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Sleep problems predict cortisol reactivity to stress in urban adolescents

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HIGHLIGHTS

- Sleep problems predicted greater cortisol reactivity to stress in urban adolescents.
- Longer sleep duration predicted post-stress cortisol over sleep problems.
- Self-reports of sleep were more consistently related to cortisol than parent reports.
- Effects of sleep problems on cortisol levels were stronger in girls than boys.

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ABSTRACT

This study examined the role of sleep problems and sleep duration on stress-related HPA axis reactivity among urban, low income adolescents. A total of 84 adolescents (M age 13.36 years; 50% male; 95% African American) and their parents provided information on adolescents' sleep problems and sleep quantity. Adolescents completed a standardized social stress test in the laboratory (the Trier Social Stress Test; TSST). Saliva samples collected before and after the TSST yielded measures of cortisol pre-test, 15 min post-test, and 55 min post-test, as well as overall cortisol secretion and its increase (AUC_G and AUC_I). More sleep problems and longer sleep duration predicted higher cortisol reactivity to the TSST, particularly among females. Self-reports of sleep were more consistently related to stress-related cortisol reactivity than parent reports. Sleep problems and longer sleep duration may place adolescents at risk for HPA axis hyper-reactivity to stress, contributing to academic, behavioral and health problems.

1. Introduction

Almost 70% of US adolescents do not receive sufficient sleep (8 or more hours per night) and only 8% report optimal sleep duration of 9 or more hours [12]. Both insufficient sleep and sleep problems contribute to cognitive problems and poor mental and physical health over time [35]. Since adequate sleep is necessary for optimal functioning of the HPA axis [42] and dysregulation of the HPA axis contributes to cognitive, emotional, and health problems [24], alteration of HPA axis activity has been proposed as a key mechanism through which sleep problems lead to negative outcomes [27]. The relationship between sleep and HPA axis activity has been studied in children and adults, but little is known about this link during adolescence when both sleep and the HPA axis undergo significant developmental changes related to puberty [10,35].

Among prepubertal children and adults, insufficient sleep and sleep problems consistently predict elevated cortisol levels throughout the day [13,19,22,27,32,38]. The only study conducted with adolescents found no effect of sleep restriction on morning and evening cortisol levels [43], but the study was limited by small sample sizes in each experimental condition (n = 12 to 17). Children with sleep problems and sleep deprived adults also had greater cortisol reactivity to stress [19,27,38], but lower stress reactivity was observed in adult women with lower objectively measured sleep quality [46] and in 10–12 year olds with more self-reported sleep–wake problems [8]. In the latter study, there was no link between sleep–wake problems and cortisol reactivity among adolescents (13–17 years old); however, the conclusions are limited by a small sample size (N = 31 across both age groups) and the use of parent reports of sleep problems. Parent reports of sleep problems have been validated for pre-adolescent children [3], but they tend to underestimate older children's sleep problems [33] and may be particularly inaccurate for adolescents who receive less parental supervision [1].

Given the paucity of research on sleep and HPA axis in adolescence, this study sought to examine the effects of sleep on HPA axis activity in this age group. Specifically, we examined two dimensions of sleep — sleep duration and sleep problems, from the perspectives of adolescents and their parents, as well as cortisol levels before and after social stress.

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Because urban African American adolescents are more likely to experience insufficient sleep [1,26] and their functioning is more negatively affected by lower sleep quality [5], we focused on the understudied population of urban and mostly African American adolescents. We hypothesized that shorter sleep duration and more sleep problems will predict higher cortisol levels and reactivity to stress (i.e., a social evaluative threat). Finally, because cortisol reactivity to stress in urban, African American adolescents and young adults varies by gender [2,11], we also examined gender differences in the effects of sleep on stress-related cortisol regulation and reactivity.

2. Material and methods

2.1. Participants and procedures

Participants were 84 adolescents (M age 13.36 years, SD = 0.95; 50% male; 95% African American, 4% Caucasian and 1% Hispanic) who took part in the Coping with Violence Study. The sample was socioeconomically heterogeneous, but comprised primarily low-income families; average annual family income was $20,000–$25,000 (range <$5000 to $70,000–$90,000) and average parental education was some college but no degree (see Table 1 for sample characteristics). The adolescents were recruited from four public middle schools (grades 6–8 or 9) serving low income, urban communities in Birmingham, AL. Across the four schools, 83% to 87% of students were eligible for free or reduced price lunch. Students received an envelope that contained a description of the study, contact information form, and informed consent and assent forms. Families interested in participating were instructed to return a completed contact information form to the school. These families were later contacted by study staff and scheduled for an interview at a university laboratory. From approximately 240 invited students, 129 (54%) provided their contact information and 84 of those (65%) completed the interview (recruitment was curtailed by limited resources). After providing parental informed consent and child assent, parents and adolescents were interviewed separately in private spaces by trained interviewers using computer assisted technology. The adolescent interview included anthropometric measurement of height and weight, as well as the Trier Social Stress Test-Children's version (TSST-C; [7]) with saliva samples collected before and after the TSST-C (see Fig. 1 for a timeline of interview procedures). Following recommendations by Granger et al. [16], all interviews were conducted in the afternoon (between 3 and 6 pm) to minimize the effects of diurnal variation in cortisol production on the likelihood of detecting an increase in cortisol levels in response to the TSST-C. None of the adolescents were diagnosed with any psychiatric disorders (per parent report) or were taking any medications [17] that would affect cortisol levels (per self-report). The interview session took two hours to complete, and adolescents and their caregivers were compensated for their time with $50 gift cards. All procedures were approved by the university Institutional Review Board.

Table 1
Descriptive statistics.

	Adolescent report		Parent report	
	M (SD)	Range	M (SD)	Range
Bedtime weeknight	22:00 (0:59)	19:00–24:00	21:32 (1:00)	19:50–1:00
Wake time weeknight	6:00 (0:32)	4:00–6:52	6:00 (0:32)	4:45–7:00
Bedtime weekend	23:50 (1:47)	20:00–6:00	23:23 (1:28)	20:00–3:00
Wake time weekend	9:32 (2:25)	5:00–15:00	9:13 (1:48)	6:00–13:00
Sleep quantity, hours	8.73 (1.12)	5.57–11.71	8.87 (1.15)	5.21–11.79
Sleep problems	1.90 (0.41)	1.23–3.20	1.57 (0.34)	1.00–2.81
Pubertal development	2.64 (0.65)	1.00–4.00	2.48 (0.63)	1.00–4.00
	Other measures			
Child age, years	13.36 (0.95)		11.82–16.56	
Pubertal development	2.56 (0.57)		1.10–3.75	
BMI%	74.15 (26.43)		0.60–99.80	
Family income[a]	5.22 (3.30)		1–12	
Parental education[b]	4.67 (2.05)		2–8	
Pre-test cortisol, μg/dL	0.10 (0.10)		0.02–0.59	
15 min post-test cortisol, μg/dL	0.11 (0.15)		0.02–0.94	
55 min post-test cortisol, μg/dL	0.07 (0.09)		0.00–0.50	
Area under the curve, ground (AUC_G)	6.77 (7.61)		1.34–51.75	
Area under the curve, increase (AUC_I)	−0.65 (7.05)		−29.50–39.49	

Note:
[a] A 13-point scale from 1 (<$5000/year) to 13 (>$90,000/year).
[b] An 8-point scale from 1 (less than 9th grade) to 8 (graduate or professional degree).

2.2. Measures

2.2.1. Sleep quantity

Adolescents and parents were asked at what time the adolescent usually goes to bed and wakes up, separately for weekdays and weekends or holidays. The bedtime and waking times were used to compute typical sleep duration on weekday and weekend nights, respectively. Then, the average daily amount of sleep was derived for a typical week consisting of five weekdays and two weekend days, separately for each reporter.

2.2.2. Sleep problems

A 22-item scale from the Sleep History section of the Adolescent Sleep Habit Survey [34] was used to measure adolescents' sleep problems. The items inquired about the frequency of various sleep problems during a typical week, including disruptions to the wake-sleep cycle (e.g., staying up late), insomnia (e.g., difficulty falling or staying asleep), daytime sleepiness (e.g., falling asleep during the day), parasomnias (e.g., nightmares), sleep disordered breathing (e.g., snoring, gasping for breath), and general sleep quality (e.g., satisfied with sleep; reverse coded). The same questions were completed by adolescents and parents using a 5-point scale ranging from 'Never' (1) to 'Every day' (5). Factor analyses confirmed that all items loaded on a single dimension. The responses were averaged (adolescent $\alpha = .73$; parent $\alpha = .64$).

2.2.3. Trier social stress test

After getting acclimated to the lab environment and being interviewed for approximately 30 min, adolescents completed the TSST-C [7]. Adolescents were given five minutes to prepare the ending to a story and then five minutes to tell that story in front of two judges. Next, the judges asked the participants to perform serial subtraction for 5 min (subtracting 7 starting at 758). However, if the adolescents made five mistakes in a row, they were given an easier task (subtracting 3 starting at 307). The judges wore white coats and provided no positive feedback; the participants were also videotaped during the test.

2.2.4. Saliva collection and determination of cortisol

Whole saliva samples were collected by passive drool [16] immediately before the TSST began (pre-test), 30 min after the 15-min test began (15 min post-test), and 70 min after the test began (55 min post-test). Samples were immediately frozen at −20 °C and later shipped on dry ice overnight to the Johns Hopkins University's Interdisciplinary Salivary Bioscience Center where they were kept frozen at −80 °C until assayed. Cortisol was assayed in duplicate by commercially available enzyme immunoassay without modification to the manufacturers recommended protocol (Salimetrics, Carlsbad, CA). Sample test volume was 25 μl, the range of sensitivity was from .007 to 3.0 μg/dL, and the inter- and intra-assay coefficients of variation were, on average, less than 15% and 10%, respectively. The average of the duplicate assays was used in all statistical analyses. Units of cortisol are presented in μg/dL (micrograms/deciliter).

2.2.5. Covariates

Possible covariates included time of day when saliva samples were taken, adolescents' age and gender, family income and parental education (reported by parents on 13- and 8-point scales, respectively), adolescents'

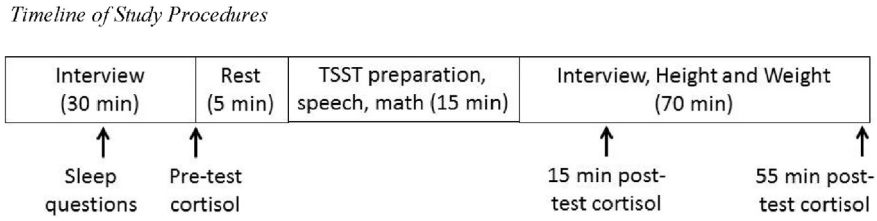

Fig. 1. Timeline of study procedures.

pubertal status computed as the average of parent and adolescent reports using the Pubertal Development Scale (Petersen et al. [47]; scores ranged from 1 — prepubertal to 5 — postpubertal), and adolescents' BMI percentile, computed from height and weight measured during the interview and age- and sex-specific CDC growth charts. Adjustments for reference standards (using percentiles or z-scores) are necessary to interpret BMI in pediatric populations, because the meaning of specific BMI values varies across age and sex [30].

2.3. Statistical analyses

Descriptive statistics were computed and outliers truncated to 3 SD from the mean [16]. Then, bivariate correlations among all variables were examined. Parent and child reports of wake and bed times, sleep quantity, and sleep problems were compared with paired samples t-tests, as were cortisol levels across the three time points. The effects of sleep variables on cortisol were tested with multiple regressions predicting cortisol levels at pre-test and 15 and 55 min post-test, respectively, from sleep quantity and sleep problems. The analyses of post-test cortisol were adjusted for pre-test cortisol levels. Total cortisol secretion and its increase during the studied period were also analyzed with areas under the curve with respect to ground and increase (AUC_G and AUC_I), respectively [37]. In order to evaluate the roles of parent and adolescent reports of sleep not confounded by overlap between raters, separate analyses were conducted for adolescent and parent reported sleep variables. Given the limited sample size, only covariates that were related to both cortisol and sleep variables (and thus could confound their relationships) were included in the main analyses. The regression analyses were conducted in Mplus version 7.11 using Full Information Maximum Likelihood (FIML) which utilizes all available data to compute parameter estimates, thus preserving the full sample size and yielding more unbiased results than listwise deletion of cases with missing data [45]. Because cortisol values were positively skewed and log transformations failed to normalize their distributions, the regression analyses were conducted with Maximum Likelihood estimation with robust standard errors (MLR), a procedure that is robust to violations of normality and independence. Gender differences in the effects of sleep duration and quality on cortisol were tested with multigroup modeling using the Satorra–Bentler Scaled Chi-Square test [41], comparing the fit of a model with sleep variables' effects constrained to be equal across gender to a model where the paths could vary between boys and girls. A Bonferroni-adjusted p level of .005 was used for these analyses.

3. Results

Preliminary data analyses identified seven cortisol observations as outliers; these values were truncated to 3 SD above the mean. Overall, 11% of participants had some missing data, but only 1.62% of observations were missing, supporting the use of FIML to utilize all available data in the regression analyses. The adolescents were on average 13 years old, early- to mid-pubertal, and at the 74th percentile for BMI (Table 1). Descriptives for all sleep items (Table 2) indicated that the most common sleep problems reported by the adolescents were the need for multiple reminders to get up in the morning (item 1), not having a good night sleep (item 15), feeling tired or sleepy during the day (item 5), and not being satisfied with their sleep (item 21). The most common sleep problems reported by the parents were the need for multiple reminders to get up (item 1), waking up once at night (item 17), snoring (item 19), and taking naps (item 22).

There were no systematic differences between adolescent- and parent-reported wake times (both weekdays and weekends) and weekday bed times, as well as overall duration of the adolescents' sleep (paired $t_{(82)} = -1.59$ to 1.38, all $p > .116$). However, adolescents reported later weekend bedtimes (paired $t_{(82)} = 2.27$, $p = .026$) and more sleep problems (paired $t_{(82)} = 6.81$, $p < .001$) compared to parent reports. On average, cortisol levels did not change from pre-test to 15 min post-test (paired $t_{(75)} = -0.28$, $p = .777$). However, there was substantial variability in individual cortisol responses to the TSST, with 36% of the adolescents having a 10% or greater increase in cortisol, and 29% of the sample showing a 10% or greater increase that was also equal or greater than 0.02 μg/dL. Additionally, there was a consistent decline in cortisol values between 15 and 55 min post-test (paired

Table 2
Descriptive statistics for sleep problem items.

	Adolescent M (SD)	Parent M (SD)
1. Need reminders to get up in the morning	3.14 (1.58)	2.90 (1.58)
2. Arrive late to class because overslept	1.48 (0.77)	1.22 (0.47)
3. Fall asleep in a morning class	1.46 (0.74)	1.18 (0.49)
4. Fall asleep in an afternoon class	1.46 (0.99)	1.07 (0.32)
5. Feel tired, dragged out or sleepy during the day?	2.83 (1.23)	2.09 (1.13)
6. Go to bed because could not stay awake any longer?	2.68 (1.35)	2.13 (1.02)
7. Sleep on past noon?	1.87 (1.08)	1.27 (0.64)
8. Stay up to at least 3 am?	1.92 (1.11)	1.32 (0.77)
9. Stay up all night?	1.62 (0.97)	1.10 (0.34)
10. Have an extremely hard time falling asleep?	2.02 (1.05)	1.24 (0.54)
11. Awaken too early in the morning and can't get back to sleep?	2.29 (1.00)	1.27 (0.58)
12. Have fearful thoughts or images as is falling asleep?	1.73 (1.05)	1.24 (0.65)
13. Have nightmares or bad dreams during night?	1.63 (0.79)	1.24 (0.57)
14. Walk in sleep?	1.13 (0.49)	1.06 (0.33)
15. Have a good night sleep?[a]	1.88 (1.03)	3.27 (0.82)
16. Wet bed?	1.02 (0.22)	1.02 (0.22)
17. Wake up once during the night?	2.48 (1.10)	2.33 (1.04)
18. Wake up more than once during the night?	1.75 (1.05)	1.47 (0.86)
19. Snore?	1.96 (1.56)	2.33 (1.63)
20. Stop breathing or wake up gasping for air?	1.23 (0.55)	1.10 (0.49)
21. Feel satisfied with sleep?[a]	2.20 (1.32)	3.19 (1.11)
22. Take naps during the day?	2.55 (1.35)	2.27 (1.30)

Note: All items are rated on a scale ranging from 1 (Never) to 5 (Everyday).
[a] Item was reverse coded before scale scores were computed.

$t_{(77)} = 4.89$, $p < .001$) and between pre-test and 55 min post-test (paired $t_{(77)} = 3.07$, $p = .003$) (see Table 1 for means).

Correlations (Table 3) indicated modest agreement between child and parent reports of sleep quantity and sleep problems. In terms of wake and bed times (not in the table), weekday wake times were most strongly correlated across informants ($r_{(81)} = .64$, $p < .001$), followed by weekend wake times ($r_{(81)} = .41$, $p < .001$) and weekend and weekday bedtimes ($r_{(81)} = .31$, $p = .004$, and $r_{(81)} = .29$, $p = .009$). Sleep quantity and sleep problems were not correlated with each other, except for a positive relationship between adolescent-reported sleep duration and parent-reported sleep problems. Pre-test cortisol was not correlated with any sleep variables, but cortisol at 15 min post-test was higher for adolescents who reported more sleep problems and whose parents reported longer sleep duration. Cortisol at 55 min post-test was higher among adolescents reporting more sleep problems. Similarly, adolescent-reported sleep problems were associated with greater overall and increased cortisol secretion, as was parent-reported longer duration of sleep. Adolescent-reported sleep duration was also related to greater AUC_I. All of these significant correlations were of medium magnitude (range .23 to .33).

Among the covariates, pubertal development was not related to any sleep or cortisol variables. Adolescents who were older and came from families with higher income had shorter sleep duration per both reporters; higher income adolescents also had fewer parent-reported sleep problems. Additionally, adolescents with higher BMI% reported fewer sleep problems. For cortisol, parental education was related to higher pre-test cortisol, family income was associated with lower AUC_I, and females had higher cortisol at 55 min post-test and greater AUC_I. Finally, sampling time of day was not related to cortisol values or any of the sleep variables. All significant correlations with the covariates were also of medium size (range .22 to .30). Because only family income was related to both sleep and cortisol and thus could confound their relationships, it was included as a covariate in all regression analyses.

Regression analyses tested whether sleep duration and sleep quality predicted cortisol secretion, with family income included as a covariate. We hypothesized that shorter sleep duration and more sleep problems will predict higher baseline cortisol and greater reactivity to the stress test. However, none of the sleep variables predicted pre-test cortisol (Table 4). Analyses of post-test cortisol levels were also adjusted for pre-test cortisol, and thus can be interpreted as predicting change in cortisol. Adolescent-reported sleep problems predicted greater cortisol levels at both 15 and 55 min post-test, as well as greater overall cortisol secretion and greater increase in cortisol secretion (AUC_G and AUC_I; see Fig. 2). Additionally, adolescents who reported longer sleep duration had higher cortisol levels at 55 min post-test and greater increase in cortisol secretion (AUC_I; Fig. 2). Parent report of sleep duration predicted greater overall cortisol secretion (AUC_G), but parent-reported sleep problems did not predict any cortisol variables. All significant effects of sleep on cortisol fell in the medium size range (.22 to .31).

Finally, multigroup modeling tested gender differences in the effects of sleep variables on cortisol secretion. After applying Bonferroni correction for multiple testing, significant gender differences emerged for child-reported sleep variables predicting cortisol levels 15-min post-test, as well as AUC_G and AUC_I (all $p < .005$). In each case, there was a significant effect of sleep problems on elevated cortisol levels for girls, but not for boys. Specifically, sleep problems uniquely predicted girls' 15-min post-test cortisol ($\beta = .15$, $p < .01$), AUC_G ($\beta = .28$, $p < .05$), and AUC_I ($\beta = .31$, $p < .01$), whereas these effects were not significant for boys ($\beta = .20$, $p = .07$ for 15-min post-test cortisol; $\beta = .19$, $p = .22$ for AUC_G; and $\beta = .21$, $p = .08$ for AUC_I). Consistent with the main results, the coefficients for both boys and girls were within the medium effect size range.

4. Discussion

This study examined relationships between sleep and HPA axis activity in adolescence, focusing on a vulnerable, yet understudied population of urban and mostly African American adolescents. The results revealed greater cortisol reactivity to social evaluative threat among adolescents who reported more sleep problems and longer sleep duration, and whose parents also reported longer sleep duration. Girls showed greater cortisol elevation in response to stress than boys, as well as stronger effects of sleep problems on cortisol reactivity. All effect sizes were in the medium range.

The association between sleep problems and elevated cortisol secretion in response to social stress is consistent with results obtained from younger children (ages 5 and 8) using objective measures of sleep, such as sleep EEG and actigraphy [19,38]. By contrast, the opposite pattern of lower cortisol reactivity in pre-adolescents (10–12 yr. old) and no association among adolescents (13–17 yr. old) were obtained in the only other study on sleep and cortisol stress reactivity in adolescents [8]. It is possible that the discrepant results in the latter study were due to the small sample size (N = 31 across both age groups) and the use of

Table 3
Correlations.

	1.	2.	3.	4.	5.	6.	7.	8.	9.
1. Sleep quantity, C	–								
2. Sleep quantity, P	.32**	–							
3. Sleep problems, C	−.14	.07	–						
4. Sleep problems, P	.22*	.00	.32**	–					
5. Pre-test cortisol	−.19	.02	.08	.02	–				
6. 15 min post-test cortisol	.08	.25*	.33**	.04	.37**	–			
7. 55 min post-test cortisol	.15	.19	.24*	.04	.33**	.85***	–		
8. AUC_G	.06	.24*	.31**	.05	.54***	.97***	.90***	–	
9. AUC_I	.24*	.23*	.26*	.00	−.41***	.68***	.64***	.55***	–
Covariates									
10. Child age	−.25*	−.29**	−.06	−.07	.03	−.02	.06	.01	−.01
11. Female	.08	.12	.01	−.04	−.21	.18	.22*	.15	.35**
12. Pubertal development	.20	.05	−.09	.19	−.02	.07	.11	.07	.08
13. BMI%	.13	.07	−.28*	.16	.10	−.01	−.01	.03	−.09
14. Family income	−.24*	−.30**	−.03	−.22*	.19	−.11	−.17	−.09	−.30**
15. Parental education	−.16	−.06	−.09	−.19	.23*	.08	−.02	.09	−.12
16. Time of pre-test cortisol	.18	−.11	.02	−.01	−.04	.06	.02	.05	.09

Note: C — child report; P — parent report; AUC_G — area under the curve, ground; AUC_I — area under the curve, increase.
* $p < .05$.
** $p < .01$.
*** $p < .001$.

Table 4
Multiple regressions predicting cortisol from sleep quantity and quality.

	Pre-test cortisol β	15 min post-test cortisol β	55 min post-test cortisol β	AUC_G β	AUC_I β
Adolescent report					
Pre-test cortisol	–	.41***	.38***	–	–
Family income	.15	−.15	−.17+	−.06	−.23*
Sleep quantity	−.14	.16	.22*	.09	.22*
Sleep problems	.06	.31***	.24*	.30*	.27**
R^2	.06	.28***	.24**	.10	.19*
Parent report					
Pre-test cortisol	–	.40***	.36***	–	–
Family income	.25*	−.14	−.20+	−.02	−.26*
Sleep quantity	.08	.21+	.13	.24*	.16
Sleep problems	.10	−.03	−.02	.02	−.09
R^2	.06	.22**	.17*	.06	.12+

Note: AUC_G and AUC_I — area under the curve with respect to ground and increase, respectively.
+ p < .10.
* p < .05.
** p < .01.
*** p < .001.

parent reports of sleep–wake problems. Indeed, in our study parent reports of adolescents' sleep problems did not correlate with or predict cortisol stress reactivity, but adolescents' own reports of sleep problems did. Given previous validation of adolescent reports of sleep problems with actigraphy [44], our results suggest that self-reports of sleep problems may be more accurate than parent reports during adolescence.

Greater cortisol increase and stronger effects of self-reported sleep problems on cortisol reactivity to stress among females than males further add to the limited literature on gender differences in cortisol levels in urban African American adolescents. Previous studies found that African American male adolescents had flatter diurnal cortisol rhythm than African American females [11], and that chronic exposure to violence was linked with higher cortisol levels in African American females but lower levels in males [2]. Our findings are consistent with these reports, indicating generally greater HPA axis reactivity to stress and greater HPA axis sensitivity to sleep problems in females, compared to more blunted HPA axis responses (to both stress and sleep problems) in males.

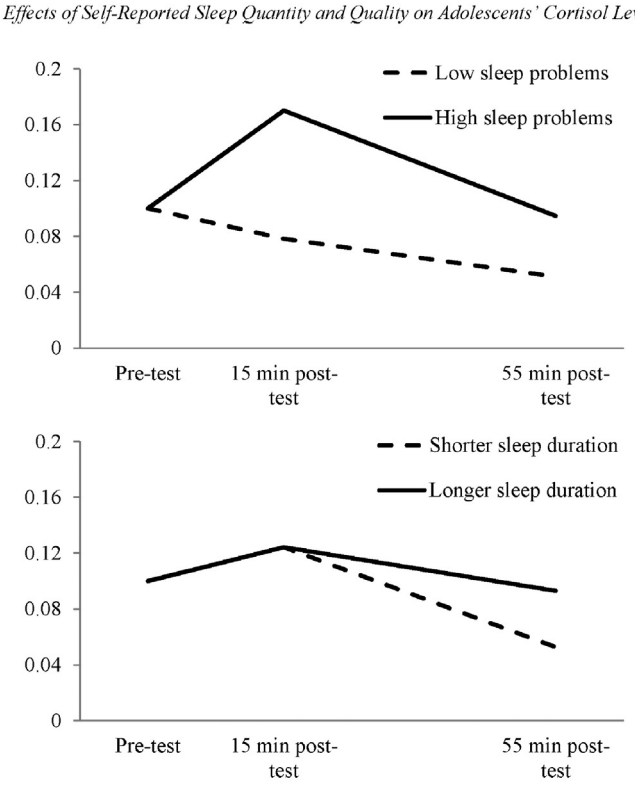

Fig. 2. Effects of self-reported sleep quantity and quality on adolescents' cortisol levels.

Over time, the enhanced and prolonged activation of the HPA axis in response to stress among adolescents experiencing sleep problems may contribute to more cognitive, emotional and health problems [24]. These mechanisms may be particularly relevant for urban minority adolescents who experience high levels of uncontrollable stress related to community and school violence [28], which has been linked to sleep problems [21], as well as to poor emotional, academic, and health outcomes [4,23,29]. In addition, higher cortisol levels in the evening may further contribute to night awakenings and other sleep problems [40], perpetuating a vicious cycle between sleep problems and elevated cortisol [6]. Thus, sleep problems and enhanced cortisol response to stress may play a key role in the negative effects of environmental stress among urban minority adolescents, and may be especially relevant for females. Interestingly, family SES also was related to cortisol levels, with higher pre-test cortisol levels among adolescents with more highly educated parents, and lower stress reactivity in those with higher family incomes. These effects are consistent with prior research [9] and may reflect greater dysregulation of the HPA axis in adolescents from low SES families.

Contrary to hypotheses and prior studies linking shorter sleep duration with enhanced cortisol stress response [27], longer sleep duration (both self- and parent-reported) predicted a stronger cortisol response to stress in this study. Interestingly, adolescent-reported sleep duration was not correlated with cortisol at 55 min post-test, but predicted these cortisol levels after adjusting for sleep problems. One possible explanation is that in this population of low-income urban adolescents, longer sleep duration does not necessarily reflect higher quality sleep, but instead may serve as another indicator of sleep problems. The positive correlation between adolescent-reported sleep duration and parent-reported sleep problems supports this possibility, but the use of objective sleep assessments (e.g., actigraphy) would be necessary to test this hypothesis and to better understand the role of sleep duration in HPA axis functioning in this population. Nevertheless, the findings indicate that urban minority adolescents who experience both sleep problems and longer sleep duration are at the greatest risk for over-reactivity of the HPA axis to stress and associated negative outcomes.

In contrast with studies of younger children and adults [13,32,38], neither sleep problems nor sleep duration predicted afternoon cortisol levels at pre-test, prior to social stress. However, this lack of association is in line with no effects of sleep restriction on evening cortisol levels in adolescents [43], suggesting that sleep may have little effect on afternoon and evening cortisol levels in adolescents. Although one study found higher diurnal cortisol levels in adolescents with lower sleep quality [25], that study included a wide age range (8 to 18), and it is possible that younger children and older adolescents were mainly responsible for the overall results. Thus, it is possible that developmental differences in the effects of sleep on diurnal cortisol levels exist, particularly between childhood and adulthood versus early and middle adolescence. Future research should investigate these possible differences by comparing different developmental groups or following individuals across stages of development. Multiple assessments of cortisol throughout the day would also be useful to detect developmental differences in diurnal cortisol levels related to sleep.

In terms of sleep quantity, adolescents in this study slept on average 8.73 h, which is similar to 8.30 h reported by more affluent middle school students [36], but higher than the average of 7–8 h reported by urban middle school students in Chicago [14]. Our adolescents also reported later bedtimes, earlier weekday wake times and later weekend wake times than other studies with North American adolescents, although overall sleep quantity was similar or higher [15]. In our sample, 23% of the adolescents reported sleeping less than 8 h per night, and 39% slept 9 or more hours. Even though parents' reports of sleep duration did not systematically differ from adolescent reports, parents seemed to be more optimistic about the amount of sleep that their children get, with 19% of parents reporting that their child sleeps less than 8 h per night, and 51% reporting their child getting 9 or more hours of sleep. Thus, a significant portion of adolescents was not receiving the recommended 8 or more hours of sleep. Consistent with other studies [12,14,15,36], older adolescents reported shorter sleep durations, likely reflecting biologically driven evening-type circadian phase preference, increased academic and social demands, and decreases in parental monitoring [35].

The adolescents in this study also reported higher levels of sleep problems compared to other adolescent studies. For instance, 17% of our adolescents reported having 'extremely hard time falling asleep' two or more days a week, compared to 7–12% of adolescents reporting often having difficulty falling asleep in other US studies [20,31,39]. Similarly, 23% of our adolescents reported feeling sleepy most days of the week, compared to 20% in a national US study [31]. Although higher levels of sleep problems would be expected among adolescents living in disadvantaged urban communities [39], comparisons across studies are difficult to make due to lack of standardized measures of sleep problems.

4.1. Limitations

Limitations of this study include lack of objective measurements of sleep (e.g., with actigraphy or EEG) or the use of sleep diaries that may be less subject to recall bias than the one-time reports of sleep duration and quality obtained in the lab. Additionally, sleep onset latency was not measured, making the interpretation of sleep duration results difficult. Another limitation is the use of the Adolescent Sleep Habit Survey, which has not been widely used in research and does not assess many dimensions of sleep quality (e.g., sleep onset or sleep efficiency). Even though several subscales can be derived from the measure (e.g., sleep–wake problems, parasomnias, night wakings, and sleep-disordered breathing), these subscales had low internal consistency in the current sample and generally replicated the reported results with the largest (10-item) sleep–wake problems subscale, suggesting that the results for sleep quality are driven primarily by sleep–wake problems. Additionally, the number of cortisol samples obtained from the participants was relatively small, with a single cortisol estimate for pre-test and two post-test time points. Obtaining more cortisol samples throughout the day would provide a more sensitive test of sleep effects on diurnal cortisol secretion, and gathering additional cortisol samples after the stress test would allow for a more fine-grained analysis of sleep effects on the cortisol stress response and its duration. Another limitation is that only a portion of the sample showed the expected increase in cortisol between pre-test and 15 min post-test, although this pattern is consistent with others' results for early adolescents. For instance, Gunnar et al. [18] found no overall cortisol increase to the TSST-C (and negative AUC_I) for 11-year old children and 13-year old boys. The substantial variability in cortisol reactivity was also consistent with great variation in subjective responses to the TSST-C: participants rated the speech and math tasks as fairly stressful overall ($M = 4.14$ and 4.98 on a 1–7 scale), but substantial individual differences were present ($SD = 2.12$ and 2.02; range 1–7 for both). However, the individual variability in cortisol responses was still meaningfully predicted by individual differences in sleep problems and duration.

The sample size of this study was substantially larger compared to many studies in this area that only included 15 or 30 participants, yet it was still relatively small and thus limited the inclusion of many covariates and detection of small effects. Finally, the measure of sleep problems had relatively low internal consistency, particularly for parent reports, although similar values have been found in other studies [13]. The low reliability likely reflects substantial heterogeneity among the different sleep problems assessed.

4.2. Conclusions

This is the first study that evaluated the role of sleep in HPA axis activity among urban, ethnic minority adolescents who experience higher levels of environmental stress, sleep problems, and related negative outcomes compared to more affluent Caucasian adolescents. The strengths of the study include examination of both sleep duration and quality, inclusion of child and parent reports of sleep, and assessment of cortisol levels before and after a standardized social stress test. The results point to robust effects of sleep problems and longer sleep duration on elevated HPA axis reactivity to stress, particularly for adolescent reports of sleep problems and both parent and adolescent reports of sleep duration. Importantly, the effects of adolescent-reported sleep quantity and quality on elevated HPA axis activity were still evident more than 30 min after the stress test ended, suggesting that poor and longer sleep may augment chronic HPA axis hyperactivity following stress in this vulnerable population.

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