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Concurrent and Longitudinal Associations between Diurnal Cortisol and Body Mass Index across Adolescence

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Abstract

Purpose—Childhood and adolescent obesity have reached epidemic levels; however little is known about the psychobiological underpinnings of obesity in youth and whether these differ from the mechanisms identified in adults. The current study examines concurrent (i.e. measured at the same point in time) and longitudinal (i.e. using earlier cortisol measures to predict later BMI) associations between diurnal cortisol and body mass index (BMI) across adolescence.

Methods—Adolescent diurnal cortisol was measured over three days at each 11, 13, and 15 years. Hierarchical linear modeling was used to extract average measures of predicted morning, afternoon, evening levels of cortisol and the diurnal slope at each assessment. Adolescent BMI (kg/m²) was measured at 11, 13, 15, and 18 years. Sex, family socioeconomic status, mother's BMI, pubertal status, and adolescent mental health were examined as possible confounding variables.

Results—Linear regressions revealed that blunted patterns of adolescent cortisol were associated with increased measures of BMI across adolescence both concurrently and longitudinally, particularly when examining measures of cortisol in early adolescence. Multinomial logistic regressions extended the linear regression findings beyond BMI scores to encompass categories of obesity.

Conclusions—The current study builds on previous research documenting diurnal cortisol-obesity findings in adults by demonstrating similar findings exist both concurrently and longitudinally in adolescents. Findings suggest the association between cortisol and BMI is developmentally influenced and that blunted diurnal cortisol patterns can be identified in overweight individuals at a younger age than previously thought.

Keywords

Diurnal Cortisol; Body Mass Index (BMI); Concurrent; Longitudinal; Adolescence

Obesity is associated with an increased risk of health problems, including cardiovascular disease, diabetes, cancer, and mortality [1]. Many studies have implicated the hypothalamic-pituitary-adrenal (HPA) axis and cortisol in its etiology, often via the induction of insulin-resistance [2]. However, the majority of human studies have been cross-sectional and conducted in adults (for a review see [3]). Given the lack of long-term studies across childhood and adolescence, it is unclear if the same psychophysiological mechanisms are implicated in the development of obesity in children and adolescents. Increasing prevalence rates of childhood obesity [4] and the continuity of obesity into adulthood [5] make it critical to examine the same neuroendocrine pathways identified in adults in younger individuals to determine their possible role in the early development and overall stability of this disorder. The overall aim of the present study is to address this issue.

Cortisol typically follows a diurnal pattern, characterized by high morning levels that gradually decline across the course of the day [6]. Repeated sampling of cortisol across the day may be more beneficial in the identification of HPA axis dysfunction in obesity, as such measurements can be considered both independently (e.g., morning, afternoon, and evening levels) and as aggregate measures (e.g., slope) of daily HPA axis function and integrity. Although not unequivocal [7,8], studies of diurnal cortisol in adults suggest that overweight and obese individuals may display blunted diurnal HPA axis functioning as demonstrated by numerous indices, including decreased cortisol variability [9,10], lower morning levels, and less change in cortisol throughout the day [11–13]. Furthermore, persistently atypical diurnal patterns often reflect the impact of chronic problems or disease states, such as those associated with obesity, rather than more temporary perturbations. Thus, measuring diurnal cortisol multiple times across development may be particularly useful for understanding cortisol–obesity associations. Although studies examining concurrent associations between diurnal cortisol and obesity in adulthood support the notion of HPA dysfunction in obesity, they provide little information about the development of such associations.

While a few cross-sectional studies of youth examine the relationship between more time-limited measures of cortisol and obesity [14–17] these reveal divergent results and do not allow any determination of causality or interpretation of temporal sequencing. As such, it is possible that researchers were detecting short-term associations that would correct over time. Except for two studies examining cortisol changes across puberty [18,19], which considered obesity secondarily, the association between diurnal cortisol and obesity in adolescence has remained largely unexplored. The lack of longitudinal studies leaves it unclear whether associations between diurnal cortisol and obesity change across development. This is plausible as the physiological changes and/or psychosocial stresses associated with puberty may affect the cortisol–obesity association. The present study addresses these issues, employing a prospective, longitudinal design and including multiple measures of diurnal cortisol and obesity across adolescence with the goal of illuminating both concurrent and longitudinal cortisol–BMI associations. We anticipate that, at each assessment point (ages 11, 13, and 15 years), cortisol will be associated with concurrent BMI. We also anticipate that cortisol assessed at each of these three time points, as well as stable patterns of cortisol over this time period, will longitudinally predict BMI at age 18 years. Due to insufficient prior evidence regarding associations between diurnal cortisol and BMI in adolescence, we take an exploratory approach to the direction of the effects in these analyses.

The present study also considers a number of other pertinent variables that may help to explain previous null associations in the adolescent obesity literature [18,19]. Numerous variables have been implicated in the association between cortisol and obesity or independently linked to both variables; therefore, it is important to establish that associations between cortisol and obesity are not a function of an underlying third variable, such as sex

[14], socioeconomic status (SES) [20,21], pubertal status [22,23], or mental health [14,24,25]. Although maternal BMI has only been associated with child BMI [26], it may serve as a proxy for genetic or environmental influences that account for changes in offspring's cortisol levels and BMI. We anticipate that adolescents with low SES, more advanced pubertal status, and elevated symptoms of mental health will demonstrate more elevated BMI at a bivariate level but that cortisol–obesity associations will persist after accounting for confounding variables.

Methods

Participants

Pregnant women and their partners/husbands were recruited at healthcare clinics in Milwaukee (80%) and Madison (20%), Wisconsin. Due to the original focus of the project, female participants were included in the study based on the following criteria: (a) over age 18; (b) living with the biological father; (c) at least one member of the couple working for pay; (d) not a student; and (e) not unemployed. Approximately 25% refused or were screened out based on exclusion criteria (see [27] for more details), yielding a sample of 560 individuals. Families were followed repeatedly over time and, of the initial eligible families, 346 adolescent participants (62%) provided saliva samples and measurements of height and weight for at least one assessment between early and mid-adolescence, which made them eligible for the present analyses. Interviews and/or questionnaires were completed by mothers, adolescents, and adolescent's teachers at ages 11, 13, and 15. At recruitment the sample was largely Caucasian and well-educated (mothers and fathers: 1% < high school degree). Annual family income ranged from \$7,500 to over \$200,000 ($Mdn = \$47,000$). The only significant difference between the 346 participating families and non-participants on demographic variables was that participating fathers were slightly older: $M = 31.7$ ($SD = 5.21$) versus $M = 30.7$ ($SD = 4.76$), $t(548) = -2.40$, $p < .05$. Parents and teachers gave informed consent at each time point; child assent was obtained beginning at age 11 years. All procedures were reviewed and approved by the University of Wisconsin Institutional Review Board. All participants were allowed to opt out at any time; however, this only happened with a small number of participating families/teachers (0.3–1.6%).

Measures

Salivary Cortisol—When participants were 11, 13, and 15 years old, they collected saliva samples at home for three days across three specific target collection times set by participants with research staff to match study needs and participants' individual schedules: (1) shortly after waking; (2) a time between 3:00 PM and 7:00 PM; and (3) a time just before bed. Average times of actual sample collection were 8:53 am ($SD = 82$ min), 4:50 pm ($SD = 75$ min), and 9:54 pm ($SD = 63$ min). At each of these three assessment periods, adolescents were instructed to record pertinent time variables, collect samples prior to eating, and freeze samples immediately after collection. After three days of collection for that assessment period, research staff collected the samples and transported them to the laboratory where they were kept frozen until assayed. All cortisol samples were assayed in duplicate using well-established, salivary enzyme immunoassay kits (Salimetrics, State College, PA). Raw cortisol scores were log-transformed and extreme values were Winsorized to normalize distributions. One adolescent taking prednisone was omitted from the analyses for that assessment period due to the possible effect of oral steroids on cortisol levels [28]. Mean intra-assay and inter-assay coefficients of variation for the assays were 3.8% and 7.4%, respectively, and the detection sensitivity limit was .02 $\mu\text{g/dL}$.

Adolescent Body Mass Index (BMI)—At ages 11, 13, and 15, researchers measured height and weight for most participants at home visits. At each assessment, participants

stood on a hard floor against a wall, and a level and tape measure were used to measure height to the nearest ¼ inch. This was repeated until two measurements were obtained within ¼ inch; the two closest readings were averaged. Weight to the nearest ½ pound was measured with a Health o meter EVERWeigh Lithium Electronic Scale (Sunbeam Health Division, Bridgeview, Illinois) until two readings within ½ pound were obtained; the two closest readings were averaged. At age 18 for all participants, and at earlier ages for the minority of participants who did not complete home visits (typically because participants lived outside a 2-hour radius of project offices; $n = 32, 50$, and 60 at ages 11, 13, and 15, respectively), detailed instructions were mailed to families directing them how to obtain measures of height and weight. At age 15, both self-measurements and later home visit assessments of height and weight were separately obtained for a subgroup of participants ($n = 252$); the resulting BMI scores were very highly correlated, ($r = .97, p < .001$), indicating that participants provided extremely accurate measurements. Consistent with other studies of community samples (e.g. [18,19]), adolescents' BMI scores were calculated by dividing body weight in kilograms by the square of height in meters (kg/m^2) and adjusted for age and sex using nationally representative data from the U.S.; and BMI categories were defined as lower weight (up to 15th percentile), normal weight (15th–85th percentile), overweight (85th–95th percentile) and obese (95th percentile and above) [29].

Control Variables

Pubertal Status—A multi-method, multi-informant approach was used to capture pubertal development [30]. Mothers and youth completed a self-administered Tanner stage measure based on description and visual inspection of line drawings [31]; mothers also completed the Pubertal Development Scale (PDS; [32]). As in previous studies [33], in order to provide the most accurate measure of total pubertal development, mothers' and youths' ratings of pubertal development were averaged when adolescents were 11 and 13 years old; however, only youths' report of development was utilized at age 15 due to low parent-child concordance. Five missing values were substituted with the mean for their sex.

Maternal BMI—Self-report of mothers' height and weight was obtained when their children were 13 years old. Mothers' BMI was computed by dividing body weight in kilograms by the square of height in meters (kg/m^2). Mean replacement was used for the 13 participants whose mothers did not report height and/or weight.

Other control variables—Adolescent mental health symptoms were assessed by parent, child, and teacher report on the MacArthur Health and Behavior Questionnaire that tapped both internalizing (e.g., depression, anxiety) and externalizing (e.g., conduct problems). Family SES comprised parental education levels and annual household income. Subsequent findings revealed SES and mental health variables were not significant predictors of BMI and therefore are not discussed at length.

Data Analytic Strategy

Hierarchical linear modeling (HLM) utilized the multiple cortisol samples within an individual to produce measures of predicted morning, afternoon, and evening levels of cortisol and change in cortisol across the day. In all HLMs, cortisol was modeled as a function of an intercept and time variables that were centered on a specific time of day (e.g. time since waking sample, time since afternoon sample, time since evening sample) and associated quadratic and cubic functions of time to account for linear and curvilinear patterns of diurnal cortisol. Using a three-level HLM that included random effects representing between-individual (level 3) variation in the intercept and linear slope, and day-to-day (level 2) variation in the intercept, the HLM was fitted to cortisol values for a given assessment period and estimated intercept (i.e. morning, afternoon, or evening) and change

(i.e. slope) values were extracted for each individual. While associations between all cortisol measures at a given assessment and BMI are presented in the results section, such cortisol measures are inherently related and thus reflect a joint phenomenon rather than independent effects. A similar model was used to extract estimated intercept and slope values for individuals' cortisol *across* time periods, however this three-level HLM included random effects representing between-individual (level 3) variation in the intercept and linear slope, and between-time-period (level 2) variation in the intercept, as well as terms modeling the effect of age on cortisol levels and slopes. The resulting assessment-period-specific or across-assessment ("stable") estimates of cortisol levels and slope were then used as predictors of BMI in regression models.

Descriptive statistics were compiled for all variables, and Pearson correlations were used to investigate bivariate associations of cortisol with BMI scores as well as the other variables. Independent samples *t*-tests were used to examine potential sex differences in BMI scores across adolescence; however, no significant differences emerged. Multiple linear regressions were used to test the concurrent and longitudinal models of primary interest. Initial models included sex, family SES, maternal BMI, adolescent mental health, pubertal development, the cortisol measure of interest, and a sex by cortisol interaction term. Only significant control variables were retained to make models more parsimonious and sex by cortisol interactions were dropped since none were significant. In longitudinal analyses, BMI scores concurrent with the cortisol measure being examined was included in the model to determine if cortisol predicted change in BMI. Lastly, given that previous research has found non-linear associations between cortisol and BMI [12], multinomial logistic regressions were computed to examine if cortisol-BMI associations differed according to BMI category in a non-linear fashion.

Results

Descriptive Statistics

Descriptive statistics are presented in Table 1. There were no differences between boys and girls in terms of age- and gender-normed BMI scores ($p = .33 - .98$). At each assessment between 20–30% of the sample was categorized as overweight or obese for their age and sex (age 11: 29%; age 13: 29%; age 15: 28%; age 18: 24%). See Table 2 for a more thorough description of categories of obesity at each assessment.

Bivariate Associations

BMI scores were quite stable across adolescence with correlations ranging from .72 to .90 ($ps < .001$), with adjacent assessments demonstrating the strongest associations. Adolescents with more advanced pubertal development, whose mother had a high BMI, or were from low SES backgrounds exhibited higher BMI scores across adolescence ($r = .19 - .32$, $p < .01$; $r = .31 - .33$, $p < .001$; $r = .11 - .16$, $p < .10$, respectively). Mental health severity scores at ages 11, 13, and 15 were not significantly associated with any assessment of BMI; however, there was a trend-level association between age 13 BMI and mental health at age 15 ($r = .11$, $p = .06$).

As shown in Table 3, cortisol–BMI associations varied over time. Overall, adolescents with lower morning levels and flattened slopes at age 11, as well as those with similar patterns that persisted longitudinally, demonstrated higher BMI scores across adolescence. In addition, there were sex differences in level of cortisol, although associations changed over time. Further, only pubertal status at age 13 was associated with morning, afternoon, evening and slope of cortisol, possibly due to the more restricted variability in pubertal status at the other ages.

Multivariate Analyses Examining Predictors of BMI

Both concurrent and longitudinal associations of cortisol and BMI scores were investigated with linear regression models. First, results revealed significant main effects of cortisol on concurrent BMI, especially at age 11 (see Table 4). Specifically, at age 11, lower levels of morning cortisol and flatter slopes were associated with increased concurrent BMI; and lower levels of morning cortisol were also associated with increased concurrent BMI at age 15. Second, longitudinal associations between cortisol at ages 11, 13, and 15 and later BMI at age 18 were examined (see Table 5). Overall, the pattern of results was the same as for the concurrent associations. In addition, results examining longitudinal cortisol (i.e., the portion of cortisol that remains stable from age 11 to age 15) revealed that individuals who displayed persistently lower levels of morning, afternoon, and evening cortisol, as well as persistently flatter slopes were more likely to have higher BMI scores at age 18. Third, to determine if cortisol predicted *change* in BMI, additional longitudinal models of age 18 BMI were run including the earlier measure of BMI that was concurrent with cortisol, but no significant associations were found.

Finally, multinomial logistic regressions were conducted to examine if cortisol-BMI associations differed according to BMI *category* in a non-linear fashion. The normal weight group was used as the reference category. Results suggested the findings for BMI scores also held at the extreme ends of the continuum (e.g. stable morning cortisol- *lower weight* log odds: $B=1.62$; 95% confidence interval (CI) =1.03, 2.55; *overweight* log odds: $B=1.15$; 95% CI = .80, 1.65; and *obese* log odds: $B=0.63$; 95% CI =0.37, 1.01; stable cortisol slope- *lower weight* log odds: $B=0.65$; 95% CI =0.41, 1.03; *overweight* log odds: $B=1.14$; 95% CI = 0.79, 1.66; and *obese* log odds: $B=1.64$; 95% CI = 1.05, 2.56).

Discussion

The current study examined whether diurnal cortisol was associated with adolescent BMI. Concurrent associations between diurnal HPA axis hypoactivity and increased BMI in adolescents parallel findings of diurnal cortisol and obesity in adults [9–11]. However, the present study is the first to demonstrate longitudinal associations of this finding in any age group. Furthermore, this is the first study to examine associations between stable, trait-like components of cortisol and BMI, revealing that persistently lower morning, afternoon, and evening levels, as well as flatter slopes, from ages 11 to 15 years were associated with higher BMI at age 18. Additional analyses using BMI categories revealed that findings may be extended beyond BMI scores to predictions of obesity.

Consistent with research on adult obesity [9–11], the present study identified patterns of HPA hypoactivity coinciding with increased BMI in adolescents as young as 11 years old. This suggests the adrenocortical underpinnings associated with obesity are established much earlier in life than previously thought. More than simply reflecting commonalities in cortisol-BMI associations for adolescents and adults, blunted diurnal cortisol profiles may indicate a vulnerability to gaining weight or may reflect neuroendocrine alterations that occur as a function of obesity.

Interestingly, the association between cortisol and BMI appears to be developmentally influenced as measures of cortisol in early adolescence seem to be the most salient predictors of concurrent, and later, BMI. The cortisol levels children possess as they come into adolescence, and which are stable across adolescence, may reflect a more biologically-driven predisposition to becoming heavier than cortisol levels at specific time points in adolescence which are likely to also include epoch-specific stresses inherent in this developmental period. A developmentally influenced change in the association between cortisol and BMI in early- to mid-adolescence may exist as the physiological and

psychological stressors inherent to puberty may introduce variance into cortisol levels that is not relevant to obesity. Research should probe such processes to gain a more comprehensive understanding of the developmental association between cortisol and obesity across the lifespan.

Previous authors have suggested that the blunted patterns of HPA axis activity found in overweight individuals may be indicative of HPA axis down-regulation [10,11]. While low cortisol levels do not necessarily prove HPA axis down-regulation, physiological processes support down-regulation in the case of obesity. Compared to their average weight counterparts, overweight and obese individuals often demonstrate increased cortisol reactivity [14,15,34], which can exert deleterious effects on the brain [35]. Following sustained exposure to elevated levels of cortisol, the HPA axis may down-regulate, possibly as a protective mechanism, resulting in decreased overall cortisol production [36]. While HPA axis down-regulation following persistent HPA axis stimulation has only been observed in non-human animals [37], naturalistic investigations of humans experiencing prolonged mental health problems reveal that these individuals also display blunted diurnal cortisol patterns [24,25], suggesting a similar process may be present in obese individuals.

While only speculative, blunted HPA functioning may reflect a psychophysiological mechanism that contributes to the persistence of obesity. Blunted diurnal cortisol predicted BMI both concurrently and longitudinally, but not changes in BMI across adolescence. When considered within the larger literature examining the continuity of obesity from childhood to adulthood [5] and the presence of blunted levels of cortisol in obese adults, it appears that obesity is associated with HPA axis hypoactivity from early adolescence on. However, the exact pathway through which lower levels of cortisol are associated with obesity remains to be determined.

Limitations

These novel findings must be considered in light of some potential limitations. It is possible that there is inherent sampling bias as the initial sample of mothers was largely Caucasian and drawn from two large cities in a single Midwestern state. Replication of these findings in a more diverse sample is needed. Additionally, the present study only examined diurnal cortisol, which provides a fairly limited view of the physiological processes associated with obesity. Research suggests that overweight/obese individuals are in a state of chronic, low-degree systemic inflammation [38]. Given the substantial cross-talk between HPA axis and immune system [39], future research should examine w interactions between these two systems independently and interactively contribute to obesity.

In summary, the current study contributed to the literature by examining both concurrent and longitudinal associations between diurnal cortisol and BMI in adolescents. HPA axis hypoactivity was identified at a much earlier age than previously established. While the longitudinal nature of this project allowed us to examine temporal associations between cortisol and BMI, we are unable to establish causation, as childhood BMI and cortisol were not measured. Future research should consider examining HPA axis functioning and BMI beginning in early childhood to determine if blunted HPA axis activity occurs alongside, or as a function of, obesity.

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Glossary

BMI	Body Mass Index
HPA	hypothalamic-pituitary-adrenal

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Implications and Contributions

Consistent with concurrent research examining diurnal cortisol and obesity in adults, the current study identifies the presence of blunted cortisol in adolescent obesity. Findings reveal that blunted diurnal cortisol predicts higher BMI concurrently and longitudinally across adolescence from 11 years, suggesting this association is present much earlier than previously thought.

Table 1

Descriptive Statistics

Variable	n	Min	Max	Mean	S.D.
Sex	346	0	1	0.51	0.50
Family Socioeconomic Status	343	-1.99	3.44	0.02	0.96
Maternal BMI	346	16.44	61.99	26.07	6.00
Age 11 Pubertal Status	322	1.00	4.50	2.13	0.81
Age 13 Pubertal Status	330	1.00	5.00	3.47	0.95
Age 15 Pubertal Status	346	2.00	5.00	4.36	0.48
Age 11 Mental Health Symptoms	307	-1.58	3.05	0.02	0.90
Age 13 Mental Health Symptoms	303	-1.54	4.41	-0.00	0.85
Age 15 Mental Health Symptoms	292	-1.57	3.46	-0.00	0.96
Age 11 Morning Cortisol	297	1.17	2.81	1.22	0.02
Age 11 Cortisol Slope	297	-0.02	-0.01	-0.02	0.00
Age 13 Morning Cortisol	306	1.16	1.38	1.23	0.03
Age 13 Cortisol Slope	306	-0.02	-0.01	-0.02	0.00
Age 15 Morning Cortisol	272	1.17	1.34	1.22	0.02
Age 15 Cortisol Slope	272	-0.02	-0.01	-0.01	0.01
Longitudinal Morning Cortisol	346	1.12	1.20	1.14	0.01
Longitudinal Cortisol Slope	346	-0.04	-0.01	-0.02	.00
Age 11 BMI	325	-2.36	2.81	0.34	1.03
Age 13 BMI	331	-2.38	2.69	0.42	1.01
Age 15 BMI	319	-3.46	2.64	0.42	0.97
Age 18 BMI	305	-2.64	2.63	0.31	1.05

Note: "Sex" is coded 0 = males, 1 = female; "Cortisol" = Predicted logged values ($\mu\text{g/dL}$); BMI = Body Mass Index. Adolescent BMI represents z-score for age and sex.

Table 2

Obesity Statistics Across Adolescence

Category	Age			
	11	13	15	18
	<i>n</i> (%)	<i>n</i> (%)	<i>n</i> (%)	<i>n</i> (%)
Lower weight (< 15 th percentile)	33 (10.2)	25 (7.6)	19 (6.0)	24 (7.9)
Normal weight (15 th – 85 th percentile)	198 (61.3)	210 (63.9)	210 (66.2)	207 (68.3)
Overweight (85 th – 95 th percentile)	49 (15.2)	49 (14.9)	59 (18.6)	39 (12.9)
Obese (> 95 th percentile)	43 (13.3)	45 (13.7)	29 (9.2)	33 (10.9)

Note: Age 11 *N*= 323; Age 13 *N*=329; Age 15 *N*= 317; Age 18 *N*=303

Percentiles were calculated using U.S. based norms for age and sex.

Table 3
Correlations between Cortisol, Control Variables, and Adolescent Body Mass Index (BMI)

	Age 11 Cortisol				Age 13 Cortisol				Age 15 Cortisol				Longitudinal Cortisol			
	Morn	Aft	Eve	Slope	Morn	Aft	Eve	Slope	Morn	Aft	Eve	Slope	Morn	Aft	Eve	Slope
Sex	.06	-.04	-.12**	-.15**	.14*	.09	.06	-.20**	.20**	.22**	.17**	-.07	.22**	.23**	.23**	-.24**
Family SES	.09	-.07	-.13*	-.18**	.14*	.09	.04	-.15*	.13*	.09	.09	-.06	.19**	.14**	.11*	-.19**
Age 13 Maternal BMI	-.00	.07	.09	.06	-.07	-.05	.04	.07	.11	.08	.06	-.08	-.01	-.00	.04	.03
Age 11 Pubertal Status	.04	.09	.05	-.02	.03	.07	.10	-.03	.08	.10	.06	-.04	.06	.12*	.14*	-.04
Age 13 Pubertal Status	.06	.08	.06	-.02	.16**	.18**	.19**	-.15**	.18**	.19**	.15**	-.07	.17**	.19**	.22**	-.12*
Age 15 Pubertal Status	-.03	-.00	.00	.03	.03	.06	.11	-.02	.02	.08	.08	.06	.00	.04	.06	-.04
Age 11 Mental Health	-.07	-.01	.06	.12*	.05	.05	.08	-.02	-.06	-.03	-.01	.05	-.06	-.05	-.03	.08
Age 13 Mental Health	-.02	.06	.11	.09	.02	.03	.09	-.01	-.05	-.03	-.00	.06	-.04	-.04	-.02	.06
Age 15 Mental Health	-.10	-.03	.04	.13*	-.02	-.02	.03	.04	-.01	.00	.00	-.00	-.07	-.07	-.03	.07
Age 11 BMI	-.17**	-.03	.05	.21**	-.03	-.01	.02	.04	-.09	-.04	-.02	.07	-.16**	-.12*	-.10	.13*
Age 13 BMI	-.15**	-.03	.06	.20**	-.05	-.02	.04	.08	-.09	-.05	-.03	.08	-.16**	-.13*	-.11	.13*
Age 15 BMI	-.15*	-.00	.10	.23**	-.06	-.04	.04	.08	-.10	-.06	-.06	.06	-.18**	-.14*	-.09	.17**
Age 18 BMI	-.14*	.02	.13*	.23**	-.06	-.05	.03	.07	-.08	-.03	-.03	.07	-.16**	-.13*	-.08	.16**

*
p < .05,

**
p < .01

Note: "Sex" is coded 0 = males, 1 = female; Adolescent BMI represents z-score for age and sex. "Cortisol" = Predicted logged values (μg/dL); Morn = Morning, Aft = Afternoon, Eve = Evening

Concurrent Associations between Diurnal Cortisol Measures and Adolescent Body Mass Index (BMI) Across Early to Mid-Adolescence

Table 4

	Age 11 Cortisol to Age 11 BMI				Age 13 Cortisol to Age 13 BMI				Age 15 Cortisol to Age 15 BMI			
	B(SE)	R ²	df	F	B(SE)	R ²	df	F	B(SE)	R ²	df	F
Sex	-.38 (.12)**				-.28 (.11)*				-.21 (.12) ^f			
Maternal BMI	.06 (.01)**				.05 (.01)**				.05 (.01)**			
Pubertal Status	.38 (.08)**				.35 (.06)**				.65 (.11)**			
Morning Cortisol	-.18 (.06)**	.22	288	20.11**	-.07 (.05)	.20	299	18.14**	-.12 (.06)*	.22	265	18.85**
Sex	-.42 (.13)**				-.28 (.11)*				-.22 (.12) ^f			
Maternal BMI	.06 (.01)**				.05 (.01)**				.05 (.01)**			
Pubertal Status	.39 (.08)**				.35 (.06)**				.67 (.11)**			
Afternoon Cortisol	-.09 (.06) ^f	.20	288	17.60**	-.06 (.05)	.19	299	17.99**	-.09 (.06)	.22	265	18.15**
Sex	-.41 (.13)**				-.29 (.11)*				-.23 (.12)*			
Maternal BMI	.06 (.01)**				.05 (.01)**				.05 (.01)**			
Pubertal Status	.37 (.08)**				.35 (.06)**				.67 (.11)**			
Evening Cortisol	-.01 (.06)	.19	288	16.75**	-.03 (.05)	.19	299	17.75**	-.09 (.06)	.22	265	18.10**
Sex	-.34 (.13)**				-.26 (.12)*				-.25 (.12)*			
Maternal BMI	.06 (.01)**				.05 (.01)**				.05 (.01)**			
Pubertal Status	.36 (.08)**				.35 (.06)**				.66 (.11)**			
Cortisol Slope	.18 (.05)**	.22	288	20.24**	.07 (.05)	.20	299	18.29**	.05 (.05)	.21	265	17.63**

^t
p < .10,

*
p < .05,

**
p < .01

Note: "Sex" is coded 0 = males, 1 = female; "Cortisol" = Predicted logged values (μg/dL)

Table 5
Longitudinal Associations between Diurnal Cortisol Measures and Age 18 Body Mass Index (BMI)

	Age 11 Cortisol to Age 18 BMI				Age 13 Cortisol to Age 18 BMI				Age 15 Cortisol to Age 18 BMI				Longitudinal Cortisol to Age 18 BMI			
	B(SE)	R ²	df	F	B(SE)	R ²	df	F	B(SE)	R ²	df	F	B(SE)	R ²	df	F
Sex	-.11 (.14)				-.10 (.13)				-.04 (.13)				.00 (.12)			
Maternal BMI	.05 (.01)**				.05 (.01)**				.06 (.01)**				.05 (.01)**			
Pubertal Status	.30 (.08)**				.33 (.07)**				.65 (.12)**				.65 (.11)**			
Morning Cortisol	-.15 (.06)*	.17	260	13.5**	-.08 (.06)	.18	272	15.1**	-.14 (.06)*	.22	255	18.1**	-.17 (.06)**	.23	300	22.1**
Sex	-.14 (.14)				-.11 (.13)				-.06 (.13)				-.00 (.12)			
Maternal BMI	.05 (.01)**				.05 (.01)**				.06 (.01)**				.05 (.01)**			
Pubertal Status	.30 (.08)**				.33 (.07)**				.67 (.12)**				.67 (.11)**			
Afternoon Cortisol	-.02 (.06)	.15	260	11.7**	-.09 (.06)	.18	272	15.2**	-.09 (.06)	.21	255	17.2**	-.15 (.06)**	.22	300	22.1**
Sex	-.11 (.14)				-.12 (.13)				-.07 (.13)				-.02 (.12)			
Maternal BMI	.05 (.01)**				.05 (.01)**				.06 (.01)**				.05 (.01)**			
Pubertal Status	.29 (.08)**				.33 (.07)**				.67 (.12)**				.65 (.11)**			
Evening Cortisol	.08 (.06)	.16	260	12.3**	-.04 (.06)	.18	272	14.7**	-.08 (.06)	.21	255	17.0**	-.12 (.06)**	.22	300	20.6**
Sex	-.04 (.14)				-.09 (.13)				-.08 (.13)				.00 (.12)			
Maternal BMI	.05 (.01)**				.05 (.01)**				.06 (.01)**				.05 (.01)**			
Pubertal Status	.27 (.08)**				.33 (.07)**				.65 (.13)**				.67 (.11)**			
Cortisol Slope	.22 (.06)**	.20	260	13.5**	.08 (.06)	.18	272	15.0**	.08 (.06)	.21	255	17.1**	.17 (.06)**	.23	300	22.3**

* $p < .05$,

** $p < .01$

Note: "Sex" is coded 0 = males, 1 = female; "Cortisol" = Predicted logged values ($\mu\text{g/dL}$)