of the slopes divided by their standard errors (Aiken & West, 1991). Mann-Whitney U median tests were used to identify health indicator levels at which Black and White men's diurnal cortisol slopes differed.

Sensitivity analyses were conducted to determine if findings differed with alternative techniques: (a) multiple imputation of missing data (Markov chain Monte Carlo method); (b) multilevel modeling to ascertain the influence of day-to-day variations in individual cortisol patterns; (c) race-stratified models; and (d) models excluding participants who used medications, had atypical sleep schedules, or were protocol nonadherent. Presented analyses were conducted with SPSS 24 (IBM Corp., Armonk, NY).

Results

Sample Characteristics

Participant sociodemographic and health characteristics by race are reported in Table 1. The sample included 695 men and was 8.5% Black (n=59) and 91.5% White (n=636). Average age was in the mid-50s. Black participants were slightly younger than White participants, were less likely to be married or cohabitating with romantic partners, and had lower socioeconomic status across multiple indicators including education, occupational prestige, and household income. Black and White men had comparable medical morbidity, though additional analyses revealed that more Black men reported two of the seven individual conditions assessed: hypertension (55.9% vs. 36.7%, $\chi^2=8.42$, p=.005) and diabetes (23.7% vs. 10.5%, $\chi^2=9.16$, p=.005). Black men reported more psychological distress than White men.

Diurnal Cortisol Slopes Among Black and White Men, and Black and White Men by Age Group

The average raw cortisol levels for Black and White men at four daily time points are reported in Table 2 (top section). These diurnal cortisol patterns are graphed in Figure 1. Although White men's average cortisol levels were higher than Black men's at all time points, the only

Table 2. Cortisol Data and Diurnal Cortisol Slopes, by Race.

		Black men $(N = 59)$		White men $(N = 636)$	
Variables	%	M (SD)	%	M (SD)	Þ
Cortisol levels, raw (nanomole	es/liter) ^a				
Upon waking (T1)	·	11.31 (6.41)		20.05 (40.64)	.100
30 min after waking (T2)		14.48 (9.33)		25.67 (38.73)	.027
Before lunch (T3)		6.86 (3.11)		9.01 (10.96)	.135
Bedtime (T4)		4.84 (3.21)		7.37 (35.29)	.582
Diurnal cortisol slopes					
Full sample		15 (.15)		2I (.II)	.004
Midlife (34–54 years)		16 (.15)		2I (.I2)	.088
		(n = 29, 49.2%)		(n = 281, 44.2%)	
Later life (55+ years)		14 (.15)		2I (.II)	.021
		(n = 30, 50.8%)		(n = 355, 55.8%)	
Factors that affect cortisol					
Smoker	32.2		15.4		.003
Atypical sleep schedule	28.8		8.8		<.001
Medications affecting cortisol	11.9		27.9		.008
Protocol nonadherent	6.8		4.9		.528

Note. ^aAveraged over all collection days.

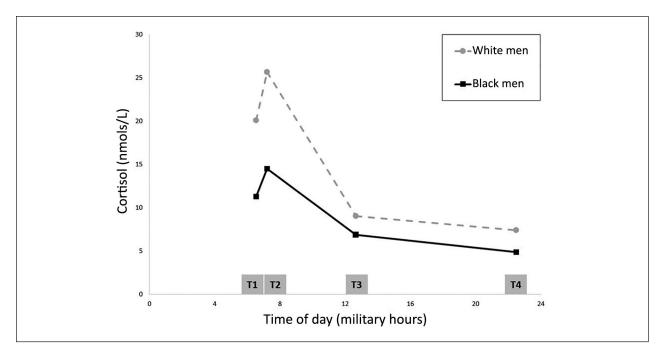


Figure 1. Diurnal cortisol patterns for Black and White men.

statistically detectable difference was 30 min after waking (T2). Further, the T2 to T3 declining cortisol slope for Black men, depicted in Figure 1, appeared blunted relative to the slope for White men, indicating HPA-axis dysregulation. Statistical comparisons of these diurnal cortisol slopes confirmed racial differences (Table 2, middle).

Black men had more blunted diurnal cortisol slopes than White men in the full sample (-.15 vs. -.21) and when divided by age group. Despite small cell sizes (~30 Black men per age group), significant racial differences between later life (55 years old and older) Black and White men were detected, while racial differences between midlife

Table 3. Factors Linked to Men's Diurnal Cortisol Slopes.

	_	Model I			Model 2			Model 3			Model 4			Model 5	
Variables b		SE	ф	9	SE	ф	q	SE	ф	9	SE	ф	9	SE	ф
Key health indicator															
# medical conditions	.012	.00	.007	10.	.004	.017	010	.004	.026	010	.004	.017	.007	.004	.133
Psychological distress	.002	010.	.824	00	010.	.921	003	010:	.791	010	010.	.300	014	010.	.149
Other															
Age	000	00	909.	000	00 	.813	000	00	.739	00	\ 00.	179	00	\ 00.	.227
Race (Black)				.057	910.	\ .001	.052	910.	100.	.049	910.	.002	012	.025	.635
Educational attainment							012	.005	.025	008	.005	.120	009	.005	180:
Smoker										.015	.012	.212	.015	.012	.204
Atypical sleep schedule										600	.014	.512	010	.014	.493
Medications										600.	010	.351	010	010	315
Protocol nonadherent										.162	.020	\ .001	091.	.020	\ \ .001
Interaction													.043	.013	.002
Race × # medical conditions	ons														
Intercept	208	.032	. 00.	214	.031	\ .001	177	.036	\ \ .001	165	.035	\ .001	156	.034	\ \ .001
\mathbf{R}^2		.013			.030			.037			.127			140	
$F\Delta R^2(\rho)$		2.93 (.033)	3		12.57 (<.001)	001)		5.04 (.025)	25)		17.65 (<.001)	(100.		10.05 (.002)	12)

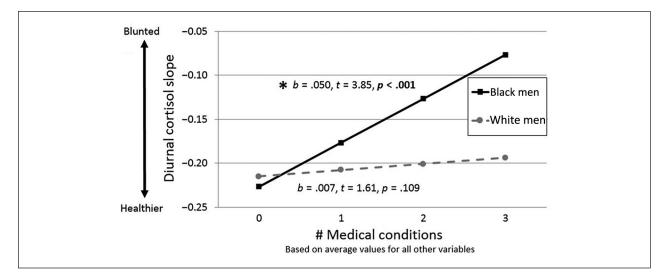


Figure 2. Relationships between diurnal cortisol slopes and number of medical conditions for Black and White men.

(34–54 years old) men trended toward significant. Multivariate analyses confirmed slope differences by race after accounting for health, sociodemographic, and other considerations (Table 3, Models 2–4), but did not detect interactions of race and age in predicting men's diurnal cortisol slopes (not shown). Among factors affecting cortisol (Table 2, bottom), more Black men than White men smoked and reported atypical sleep schedules, but fewer used medications.

Factors Linked to Black and White Men's Diurnal Cortisol Slopes

The relationships between diurnal cortisol slopes and the health indicators, controlling for age, are presented in Table 3, Model 1. Greater medical morbidity was associated with more blunted diurnal cortisol slopes; psychological distress and age were not. In Model 2, although the race indicator variable slightly reduced the medical morbidity effect size, both variables accounted for variation in men's cortisol slopes. In Model 3, the relationship between race and men's cortisol slopes was still significant, but slightly diminished in strength after adding educational attainment to the model. Education demonstrated a small, independent relationship with men's cortisol slopes. In Model 4, other variables identified as important in previous cortisol research were added; protocol nonadherent was the only one of these associated with men's cortisol slopes. This group of variables suppressed the education effect size, likely due to education's correlations with smoking (r = -.19, p < .001) and atypical sleep schedule (r = -.15, p < .001). Accordingly, education could not substantially mediate the relationship between race and men's cortisol slopes. Interaction tests (not shown) revealed that education also did not moderate the relationship between race and men's cortisol slopes. Medical morbidity and race remained predictive of men's cortisol slopes. In Model 5, the significant interaction term indicates that race moderated the relationship between medical morbidity and men's cortisol slopes.

To explore this interaction, Figure 2 depicts racial differences in relationships between men's diurnal cortisol slopes and medical morbidity. All other variables were held at the sample average, which provides a conservative depiction of race-based differences given Black-White differences in the sample's characteristics. Simple slopes tests (reported in Figure 2) showed that medical morbidity was associated with cortisol slopes for Black men but not White men. To assess the medical morbidity level at which Black and White men's cortisol slopes differed, the sample was divided into three roughly equal subgroups of men that mapped onto zero, one, and two or more medical conditions. Despite limited statistical power, it was confirmed that Black men with two or more medical conditions exhibited more blunted cortisol slopes than White men with two or more conditions (-.13 vs. -.20, U = 3,908, p = .001). A similar trend was documented when comparing Black and White men with one condition (-.17 vs. -.20, U = 2,244, p = .081). Black and White men with no medical conditions had comparable cortisol slopes.

Post hoc analyses examined the contributions of individual conditions within the medical morbidity measure to the findings (not shown). In the full sample, men's cortisol slopes showed some association with cancer and diabetes, but no other individual conditions. Hypertension and chronic lower respiratory disease were the only individual conditions that interacted with race: both were

associated with more blunted cortisol slopes among Black men, but not White men. Therefore, these conditions played a larger role than others in the race-based differences detected in the relationships between medical morbidity and men's cortisol slopes. Adding the medical morbidity variable to the models testing individual conditions suppressed cancer and diabetes main effects, slightly diminished the still significant interactions, and was a stronger determinant of men's cortisol slopes disparities than any individual conditions. All sensitivity analyses were consistent with presented findings.

Discussion

This exploratory study identified two physiological pathways that may account, in part, for racial health disparities affecting Black men. First, midlife and older Black men exhibited more blunted diurnal cortisol slopes, which have been associated with various poor physical and mental health outcomes and early mortality in numerous other studies (Adam et al., 2017), than White men in the same age groups. Second, Black men appeared to be more vulnerable to some negative health outcomes linked to blunted diurnal cortisol slopes, as Black men exhibited stronger relationships between blunted diurnal cortisol slopes and medical morbidity than White men. On the other hand, men's cortisol slopes were unrelated to their psychological distress, suggesting that while diurnal cortisol dysregulation may be a pathway of physical health and health disparities among older men, it may not meaningfully contribute to men's emotional health.

We documented racial disparities in diurnal cortisol slopes among midlife and older U.S. men, in which Black men exhibited more blunted diurnal cortisol slopes indicative of impaired functioning of biological systems involved in the stress response when compared to White men. This was consistent with findings from previous studies on Black-White disparities in diurnal cortisol slopes among mixed sex and female samples (Cohen et al., 2006; DeSantis et al., 2007, 2015; Hajat et al., 2010; Karlamangla et al., 2013; Samuel et al., 2018). One difference, however, was the finding in the current study that the average bedtime cortisol level (T4) of Black men was lower, though not statistically different, than the bedtime cortisol level of White men. Previous studies with mixed sex and all female samples (Cohen et al., 2006; DeSantis et al., 2007, 2015; Hajat et al., 2010; Karlamangla et al., 2013; Samuel et al., 2018) and one study comparing diurnal cortisol patterns among midlife (ages 32-45 years) Black and White males and females (Cohen et al., 2006) have documented higher bedtime cortisol levels among Blacks than Whites. More research is needed with larger subsamples of Black men and nationally representative samples to determine whether the intersectional

approach adopted in the current study illuminated actual differences by race, sex, and possibly age that were masked in previous studies. For example, it may be that Black women and younger Black men and women have higher bedtime cortisol levels than same-aged Whites, while older Black men do not and this distinction gets lost when data are aggregated.

The finding that men's diurnal cortisol slopes varied in a manner consistent with membership in a marginalized racial group suggests the utility of diurnal cortisol biomarkers in research to track, understand, and identify evidence-based strategies to reduce and eliminate racial health disparities. The linkages this study documented between race and men's diurnal cortisol slopes are likely due to social and contextual factors patterned by and/or linked to race (e.g., socioeconomic status, racial segregation) rather than essential biological differences (Williams & Sternthal, 2010). Racial categories are poor indicators of genetic diversity (Yudell, Roberts, DeSalle, & Tishkoff, 2016). As a proxy for exposure to race-related adversity and privilege, however, race continues to be an enduring social determinant of health in the contemporary U.S. (Geronimus & Thompson, 2004). Race intersects with sex, age group, and other sociodemographic characteristics to shape lived experiences, exposure to stressors, coping, and, ultimately, health (Ellis et al., 2015; Gilbert et al., 2016; Griffith et al., 2013; Jackson, Williams, & VanderWeele, 2016). For example, Black men nationwide and in the current study are more likely than White men to be single and have low socioeconomic status. The current study and others have reported that racial disparities in health, including diurnal cortisol slopes, persist after accounting for these sociodemographic differences (Jackson et al., 2016; Samuel et al., 2018). These differences may result in many Black men being multiply marginalized and having less access to buffering resources, which, in turn, exacerbate existing stressors, generate additional stressors, and create barriers to effective coping.

The Black—White disparities and age-related patterns of men's diurnal cortisol slopes documented in this study support the growing body of evidence that stress and coping processes, and resultant impaired functioning of biological systems involved in the stress response, are key mechanisms through which social determinants of health such as race and gender generate and reproduce health disparities over the life span. This study found suggestive evidence that racial disparities may be more pronounced among older men than younger men, though more research with larger subsamples of Black men and nationally representative samples is needed to confirm these findings. If the findings are extrapolated to reflect trajectories of men's diurnal cortisol slopes over the life course, the authors would speculate that, on average, White men

exhibit relatively consistent diurnal cortisol slopes from midlife (the younger age boundary of this dataset) on, while the diurnal cortisol slopes of Black men become increasingly blunted in later life. Longitudinal studies are needed to test this. It is particularly noteworthy that given high mortality rates of young to middle adult U.S. Black males relative to females and their counterparts in other racial/ethnic groups (Gilbert et al., 2016), study findings may reflect a survival bias of healthier Black men who lived into the older age range of the MIDUS II sample (Gaffey et al., 2016). Accordingly, the current study may underestimate racial disparities in diurnal cortisol slopes between older Black and White men.

A novel contribution of the current study is the documentation of a relationship between diurnal cortisol slopes and medical morbidity, an indicator of multimorbidity, among Black men but not White men. This may reflect the differing characteristics of Black and White men's individual and multiple medical conditions in several ways. First, if Black men in the study sample experienced conditions considered more dangerous (e.g., heart disease or stroke) or more consistently linked to blunted diurnal cortisol slopes (e.g., cancer; Adam et al., 2017) than White men, that would help to explain the racial differences detected; however, this was not the case. Instead, findings were driven by differences related to two less life-threatening but long-term chronic conditions: hypertension and chronic lower respiratory diseases. These conditions may function as greater stressors on Black men's bodies and for a longer duration of their lives than the same conditions for White men, as Black males tend to develop hypertension and asthma (the most common chronic lower respiratory disease) at younger ages than White males (Geronimus et al., 2007; Hill, Graham, & Divgi, 2011), and their conditions are often more severe and poorly controlled (Hill et al., 2011; Olives et al., 2013). As a result, these conditions may further blunt Black men's diurnal cortisol slopes over time through reciprocal relationships. Second, study findings also allude to differences in the origins of Black and White men's medical conditions. Medical morbidity was a stronger determinant of disparities in men's diurnal cortisol slopes than individual conditions. If number of medical conditions is considered to be a proxy for the deterioration of multiple biological systems (Sturmberg et al., 2017), findings from the current study suggest that Black men develop medical conditions and multimorbidity as a result of weathering, which is linked to fundamental social and structural inequities that function as pervasive stressors in Black men's lives. White men, on the other hand, do not and instead develop medical conditions due to other causes, such as aging. Future research is needed to foster a deeper and more nuanced understanding of the ramifications of the documented disparity for Black men's health and for the development of effective, race-specific prevention and treatment strategies.

It is notable that diurnal cortisol slopes were not associated with psychological distress in this study. Psychological distress and indicators of dysregulation within biological systems involved in the stress response, such as blunted diurnal cortisol slopes, represent different types of negative proximal outcomes of stress and coping processes; for this reason, they are often assumed to be closely related. Studies examining relationships between these constructs, however, have generated mixed findings (Chida & Steptoe, 2009; Staufenbiel, Penninx, Spijker, Elzinga, & van Rossum, 2013). Questions remain about whether these inconsistencies reflect distinct psychological and physiological pathways linking stress processes and health, measurement validity issues (e.g., psychological distress is susceptible to self-report bias and may be especially underreported among men given gender norms discouraging emotionality), or something else. Despite this ambiguity, this study clearly demonstrates that diurnal cortisol slopes are a valuable tool for better understanding morbidity and mortality and health disparities among men in general, and particularly when considering the ramifications of social inequities for health. Therefore, expansion of biomarker collection in U.S. survey research tracking population-level health may be beneficial for additional investigation of mechanisms contributing to health and health disparities, while also potentially serving as indicators used for early detection of increased morbidity and mortality risk.

Limitations

Findings should be interpreted in light of limitations. First, sample size and characteristics constrained analyses and the generalizability of the findings. Comparisons to men of other races/ethnicities could not be conducted because of insufficient subsample sizes in the dataset. The modest subsample of Black men precluded more complex age group-specific analyses and identifying factors related to Black men's diurnal cortisol slopes with small effect sizes. However, this meant that any statistically significant parameters in analyses represented sizable racial differences or effect sizes. Findings were not weighted to be nationally representative given the variety of sampling strategies used in MIDUS II and NSDE II. Collecting data for assessing diurnal cortisol patterns among larger subsamples of Black men and nationally representative samples is essential to confirm and build on the findings from the current study. Second, the medical morbidity measure was self-reported and reflected clinically diagnosed conditions. The validity and reliability of these types of measures can vary by race, age, and education (Leikauf & Federman, 2009; Zajacova &

Dowd, 2011), in part, because they do not capture other indicators of health such as undiagnosed and preclinical conditions, condition severity, or condition duration. Third, lifestyle and measurement factors can bias diurnal cortisol slopes, which demonstrate only moderate stability (Ross, Murphy, Adam, Chen, & Miller, 2014). To mitigate these shortcomings, the authors controlled for several confounding factors, averaged slopes over 4 days, and conducted a series of sensitivity analyses. Finally, changes in diurnal cortisol slopes over the life course could not be examined and causality between diurnal cortisol slopes and medical morbidity could not be ascertained.

Conclusions

This exploratory study enriches the literature on stressrelated biological mechanisms underlying racial health disparities affecting Black men and alludes to the potential advances in understanding men's health and health disparities that may result from expanded biomarker research, particularly with larger, nationally representative samples. This may be the first study to document more blunted diurnal cortisol slopes among older Black men than their White counterparts. Another novel contribution was the linkage identified between diurnal cortisol slopes and medical morbidity among Black, but not White, men, suggesting that older Black men are more vulnerable to poor health outcomes associated with blunted cortisol slopes than comparable White men. Individual medical conditions and multimorbidity experienced by Black men likely have inherently different characteristics and origins than the same health issues for White men, which may have harmful effects on their diurnal cortisol slopes and overall health. Collectively, findings provide support for the growing body of evidence that the disproportionate burden of medical morbidity and mortality among Black men may result from physiological weathering rooted in living within a racially stratified society (Geronimus et al., 2006, 2015).

Declaration of Conflicting Interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Funding

The author(s) disclosed receipt of the following financial support for the research, authorship, and/or publication of this article: This work was supported by a fellowship from the University of Michigan Rackham Graduate School for Graduate Studies; and a grant from the National Institute on Aging at the National Institutes of Health to the Population Studies Center at the University of Michigan [T32-AG000221]. Data used for

this research were provided by the longitudinal study titled "Midlife in the United States" (MIDUS), managed by the Institute on Aging at the University of Wisconsin and supported by a grant from the National Institute on Aging at the National Institutes of Health [P01-AG020166].

ORCID iD

Julie Ober Allen https://orcid.org/0000-0002-3969-8130

References

- Adam, E. K., Heissel, J. A., Zeiders, K. H., Richeson, J. A., Ross, E. C., Ehrlich, K. B., . . . Eccles, J. C. (2015). Developmental histories of perceived racial discrimination and diurnal cortisol profiles in adulthood: A 20-year prospective study. *Psychoneuroendocrinology*, 62, 279–291. doi:10.1016/j.psyneuen.2015.08.018
- Adam, E. K., & Kumari, M. (2009). Assessing salivary cortisol in large-scale, epidemiological research, *Psychoneuroendocrinology*, *34*, 1423–1436. doi:10.1016/j. psyneuen.2009.06.011
- Adam, E. K., Quinn, M. E., Tavernier, R., McQuillan, M. T., Dahlke, K. A., & Gilbert, K. E. (2017). Diurnal cortisol slopes and mental and physical health outcomes: A systematic review and meta-analysis. *Psychoneuroendocrinology*, 83, 25–41. doi:10.1016/j.psyneuen.2017.05.018
- Aiken, L. S., & West, S. G. (1991). Multiple regression: Testing and interpreting interactions. Thousand Oaks, CA: Sage.
- Almeida, D. M., McGonagle, K. A., & King, H. (2009).
 Assessing daily stress processes in social surveys by combining stressor exposure and salivary control. *Biodemography and Social Biology*, 55, 219–237. doi:10.1080/19485560903382338
- Braveman, P., Egerter, S., & Williams, D. R. (2011). The social determinants of health: Coming of age. *Annual Review of Public Health*, 32, 381–398. doi:10.1146/annurev-publ-health-031210–101218
- Chae, D. H., Nuru-Jeter, A. M., Adler, N. E., Brody, G. H., Lin, J., Blackburn, E. H., & Epel, E. S. (2014). Discrimination, racial bias, and telomere length in African-American men. *American Journal of Preventive Medicine*, 46(2), 103–111. doi:10.1016/j.amepre.2013.10.020
- Chida, Y., & Steptoe, A. (2009). Cortisol awakening response and psychosocial factors: A systematic review and meta-analysis. *Biological Psychology*, 80, 265–278. doi:10.1016/j.biopsycho.2008.10.004
- Cohen, S., Schwartz, J. E., Epel, E., Kirschbaum, C., Sidney, S., & Seeman, T. (2006). Socioeconomic status, race, and diurnal cortisol decline in the Coronary Artery Risk Development in Young Adults (CARDIA) Study. *Psychosomatic Medicine*, 68(1), 41–50. doi:10.1097/01. psy.0000195967.51768.ea
- Das, A. (2013). How does race get "under the skin"?: Inflammation, weathering, and metabolic problems in late life. Social Science & Medicine, 77, 75–83. doi:10.1016/j. socscimed.2012.11.007
- DeSantis, A. S., Adam, E. K., Doane, L. D., Mineka, S., Zinbarg, R. E., & Craske, M. G. (2007). Racial/ethnic differences in cortisol diurnal rhythms in a community sample

- of adolescents. *Journal of Adolescent Health*, 41(1), 3–13. doi:10.1016/j.jadohealth.2007.03.006
- DeSantis, A. S., Adam, E. K., Hawkley, L. C., Kudielka, B. M., & Cacioppo, J. T. (2015). Racial and ethnic differences in diurnal cortisol rhythms: Are they consistent over time? *Psychosomatic Medicine*, 77(1), 6–15. doi:10.1097/PSY.0000000000000131
- Direk, N., Newson, R. S., Hofman, A., Kirschbaum, C., & Tiemeier, H. (2011). Short and long-term effects of smoking on cortisol in older adults. *International Journal of Psychophysiology*, 80(2), 157–160. doi:10.1016/j.ijpsycho.2011.02.007
- Dmitrieva, N. O., Almeida, D. M., Dmitrieva, J., Loken, E., & Pieper, C. F. (2013). A day-centered approach to modeling cortisol: Diurnal cortisol profiles and their associations among U.S. adults. *Psychoneuroendocrinology*, 38, 2354–2365. doi:10.1016/j.psyneuen.2013.05.003
- Dowd, J. B., Simanek, A. M., & Aiello, A. E. (2009). Socioeconomic status, cortisol and allostatic load: A review of the literature. *International Journal of Epidemiology*, 38(5), 1297–1309. doi:10.1016/j.psyneuen.2015.08.018
- Ellis, K. R., Griffith, D. M., Allen, J. O., Thorpe, R. J., Jr., & Bruce, M. A. (2015). "If you do nothing about stress, the next thing you know, you're shattered": Perspectives on African American men's stress, coping and health from African American men and key women in their lives. *Social Science & Medicine*, 139, 107–114. doi:10.1016/j. socscimed.2015.06.036
- Gaffey, A. E., Bergeman, C. S., Clark, L. A., & Wirth, M. M. (2016). Aging and the HPA axis: Stress and resilience in older adults. *Neuroscience & Biobehavioral Reviews*, 68, 928–945. doi:10.1016/j.neubiorev.2016.05.036
- Geronimus, A. T. (1992). The weathering hypothesis and the health of African-American women and infants: Evidence and speculations. *Ethnicity and Disease*, 2, 207–221.
- Geronimus, A. T., Bound, J., Keene, D., & Hicken, M. (2007). Black-White differences in age trajectories of hypertension prevalence among adult women and men, 1999–2002. *Ethnicity and Disease*, 17(1), 40–48.
- Geronimus, A. T., Hicken, M., Keene, D., & Bound, J. (2006). "Weathering" and age patterns of allostatic load scores among Blacks and Whites in the United States. *American Journal of Public Health*, *96*(5), 826–833. doi:10.2105/AJPH.2004.060749
- Geronimus, A. T., Pearson, J. A., Linnenbringer, E., Schulz, A. J., Reyes, A. G., Epel, E. S., . . . Blackburn, E. H. (2015). Raceethnicity, poverty, urban stressors, and telomere length in a Detroit community-based sample. *Journal of Health and Social Behavior*, 56(2), 199–224. doi:10.1177/0022146515582100
- Geronimus, A. T., & Thompson, J. P. (2004). To denigrate, ignore, or disrupt: Racial inequality in health and the impact of a policy-induced breakdown of African American communities. *Du Bois Review*, 1(2), 247–279. doi:10.1017/S1742058X04042031
- Gilbert, K. L., Ray, R., Siddiqi, A., Shetty, S., Baker, E. A., Elder, K., & Griffith, D. M. (2016). Visible and invisible trends in Black men's health: Pitfalls and promises for addressing racial, ethnic, and gender inequities in health.

- Annual Review of Public Health, 37, 295–311. doi:10.1146/annurev-publhealth-032315-021556
- Granger, D. A., Hibel, L. C., Fortunato, C. K., & Kapelewski, C. H. (2009). Medication effects on salivary cortisol: Tactics and strategy to minimize impact in behavioral and developmental science. *Psychoneuroendocrinology*, 34, 1437–1448. doi:10.1016/j.psyneuen.2009.06.017
- Griffith, D. M. (2012). An intersectional approach to men's health. *Journal of Men's Health*, *9*(2), 106–112. doi:10.1016/j.jomh.2012.03.003
- Griffith, D. M., Ellis, K. R., & Allen, J. O. (2013). An intersectional approach to social determinants of stress and African American men's health: Men's and women's perspectives. American Journal of Men's Health, 7(Suppl 4), S19–S30. doi:10.1177/1557988313480227
- Griffith, D. M., Gunter, K., & Allen, J. O. (2011). Male gender role strain as a barrier to African American men's physical activity. *Health Education and Behaviors*, 38(5), 482–491. doi:10.1177/1090198110383660
- Hajat, A., Diez-Roux, A., Franklin, T. G., Seeman, T., Shrager, S., Ranjit, N., . . . Kirschbaum, C. (2010). Socioeconomic and race/ethnic differences in daily salivary cortisol profiles: The multi-ethnic study of atherosclerosis. *Psychoneuroendocrinology*, 35(6), 932–943. doi:10.1016/j. psyneuen.2009.12.009
- Hill, T. D., Graham, L. M., & Divgi, V. (2011). Racial disparities in pediatric asthma: A review of the literature. *Current Allergy and Asthma Reports*, 11(1), 85–90. doi:10.1007/s11882-010-0159-2
- Jackson, J. W., Williams, D. R., & VanderWeele, T. J. (2016). Disparities at the intersection of marginalized groups. Social Psychiatry and Psychiatric Epidemiology, 51(10), 1349–1359. doi:10.1007/s00127–016–1276–6
- Johnson-Lawrence, V., Griffith, D. M., & Watkins, D. C. (2013). The effects of race, ethnicity, and mood-anxiety disorders on the chronic physical health conditions of men from a national sample. *American Journal of Men's Health*, 7(Suppl 4), S58–S67. doi:10.1177/1557988313484960
- Juster, R. P., McEwen, B. S., & Lupien, S. J. (2010). Allostatic load biomarkers of chronic stress and impact on health and cognition. *Neuroscience & Biobehavioral Reviews*, 35(1), 2–16.
- Karasek, R. A., & Theorell, T. (1990). Healthy work: Stress, productivity, and the reconstruction of working life. New York. NY: Basic Books.
- Karb, R. A., Elliott, M. R., Dowd, J. B., & Morenoff, J. D. (2012). Neighborhood-level stressors, social support, and diurnal patterns of cortisol: The Chicago Community Adult Health Study. *Social Science and Medicine*, 75(6), 1038–1047. doi:10.1016/j.socscimed.2012.03.031
- Karlamangla, A. S., Friedman, E. M., Seeman, T. E., Stawksi, R. S., & Almeida, D. M. (2013). Daytime trajectories of cortisol: Demographic and socioeconomic differences—findings from the National Study of Daily Experiences. *Psychoneuroendocrinology*, 38(11), 2585–2597. doi:10.1016/j.psyneuen.2013.06.010
- Kumari, M., Badrick, E., Ferrie, J., Perski, A., Marmot, M., & Chandola, T. (2009). Self-reported sleep duration and

sleep disturbance are independently associated with cortisol secretion in the Whitehall II study. *The Journal of Clinical Endocrinology & Metabolism*, *94*(12), 4801–4809. doi:10.1210/jc.2009–0555

- Lam, J. C. W., Shields, G. S., Trainor, B. C., Slavich, G. M., & Yonelinas, A. P. (2018). Greater lifetime stress exposure predicts blunted cortisol but heightened DHEA responses to acute stress. *Stress and Health*. Advance online publication. doi:10.1002/smi.2835
- Leikauf, J., & Federman, A. D. (2009). Comparisons of self-reported and chart-identified chronic diseases in inner-city seniors. *Journal of the American Geriatrics Society*, 57(7), 1219–1225. doi:10.1111/j.1532–5415.2009.02313.x
- Martin, L. A., Neighbors, H. W., & Griffith, D. M. (2013). The experience of symptoms of depression in men vs women: Analysis of the national comorbidity survey replication. *JAMA Psychiatry*, 70(10), 1100–1106. doi:10.1001/jama-psychiatry.2013.1985
- McEwen, B. S., & Gianaros, P. J. (2010). Central role of the brain in stress and adaptation: Links to socioeconomic status, health, and disease. *Annals of the New York Academy of Sciences*, *1186*, 190–222. doi:10.1111/j.1749–6632.2009.05331.x
- Mroczek, D. K., & Kolarz, C. M. (1998). The effect of age on positive and negative affect: A developmental perspective on happiness. *Journal of Personality and Social Psychology*, 75(5), 1333-1349. doi:10.1037/0022-3514.75.5.1333
- National Center for Health Statistics (NCHS). (2017). *Health, United States, 2016: With chartbook on long-term trends in health* (DHHS Publication No. 2017–1232). Hyattsville, MD: US Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Health Statistics.
- National Center for Health Statistics (NCHS). (2018). *Life expectancy at birth, at age 65, and at age 75, by sex, race, and Hispanic origin. United States, selected years 1900–2016. Health, United States, 2017.* Hyattsville, MD: US Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Health Statistics.
- Olives, C., Myerson, R., Mokdad, A. H., Murray, C. J., & Lim, S. S. (2013). Prevalence, awareness, treatment, and control of hypertension in U.S. counties, 2001–2009. *PLoS One*, 8(4): e60308. doi:10.1371/journal.pone.0060308
- Paradies, Y. (2006). A review of psychosocial stress and chronic disease for 4th world indigenous people and African Americans. *Ethnicity & Disease*, *16*, 295–308.
- Piazza, J. R., Charles, S. T., Stawski, R. S., & Almeida, D. M. (2013). Age and the association between negative affective states and diurnal cortisol. *Psychology and Aging*, 28(1), 47–56. doi:10.1037/a0029983
- Radler, B. T., & Ryff, C. D. (2010). Who participates? Accounting for longitudinal retention in the MIDUS national study of health and well-being. *Journal of Aging Health*, 22, 307–331. doi:10.1177/0898264309358617
- Ross, K. M., Murphy, M. L., Adam, E. K., Chen, E., & Miller, G. E. (2014). How stable are diurnal cortisol activity indices in healthy individuals? Evidence from three multi-wave studies.

- *Psychoneuroendocrinology*, *39*, 184–193. doi:10.1016/j. psyneuen.2013.09.016
- Samuel, L. J., Roth, D. L., Schwartz, B. S., Thorpe, R. J., Jr., & Glass, T. A. (2018). Socioeconomic status, race/ethnicity, and diurnal cortisol trajectories in middle-aged and older adults. *The Journal of Geronotology: Series B*, 73(3), 468– 476. doi:10.1093/geronb/gbw080
- Smith, W. A., Allen, W. R., & Danley, L. L. (2007). "Assume the position. . . you fit the description" psychosocial experiences and racial battle fatigue among African American male college students. *American Behavioral Scientist*, 51(4), 551–578. doi:10.1177/0002764207307742
- Staufenbiel, S. M., Penninx, B. W., Spijker, A. T., Elzinga, B. M., & van Rossum, E. F. (2013). Hair cortisol, stress exposure, and mental health in humans: A systematic review. *Psychoneuroendocrinology*, 38(8), 1220–1235. doi:10.1016/j.psyneuen.2012.11.015
- Sturmberg, J. P., Bennett, J. M., Martin, C. M., & Picard, M. (2017). 'Multimorbidity' as the manifestation of network disturbances. *Journal of Evaluation in Clinical Practice*, 23(1), 199–208. doi:10.1111/jep.12587
- Thorpe, R. J., Jr., & Kelley-Moore, J. A. (2012). Life course theories of race disparities: A comparison of cumulative dis/advantage perspective and the weathering hypothesis. In T. A. LaVeist, & L. A. Isaac (Eds.), *Race, ethnicity, and health: A public health reader* – second edition (pp. 355– 375). San Francisco: Jossey-Bass.
- van den Beld, A. W., Kaufman, J. M., Zillikens, M. C., Lamberts, S. W. J., Egan, J. M., & van der Lely, A. J. (2018). The physiology of endocrine systems with ageing. *The Lancet Diabetes & Endocrinology*, *6*(8), 647–658. doi:10.1016/S2213-8587(18)30026-3
- Wang, X., Sánchez, B. N., Golden, S. H., Shrager, S., Kirschbaum, C., Karlamangla, A. S., & Roux, A. V. D. (2014). Stability and predictors of change in salivary cortisol measures over six years: MESA. *Psychoneuroendocrinology*, 49, 310– 320. doi:10.1016/j.psyneuen.2014.07.024
- Watkins, D. C., & Johnson, N. C. (2018). Age and gender differences in psychological distress among African Americans and Whites: Findings from the 2016 national health interview survey. *Healthcare*, 6(1), E6. doi:10.3390/ healthcare6010006
- Williams, D. R., & Mohammed, S. A. (2013). Racism and health I: Pathways and scientific evidence. *American Behavioral Scientist*, 57(8), 1152–1173. doi:10.1177/ 0002764213487340
- Williams, D. R., & Sternthal, M. (2010). Understanding racialethnic disparities in health: Sociological contributions. *Journal of Health and Social Behavior*, 51(Suppl 1), S15– S27. doi:10.1177/0022146510383838
- Yudell, M., Roberts, D., DeSalle, R., & Tishkoff, S. (2016). Science and society: Taking race out of human genetics. *Science*, 351(6273), 564–565. doi:10.1126/science.aac4951
- Zajacova, A., & Dowd, J. B. (2011). Reliability of self-rated health in US adults. *American Journal of Epidemiology*, 174(8), 977–983. doi:10.1093/aje/kwr204