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Reciprocal Relations Between Objectively Measured Sleep Patterns and Diurnal Cortisol Rhythms in Late Adolescents

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Abstract

Purpose—To examine how hours of sleep and wake times relate to between-person differences and day-to-day changes in diurnal cortisol rhythms in late adolescents

Methods—Older adolescents (N = 119) provided six cortisol samples (wakeup, +30min, + 2 hours, +8 hours, + 12 hours, and bedtime) on each of three consecutive days while wearing an actigraph. We examined how average (across 3 days) and day-to-day changes in hours of sleep and wake times related to diurnal cortisol patterns.

Results—On average, greater hours of sleep related steeper decline in cortisol across the days. Day-to-day analyses revealed that prior night's hours of sleep predicted steeper diurnal slopes the next day, while greater waking cortisol levels and steeper slopes predicted greater hours of sleep and a later wake time the next day.

Conclusions—Our results suggest a bidirectional relationship between sleep and HPA axis activity.

Keywords

Actigraphy; Sleep; HPA axis; Cortisol; Diurnal Rhythms; Adolescence; Naturalistic; Diary Studies

Adolescence is marked by a biological shift in sleep patterns often resulting in more "owl-like" patterns of sleep (e.g., later bed times, later wake times) [1–3]. Such changes often conflict, particularly in later adolescence, with the growing social demands many individuals face [2, 4]. Findings indicate that late adolescents report more erratic sleep schedules, later waking times, and less sleep than early adolescents [4]. Less than 30% of late adolescents get the recommended 8 or more hours of sleep per night and nearly 40% of individuals report consistent poor sleep quality [5,6]. Such sleep patterns have negative implications for individuals' outcomes including mood disorders [7], difficulties in school [8,9], and physical health (e.g., obesity[10]).

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Sleep patterns have also been known to play an important role in regulating other aspects of physiology, including the hypothalamic-pituitary-adrenal (HPA) axis. The HPA axis' main hormone, *cortisol*, exhibits a strong diurnal rhythm with levels high at waking, increasing further to a peak 30 minutes after waking (called the cortisol awakening response [CAR]) and then decreasing rapidly across the day, reaching nadir around midnight [11]. These changes in cortisol coincide with individuals' sleep cycles; low levels of cortisol are present during the first half of the night which is dominated by deep sleep [or slow wave sleep (SWS)], while increases in cortisol accompany the second half of the night which is dominated by rapid eye movement (REM) sleep (more wakeful period) [12,13].

This correspondence has been the topic of research, primarily among adults, revealing a bidirectional and complex relation between sleep and HPA axis functioning. That is, HPA axis functioning has been shown to influence sleep patterns [14,15] and changes in sleep patterns, specifically waketimes and hours of sleep, have been found to affect daytime cortisol rhythms [16, 17]. Surprisingly, however, the relation between sleep and HPA axis functioning has yet to be examined among adolescents. This is an important gap, given that adolescence is a critical developmental period in which physiological changes occur [18, 19] and sleep patterns are shifting (especially among late adolescents [20]), Further, recent research has linked diurnal cortisol functioning, specifically, flattened diurnal slopes across the *waking* day, to a variety of physical and mental health outcomes [21 – 24]. A better understanding of the relation of nighttime sleep and daytime cortisol rhythms may have important implications for our understanding of the development of a variety of disorders relating to *both* sleep and the HPA axis. In this study, we take an initial step in trying to understand associations between adolescent sleep and diurnal cortisol by examining how hours of sleep and wake times relate to late adolescents' waking cortisol values, CAR, and diurnal decline across the waking day. Utilizing multiple salivary cortisol samples over consecutive days, we first examined *between-person* differences by investigating how individuals' *average* (across days of sampling) hours of sleep and *average* wake times related to *average* cortisol rhythms. Then, to gain a better understanding of the reciprocal relation between sleep and HPA axis functioning, we investigated how *within-person* day-to-day variations in sleep patterns (i.e. wake time, hours of sleep) related to day-today variations in diurnal cortisol.

Method

Participants

Data for the current study come from a larger longitudinal study focused on adolescents from two diverse public high schools (one in the Midwest, one on the West Coast). For the larger study, participants high in neuroticism [25] were oversampled, resulting in 61% of the sample scoring in the top third of the neuroticism screener. Seventy-nine percent of the Midwest participants (n = 243) were randomly invited to participate in a longitudinal cortisol sampling protocol. Of those invited, 173 (71%) agreed to participate and completed Wave 1 cortisol sampling. The current study utilizes data from 152 adolescents who participated in Wave 2 of cortisol collection (approximately 1.5 years after Wave 1) at which point actigraph measures of sleep were added. Adolescents who were pregnant (n = 1), currently taking steroids (n = 7) or had missing data on study variables (n = 25) were excluded. This resulted in 119 adolescents (77% female). The greater proportion of females is accounted for by the fact that females on average report higher levels of neuroticism [26]. Of the current study's sample, 64.7% scored in the top third of the neuroticism screener.[1]

[1]Study variables were examined for differences by neuroticism risk score. Only differences in caffeine use and asthma medication emerged. High risk individuals reported greater caffeine use than low or medium risk individuals [F (2,116) = 3.04, p = .05], while low risk individuals were more likely to be taking asthma medication than the other two groups [χ^2 (2) = 9.02, p < .05].

Adolescents ranged in age from 17.93 to 20.10 years old (M = 19.01) and came from varying ethnic/racial backgrounds (see Table 1).

Procedures

Participants were sent a study packet that contained an actigraph [27], dairy booklets, a mechanical kitchen timer (to assist with the timing of 2nd sample), straws, vials, labels and a health/medical questionnaire. Study personnel contacted participants to review the study protocol. Participants provided six salivary cortisol samples and completed six diary entries per day for three consecutive typical weekdays. Participants wore an actigraph the night before starting the cortisol sampling and left it on until the morning after the last day of the study. Participants were paid $30 for completion of the sampling protocol and given a summary of basic sleep statistics across the 3 days of sampling. As approved by our Institutional Review Board, individual sleep data were used only for descriptive and hypothesis testing purposes, not for assessment or diagnosis of sleep disorders.

Measures

Salivary cortisol—Saliva samples were gathered each day for three days, at wake-up, 30 minutes after waking, at bedtime and three semi-random times throughout the day signaled by the actigraph watch (approximately 2, 8, and 12 hours post-awakening)[2]. Participants expelled saliva through a small straw into a 2 mL polypropylene tube and labeled tubes with the time and date. Participants were instructed not to eat, drink, or brush their teeth 30 minutes before sampling. Samples were returned by mail, refrigerated at −20 degrees Celsius, and then sent on dry ice by courier to Biochemisches Labor, Trier, Germany to be assayed for cortisol. Cortisol levels are stable at room temperature for several weeks and are unaffected by shipping [28]. Assays were conducted using a time-resolved immunoassay with fluorometric detection (DELFIA; see reference 29 for greater assay description). Intra-assay coefficients of variation (CVs) were between 4.0% and 6.7%, and inter-assay CVs ranged from 7.1% to 9.0%.

Objective sleep—During the 3-day salivary cortisol data collection, individuals wore the Actiwatch Score (Phillips Respironics, Inc.), a wrist-based accelerometer placed on the non-dominant hand that quantifies movement across the waking day and during sleep. To score data, the Actiware-Sleep software (version 3.4) validated algorithm was used [30][3]. Utilizing one minute epochs and based on significant movement after at least 10 minutes of inactivity, this algorithm calculates a variety of sleep parameters that include sleep end (wake time) and hours of sleep (sleep time excluding all periods of wakefulness during the total sleep period)[31]. Actigraph sleep estimates have been validated against concurrent polysomnography [32]. Aggregate sleep parameters were created by averaging individuals' sleep parameters across the 3 sampling days; sleep parameters for each day were used in the day-to-day analyses.

Diary and health variables—Using paper and pencil dairies, adolescents reported on sleep and health behaviors for each day of cortisol sampling. Specifically, adolescents reported their waking time and previous night's bedtime as well as any duration of time spent in a nap during the day. Adolescents also completed diary entries with *each* saliva sample, reporting whether they consumed caffeinated drinks, cigarettes, alcohol, medication

[2]Participants were asked for their typical waketime for each day of the study and watches were programmed to signal 2, 8, and 12 hours from this time (day 2). Thirty minute variations were added for day 1 and day 3 to make signals less expected [i.e., day 1 participants took samples 30 min early (1.5, 7.5, and 11.5 hours from waking) and day 3 was 30 min later (2.5, 8.5, 12.5 hours from waking)].

[3]Activity counts (A) within each epoch were calculated based on activity levels during the adjacent 2-min period using the following algorithm: $A = E - 2(1/25) + E - 1(1/5) + E + E + 1(1/5) + E + 2(1/25)$. Threshold set to 40, with a range of 20 to 80.

or exercised within an hour of each sample. Youth also reported in a health questionnaire if they were taking birth control (females only) or anti-depressant medication.

Analytic Plan

A 3-level multilevel growth-curve analysis was utilized to account for the nested nature of our data [33,34]. This modeling controls for nonindependence associated with nesting and allows for levels of cortisol to be predicted by moment-level variables (Level 1), day-varying variables (Level 2), and individual-level variables (Level 3). In line with previous studies [34, 35], day level variables were added, both lagged (minus 1 day) and non-lagged (night after cortisol collection) at Level 2 to model, simultaneously, the effect of prior night sleep parameters on next-day cortisol, and the effect of cortisol on sleep later that evening. As recommended [36], Level 2 variables were centered within cluster (CWC) and Level 3 variables were centered at the grand mean (CGM). At Level 1, time was centered as hours since waking (e.g., waking = 0).

We first modeled the latent estimates of the parameters defining each individual's diurnal cortisol rhythm. Next, we entered individuals' 3-day average sleep parameters (i.e., wake time, hours of sleep) at Level 3 to examine their associations with average cortisol levels (See Equation 1, Table 2). Cortisol values are predicted by the time of each sample, scaled as hours since waking each day, such that the β_{000} (the intercept) reflects the average wakeup cortisol level across individuals, β_{100} reflects individuals' average CAR[4] and β_{200} reflects the average linear slope of participants' diurnal cortisol rhythms[5]. Coefficients β_{001}, β_{101}, and β_{201} reflect the effect of average sleep parameters on average wakeup cortisol level, average CAR and average linear slope, respectively. Average wake time and sleep were entered in separate models, followed by an analysis that included both.

Finally, to understand the impact of changes in sleep parameters on day-to-day changes in cortisol rhythms *within* individuals, we examined the relation of sleep parameters the night before and after cortisol sampling to diurnal cortisol profiles each day by entering day level sleep parameters at Level 2 (See Equation 2, Table 2). For wake time, the *sleep parameter before cortisol* refers to the wake time in the morning before cortisol sampling that day, while *sleep parameter after cortisol* refers to wake time the morning after cortisol sampling. For hours of sleep, the *sleep parameter before cortisol* refers to the previous night's hours of sleep before the start of the next day's cortisol sampling, while the *sleep parameter after cortisol* refers to the hours of sleep after that day's cortisol sampling. Coefficients γ_{01j}, γ_{11j} and γ_{21j} reflect the relation of the sleep parameter the night before cortisol collection to subsequent day's wakeup cortisol level, CAR, and linear slope, respectively, while coefficients γ_{02j}, γ_{12j} and γ_{22j} reflect the relation of wakeup cortisol, CAR and linear slope, respectively to the sleep parameters taken that night, after cortisol sampling each day. Wake time and average hours of sleep were entered first separately and then simultaneously in the final model. For all analyses, adolescents' diary reports of their caffeine, nicotine, and alcohol consumption, exercise, and medication use within the hour prior to each sample were entered as covariates at Level 1 while birth control, asthma, depression medication, and daytime sleep (naps) were entered as covariates at Level 3.

[4]Individuals' 2nd sample (30 minutes after waking) was dummy coded where 1 = 2nd cortisol sample and 0 = all other samples.
[5]The current analysis did not include a quadratic time variable because the inclusion of this term in the unconditional growth model did not improve our model fit ([$\chi^2\Delta$ (1) = .78, p = .37]). Given this, the slope can be interpreted as a linear decline across the day rather than the linear decline at waking.

Results

See Table 1 for descriptive information. Cortisol values are presented in original μg/dL units in Table 1; because they were skewed (2.48) and kurtotic (9.57), natural log transformed values were used in all analyses. In line with recommendations [37], cortisol values were top coded at 1.80 μg/dl (equivalent to 50 nmol/L). The base model revealed the expected diurnal patterns; high values upon awakening ($\beta_{000} = -1.40$, $SE = .06$, $p < .0001$; equivant to .25 μg/dl.), a strong increase (68%) in levels in the first 30 minutes (CAR; $\beta_{100} = .52$, SE = .04, p < .0001), and approximately an 8% per hour decline in cortisol levels from wake up to bedtime[6] ($\beta_{200} = -.09$, SE = .01, $p < .0001$).

Between-Individual Analysis

As seen in Table 3, average wake time did not relate to average diurnal cortisol profiles (Model 1). Average hours of sleep (Model 2), however, related to rate of decline in cortisol; greater hours of sleep related a steeper decline in cortisol across the day (1.1% steeper slope for every additional hour of sleep). These effects were maintained even after controlling for wake time (Model 3).

Within-Individual Analysis

As seen in Table 4 (Model 1), when relating changes in sleep to changes in cortisol rhythms within individuals across multiple days, same day wake time was a significant predictor of subsequent wakeup cortisol values, CAR, and decline in cortisol. That is, adolescents who woke up later that day had higher wakeup cortisol, compared to days they woke up earlier (9.5% increase in morning cortisol for every 1 hour later wake time), a less pronounced CAR (10.5 % decrease in CAR for every 1 hour increase in wake time) and steeper decline in cortisol (1.3% steeper slopes for every 1 hour increase in wake time) across the day. In Model 2, prior nights' hours of sleep predicted wakeup cortisol, CAR, and decline in cortisol; greater hours of sleep the night before predicted higher wakeup cortisol (15.5% increase for every hour increase in sleep), a lower CAR (10.4% decrease for every hour increase in sleep), and steeper decline in cortisol (1.3% steeper slope for every hour increase in sleep) the next day. Further, greater levels of wakeup cortisol predicted greater hours of sleep the next day.

Results examining wake time and hours of sleep together (Model 3) revealed that prior and next day hours of sleep both predicted wakeup cortisol levels; greater hours of sleep the night prior predicted greater wakeup cortisol the next day, whereas greater wakeup cortisol that day related to greater hours of sleep that night (controlling for prior night hours of sleep). For diurnal slopes, a similar bidirectional relation emerged. Specifically, prior and next day hours of sleep both predicted steeper decline in cortisol slopes; greater hours of sleep the night before predicted a steeper decline in cortisol the subsequent day, and steeper slopes that day related to greater hours of sleep that night. Finally, next day wake time related to diurnal slopes, suggesting that flatter diurnal slopes that day predicted later waking times the following day.

Discussion

The current study took an important first step in understanding how typical hours of sleep and wake times relate to cortisol diurnal rhythms and how late adolescents' day-to-day variation in sleep relates to day-to-day variation in cortisol. Our findings suggest that across

[6]Coefficients can be interpreted as the % change in outcome (cortisol) per unit change in the independent variable using the following formula $\beta_{\%change} = [\exp(\beta_{raw})] - 1$ (Neter et al., 1990; Woolridge, 2000).

individuals, *average* hours of sleep are a steeper diurnal decline in cortisol across the day, even after accounting for adolescents' average wake times. These results are consistent with findings among adults [16] suggesting that individuals who sleep more have diurnal cortisol rhythms characterized by a steeper decline across the day and align with health findings suggesting that such patterns are considered normative and healthy [38]. The potentially bidirectional interrelations between sleep and the HPA axis [12], however, make it difficult to know if hours of sleep are impacting changes in diurnal rhythms or if diurnal rhythms are impacting hours of sleep.

A better understanding of causal directionality is gained from our day-to-day within-person examination. Findings suggest that on nights adolescents slept more, they woke up the next morning with higher waking cortisol levels and exhibited a steeper decline in their cortisol across the day. Further, on days adolescents had higher wakeup cortisol values and a steeper diurnal decline in their cortisol, they experienced greater hours of sleep that night and tended to wake up earlier the following day. An alternate way of viewing these day-to-day changes is that on days adolescents' slept less, they woke up the following morning with lower waking cortisol levels and had flatter cortisol slopes that day. On days that adolescents had lower wakeup values and flatter slopes, they subsequently experienced less hours of sleep that night and woke up later the next day. It is important to emphasize that these day-to-day analyses examine within-person changes in sleep patterns and diurnal cortisol from one day to the next, rather than trait or between-person differences. As such, these analyses are less subject to third variable explanations, such as the possibility that stable genetic or personality factors determine both sleep timing and cortisol patterns.

Together, our findings suggest a bidirectional relation between sleep and the HPA axis. While the exact mechanisms underlying the relations between cortisol and the HPA axis are not fully known, our findings could be linked with the correspondence of sleep cycles and cortisol secretion; deeper sleep accompanies the quiescent period of the HPA axis, while greater cortisol output has been found during the later stages of sleep (dominated by REM sleep) [12,13]. Individuals who sleep less and wake earlier might have less time to "ramp up" their cortisol levels (which happen during REM sleep) and in turn end up with lower awakening cortisol and flatter slopes than individuals who slept more. Further, guided by previous laboratory findings that suggest that higher levels of cortisol have the ability to suppress SWS leading to subsequent sleep deprivation [39,40], individuals with flatter slopes (partially due to higher evening levels[7]) might have a difficult time falling asleep or staying asleep leading to fewer total hours of sleep. In an attempt to recover from sleep loss during the night, individuals then sleep later the next morning. Given that the specific sleep stages cannot be examined using actigraphy and we do not have overnight cortisol samples, these explanations remain speculative.

In sum, our study is the first to examine these relations in adolescents using an objective sleep measure and examining within-individual covariation between sleep and cortisol in a naturalistic setting. Further, our study examines these relations during a sensitive and unique developmental period in which changes in physiology and sleep patterns are especially relevant. [19, 20]. Our findings suggest that the choices made about sleep during this time may have consequences for physiology, and in turn, physiology (the HPA axis) attempts to adapt to late adolescents' changing daily schedules. Despite these contributions, however, it is important to note several limitations of our study. First, while our findings suggest that sleep relates to fluctuation in diurnal cortisol, the changes were relatively small (1–2%

[7]Time was centered at bedtime to examine if flatter slopes were partially due to higher evening levels in cortisol. Findings revealed that levels were trending [$\gamma 02\ j = -.079$, p = .18]; flatter lines were associated with less prior nights sleep partially due to higher evening cortisol levels.

change for diurnal slopes and 9–16% changes in waking cortisol). Second, we examined a limited number of days in our examination of day-to-day changes and might not be fully capturing the complexities of sleep and its relations to HPA axis functioning over a longer period of time. Third, we did not use electronic monitoring devices to track the exact timing of cortisol sampling; we are reliant on participant self-report of sampling timings. Failure to time samples appropriately, however, would most likely lead to a reduction in the size of our effects, rather than systematic bias in our data. Finally, individuals with greater neuroticism and females were over represented in our sample. We found no differences in study variables by neuroticism risk; however, future research should examine whether associations between sleep and HPA axis functioning are similar across personality characteristics and gender. Despite these limitations, our study provides an important first step in understanding the complex and bidirectional relations between adolescent sleep and HPA axis functioning.

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Table 1

Descriptive statistics for study variables (N = 119)

Variable	%	n	Mean	SD	Range
Age of adolescent			19.01	0.42	17.93 – 20.10
ake time (self-report) hh:mm			08:38	1:28	03:13 – 13:30
Wake time (Actigraph) hh:mm			08:47	1:22	05:48 – 12:33
Hours of nighttime sleep (self-report)			7.71	1.34	3.00 – 10.61
Hours of nighttime sleep (Actigraph)			6.41	1.07	1.41 – 9.55
Sleep efficiency % (Actigraph)			81.85	7.61	48.80 – 92.60
Hours of naptime (self-report)			0.41	0.77	0.00 – 04.72
Average waketime cortisol (μg/dl)			0.30	0.21	0.01 – 1.37
Average +30 min cortisol (μg/dl)			0.50	0.25	0.01 – 1.78
Average + 3 hours cortisol (μg/dl)			0.28	0.20	0.03 – 1.80
Average + 8 hours cortisol (μg/dl)			0.16	0.11	0.03 – 0.82
Average + 12 hours cortisol (μg/dl)			0.12	0.11	0.01 – 0.76
Average bedtime cortisol (μg/dl)			0.10	0.11	0.01 – 0.71
Caffeine (drinks per day)			0.91	1.40	0.00 – 8.00
Nicotine (cigarettes per day)			0.28	0.82	0.00 – 5.00
Alcohol (drinks per week)			4.38	7.45	0.00 – 50.00
Exercise (hours per week)			2.77	4.07	0.00 – 30.00
Oral contraceptive use (females only)	35.5	33			
Asthma medication	7.6	9			
Depression medication	4.2	5			
Male	21.8	26			
European American	61.4	73			
African American	17.6	21			
Hispanic	4.2	5			
Multiple/other race	16.8	20			

Table 2

HLM 3-Level equations modeling diurnal cortisol.

$$\text{Level 1}: Cortisoltij = \pi_{0ij} + \pi_{1ij}(CAR) + \pi_{2ij}(Time\ Since\ Waking) + e_{tij}$$

$$\text{Level 2}: \pi_{0ij} = \gamma_{00j} + \zeta_{0ij}$$

$$\pi_{1ij} = \gamma_{10j} + \zeta_{1ij}$$

$$\pi_{2ij} = \gamma_{20j} + \zeta_{2ij} \qquad \text{Equation 1}$$

$$\text{Level 3}: \gamma_{00j} = \beta_{000} + \beta_{001}(Aggregate\,Sleep\ parameter) + r_{00j}$$

$$\gamma_{10j} = \beta_{100} + \beta_{101}(Aggregate\,Sleep\ parameter) + r_{10j}$$

$$\gamma_{20j} = \beta_{200} + \beta_{201}(Aggregate\,Sleep\ parameter) + r_{20j}$$

$$\text{Level 2}: \pi_{0ij} = \gamma_{00j} + \gamma_{01j}(Sleep\ Parameter\ before\ Cort) + \gamma_{02j}(Sleep\ Parameter\ after\ Cort) + \zeta_{0i}$$

$$\pi_{1ij} = \gamma_{10j} + \gamma_{11j}(Sleep\ Parameter\ before\ Cort) + \gamma_{12j}(Sleep\ Parameter\ after\ Cort) + \zeta_{1ij} \qquad \text{Equation 2}$$

$$\pi_{2ij} = \gamma_{20j} + \gamma_{21j}(Sleep\ Parameter\ before\ Cort) + \gamma_{22j}(Sleep\ Parameter\ after\ Cort) + \zeta_{2ij}$$

Table 3

Effects of average wake time and hours of sleep on diurnal cortisol. Level 1 (n = 1521), Level 2 (n = 285), Level 3 (n = 119)

	Model 1	Model 2	Model 3
Fixed effects	**Coefficient (SE)**	**Coefficient (SE)**	**Coefficient (SE)**
Wake up cortisol level			
Intercept	−1.423 (.059)***	−1.423 (.059)***	−1.424 (.060)***
Average wake time	−0.005 (.036)		−0.009 (.036)
Average hours of sleep		0.032 (.051)	0.036 (.052)
CAR			
Intercept	0.544 (.041)***	0.543 (.042)***	0.544 (.041)***
Average wake time	0.017 (.032)		0.020 (.032)
Average hours of sleep		−0.013 (.043)	−0.019 (.042)
Time since waking			
Intercept	−0.091 (.005)***	−0.092 (.005)***	−0.091 (.005)***
Average wake time	0.008 (.004)		0.010 (.004)*
Average hours of sleep		−0.009 (.005)*	−0.012 (.005)*

Note.

* $p \leq .05$,

** $p < .01$,

*** $p < .001$.

Race/ethnicity, gender, birth control, asthma medication, depression medication, and daytime sleep (naps) were added as covariates at Level 3. Caffeine use, nicotine use, and alcohol use, any medication and hours of exercise were added as covariates at Level 1.

Table 4

Effects of same day wake time and prior day hours of sleep on diurnal cortisol. Level 1 (n = 1521), Level 2 (n = 285), Level 3 (n = 119)

	Model 1	Model 2	Model 3
Fixed effects	**Coefficient (SE)**	**Coefficient (SE)**	**Coefficient (SE)**
Wake up cortisol level			
Intercept	−1.421 (.058)***	−1.422 (.060)***	−1.418 (.058)***
Same day wake time	0.091 (.036)*		−0.002 (.037)
Next day wake time	0.007 (.031)		−0.055 (.031)
Prior day hours of sleep		0.144 (.035)***	0.150 (.040)***
Next day hours of sleep		0.079 (.028)**	0.110 (.028)***
CAR			
Intercept	0.540 (.041)***	0.544 (.041)***	0.540 (.041)***
Same day wake time	−0.114 (.054)*		−0.056 (.059)
Next day wake time	−0.055 (.042)		−0.058 (.047)
Prior day hours of sleep		−0.110 (.043)*	−0.075 (.045)
Next day hours of sleep		−0.039 (.044)	0.001 (.048)
Time since waking			
Intercept	−0.092 (.005)***	−0.092 (.005)***	−0.092 (.005)***
Same day wake time	−0.013 (.004)**		− 0.009 (.005)
Next day wake time	0.007 (.004)		0.015 (.004)***
Prior day hours of sleep		−0.013 (.004)**	−0.010 (.004)*
Next day hours of sleep		−0.005 (.004)	−0.015 (.004)**

Note.

*$p < .05$,

**$p < .01$,

***$p < .001$.

Race/ethnicity, gender, birth control, asthma medication, depression medication were added as covariates at Level 3; Caffeine use, nicotine use, and alcohol use, any medication and hours of exercise were added as covariates at Level 1.