Positive and Negative Affect and Arousal: Cross-Sectional and Longitudinal Associations With Adolescent Cortisol Diurnal Rhythms

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ABSTRACT

Objectives: Psychobiological research with adolescent populations tends to focus on negative mood, stress, and psychopathology, but the role of positive emotions is insufficiently understood. The current study examines the relative contributions of both negative and positive affective experiences to the basal activity of the hypothalamic-pituitary-adrenal axis, measured by levels of cortisol across the waking day.

Methods: A sample of 315 ethnically and racially diverse high school students (mean age = 17.1 years, 73% female) completed a multiple-day naturalistic salivary cortisol protocol twice over a 5-year period. Along with each saliva sample, youth provided diary reports of their current mood states. Principal components analysis revealed four factors: high arousal positive affect (PA), low arousal PA, high arousal negative affect (NA), and low arousal NA.

Results: Multilevel growth curve models suggested that greater high arousal PA was associated with adaptive patterns of hypothalamic-pituitary-adrenal activity: steeper cortisol slope from waking to bedtime and lower evening cortisol, independent of NA. In addition, increases in high arousal PA over the 5-year follow-up period were associated with a steepening of the diurnal cortisol slope ($\beta = -0.038$, $p = .009$; negative values indicate the decrease of cortisol throughout the day) and lower evening cortisol levels ($\beta = -0.661$, $p = .027$) based on within-person fixed-effect regression analysis.

Conclusions: This study shows that high arousal PA, such as feeling alert and active, is associated with a steeper decline in cortisol throughout the day. Low arousal positive emotions did not display this relationship.

Key words: adolescent development, circumplex model of affect, diurnal cortisol, HPA axis.

INTRODUCTION

Psychobiological research tends to focus on negative mood, stress, and psychopathology, with relatively little emphasis on the role of positive emotions. This trend is evident in research on the hypothalamic-pituitary-adrenal (HPA) axis, one of the key biological stress systems thought to mediate the connections between psychological experiences and health outcomes (1). There is now convincing evidence that negative social and affective experiences such as anger, sadness, loneliness, depressed mood, and perceived stress are associated with dysregulated patterns of cortisol secretion (2–5), the major hormonal output of the HPA axis. However, much less is known about the role of positive affective experiences in relation to cortisol, especially during adolescence. Although traditionally described as a vulnerable period for stress and emotional turmoil, recent research and theory has now reframed adolescence as a time of heightened arousal and emotional intensity (6,7). Therefore, psychobiological approaches to adolescence would be enhanced by studying both negative and positive expressions of affect and arousal.

Cortisol follows a strong circadian rhythm. Typically, cortisol levels are high upon waking, increase to a peak approximately 30 to 45 minutes after waking (the cortisol awakening response, CAR), and then decline throughout the remainder of the day (8,9). The rate of decline in cortisol

CAR = cortisol awakening response, **HPA** = hypothalamic-pituitary-adrenal, **MDD** = major depressive disorder, **NA** = negative affect, **PA** = positive affect

SDC Supplemental Content

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from waking to bedtime (cortisol slope) represents an important marker of diurnal HPA activity, with flatter slopes indicating a slower decline in cortisol from waking to bedtime. There is empirical evidence that flattened diurnal cortisol slopes may be implicated in risk for diseases, including depression and heart disease, and faster progression of cancer (10–13). Therefore, diurnal changes in cortisol activity represent meaningful individual differences in stress biology that may play a role in future health (14).

Accumulating evidence from medical and social sciences suggests that negative affect (NA) and positive affect (PA) both make important and independent contributions to health outcomes, which underlines the need for a revised model of the role of affect in the stress process (15,16). The current evidence regarding PA and HPA activity remains limited and inconsistent. For instance, Steptoe and colleagues (17,18) found that PA (measured as happiness only, averaged for 2 days) was associated with lower total salivary cortisol across the day, independent of age, sex, socioeconomic status (SES), smoking, and NA. However, this finding has not been replicated across other adult samples (19,20) and has never been examined in an adolescent sample. Similarly, although there is some empirical support that PA is associated with a steeper cortisol slope in adulthood (21,22), other studies have failed to find a direct association between PA and cortisol slope (23–25) or even found that PA was associated with a flatter diurnal slope (19). None of the research to date has taken into account level of arousal, an important dimension introduced by the circumplex model of affect (26).

The circumplex model of affect suggests that every affective experience is the consequence of both valence and arousal, two independent neurophysiological systems that together can be used to represent a particular emotional experience (26,27). In this model, affect exists on two axes: one describing positive versus negative valence and the other describing high versus low activation. Therefore, the circumplex model allows the prediction of differential effects of high arousal PA (e.g., excited and active), low arousal PA (e.g., calm and placid), high arousal NA (e.g., fearful and hostile), and low arousal NA (e.g., drowsy and dull). This distinction across valence and arousal may be especially important when studying the physiology of the HPA axis during adolescence, a developmental period associated with alterations in both stress physiology as well as alterations in the neurobiology of affect and, in particular, the neurobiology of PA (28,29).

There are biologically based changes in neural systems of emotions at the beginning of adolescence, which contribute to a natural inclination toward strong emotions, replete with intensity and arousal (7). The impact of strong negative emotions on health, stress, and well-being has received significant attention, particularly the sharp rise in clinical depression and anxiety disorders during this developmental period (30,31) and the relationship between negative mood and HPA dysfunction (2,4,32). However, current research and theory on positive youth development underlines the equally important role of positive impacts (33,34). Beyond risk, adolescents seek excitement, thrills, and activity—positive feelings characterized by high arousal and intensity that youth experience in their daily lives (7). The current study examines the contribution of affect (positive and negative valence), activation (high and low arousal), and intensity (strong emotions) to adolescent HPA axis activity, both across individuals and within-individuals over time.

DESIGN AND METHODS

Participants

Data for the current study were collected as part of the Youth Emotion Project, a two-site longitudinal investigation examining risk factors for mood and anxiety disorders in late adolescence (35–40). Participants were drawn from two ethnically and socioeconomically diverse high schools in suburban Chicago and Los Angeles. Students in their junior year of high school were screened and selected to participate based on their neuroticism score from the Eysenck Personality Questionnaire–Revised (41), a personality variable that increases the risk for subsequent development of mood and anxiety disorders (42,43). Those scoring in the top third of this measure were oversampled, comprising 59% of the final group of 627 who consented to be part of the longitudinal study and completed the baseline assessment across three consecutive cohorts. Two-thirds of the participants were randomly selected for the cortisol study subsample. Of those invited, 350 youth (71%) agreed to participate and completed the cortisol task. Exclusion criteria for the current analyses were use of corticosteroid-based medications ($n = 11$) or antipsychotic medications ($n = 3$). In addition, 20 participants were excluded due to insufficient cortisol data (i.e., fewer than 50% of all samples and/or missing all wakeup or bedtime samples). The final analytic sample included 315 adolescents (230 were female) with a mean (standard deviation [SD]) age of 17.05 (0.38) years at baseline.

The greater proportion of females over males is accounted for by the fact that females are, on average, higher on the neuroticism personality trait (44). Although use of this high neuroticism sample limits generalizability, it also provides a larger range and more normal distribution of PA for both males and females, which usually has ceiling effects in the general population (i.e., positive moods are negatively skewed, with most peoples' responses clustering at the high end). We examined the differences between those in the top third of the neuroticism measure (i.e., high neuroticism risk, $n = 192$) compared with the rest of the sample on key demographic and cortisol parameters. Results revealed that youth in the high-risk group did not differ from the rest of the sample based on age, sex, racial/ethnic background, SES, waking cortisol, CAR, bedtime cortisol, or diurnal cortisol slope.

Procedure

As part of the larger investigation, all youth completed the Structured Clinical Interview for DSM-IV-TR (45), interviews for chronic and episodic stress, and a series of questionnaires to measure additional health and demographic characteristics every year. In addition, the cortisol study subsample participated in a diary study using a modified experience sampling method (ESM) (46) protocol six times a day for three consecutive weekdays (18 samples total). Adolescents were asked to provide saliva samples and diary reports at wakeup, 40 minutes after wakeup, and immediately before bedtime (to model the cortisol diurnal rhythm). In addition, adolescents wore wristwatches that beeped at semirandom moments to prompt three additional sampling times across the day (approximately 3, 8, and 12 hours after participants' typical wakeup times).

Along with every saliva sample, youth were asked to report, in paper diaries, where they were, who they were with, and what they were thinking and feeling at the time. Diary reports also asked youth specific questions regarding sleep and health-related behaviors occurring in the past hour that may influence cortisol levels. All procedures were reviewed and approved by institutional review boards at Northwestern University and the University of California at Los Angeles.

Measures

Cortisol

At each sampling point, participants expelled a small, passive drool saliva sample through a straw into a sterile 2-ml polypropylene cryogenic vial. They were asked to report the exact time of each sample and to store the sample in the refrigerator as soon as possible after completion. Participants were asked not to eat or drink in the 30 minutes before each sample and, for the unanticipated beeps, were asked to record whether or not they had consumed food or beverages in the hour before that beep. Completed samples (labeled with exact date and time) and all other study materials were returned in one packet through a school drop box or by regular postal mail. Once returned to the laboratory, samples were refrigerated at −20 °C until they were sent by courier on dry ice to Biochemisches Labor at the University of Trier, Germany, to be assayed.

Assays were conducted in duplicate using a time-resolved immunoassay with fluorometric detection (DELFIA) (47). Intra-assay coefficients of variation were between 4.0% and 6.7%, and interassay coefficients of variation ranged from 7.1% to 9.0%. To correct for a strong positive skew in the cortisol distribution, cortisol values were natural logarithmically transformed before analysis. The transformation substantially reduced the nonnormality of the data, and all analyses include robust standard errors, which can compensate for minor deviations from normality.

Positive and Negative Affect

We combined ESM measures across all 3 days of data collection as our measure of typical affective state. Aggregation of ESM measures is thought to provide a more valid estimate of typical affective states than questionnaire-based reporting of typical affect (48). Notably, in a study that examined both ESM and survey methods to capture PA, effects on neuroendocrine and cardiovascular activity were substantially stronger when PA was assessed by aggregating ESM samples than with questionnaire measures (25).

Participants were asked to indicate in their diary reports how much they felt each of the following mood states at the time of each sampling occasion: happy, nervous, lonely, active, frustrated, alert, worried, relaxed, irritable, stressed, sad, and cheerful. Each mood state was rated on a scale of 0 (not at all) to 3 (very much). Four affect scales were created based on a principal components analysis (PCA) of all diary-reported moods states, with orthogonal rotation. Principal components analysis weights were used to construct scales such that the resulting uncorrelated factors could be included in a single model. The four affect scales align with the circumplex model of affect (26,49): PA–high arousal (active, alert; $\rho = 0.700$), PA–low arousal (happy, cheerful, relaxed; $\alpha = .839$), NA–high arousal (nervous, frustrated, worried, irritable, stressed; $\alpha = .906$), and NA–low arousal (sad, lonely; $\rho = .802$). Each component had an eigenvalue > 1 and together explains 78% of the variance (Table S1, Supplemental Digital Content 1, http://links.lww.com/PSYMED/A204). We also created a quadratic version of each affect scale by squaring scale values, for use in modeling the impact of high-intensity moods (i.e., strong emotions).

Covariates

Basal cortisol levels may be influenced by a number of health and demographic factors including sleep, nicotine, oral contraceptive use, race/ethnicity, SES, and major depressive disorder (MDD) (38,50–54). Each of these factors was therefore measured and included as covariates in the final models. In the daily diaries, participants' recorded their wake time each day. Participants also responded to health and demographic questionnaires that assessed their typical hours of sleep, use of oral contraceptives (females only), nicotine use, race/ethnicity, and family SES (computed from parents' average level of education). Recent MDD (current or within the last 3 months) was assessed using the Structured Clinical Interview for DSM-IV-TR Axis I Disorders. Finally, we included an indicator for neuroticism risk, the sampling variable, which was measured by the Eysenck Personality Questionnaire–Revised.

Analytic Plan

Multilevel Growth Models

A three-level growth curve analysis was run to model the diurnal rhythm of cortisol, to examine person-level predictors of that rhythm, and to adjust for the nonindependence of observations associated with nested data. The model included momentary salivary cortisol samples (Level 1) nested within day (Level 2) and nested within person (Level 3). Based on prior work (2,55), three time of day indicators were included in Level 1 to capture the key elements of the cortisol diurnal rhythm: a dummy variable representing the CAR (to model the increase in cortisol from waking to 40 minutes after waking), time of day (to model the decline of cortisol across the day), and a time of day squared parameter (to model the curvilinear shape of the rhythm). Time was centered at waking and represented hours since waking, which allows the intercept to be interpreted as the waking value. In a separate set of models, time was recentered at bedtime, representing hours until bedtime, such that the intercept could be interpreted as bedtime cortisol level, and we could examine associations between our affect variables and bedtime cortisol levels.

Daily wake time was entered at Level 2 to adjust for any impact of time of waking on cortisol rhythms each day, and person-level predictors and covariates were entered at Level 3 to predict differences in basal cortisol rhythms estimated across all three study days. The affect factors (both linear and quadratic) were included in Level 3 to determine whether high and low arousal PA predicts wakeup cortisol, the CAR, cortisol slopes across the day, and bedtime cortisol levels. Importantly, high and low arousal NAs were entered into the model at the same time as the PA variables, to test the unique effect of each affect factor on cortisol parameters.

$$(1.1)\ \text{Level 1: Cort}_{tij} = \pi_{0ij} + \pi_{1ij}\text{CAR}_{tij} + \pi_{2ij}\text{Time of Day}_{tij} + \pi_{3ij}\text{Time of Day}^2{}_{tij} + e_{tij}$$

$$(1.2)\ \text{Level 2: } \pi_{0i} - \pi_{3i} = \beta_{00} + \beta_{01}\,\text{WakeTime} + \rho_{0i}$$

$$(1.3)\ \text{Level 3: } \beta_{00j} - \beta_{30j} = \gamma_{00j} + \gamma_{01j}\text{Demographic Covariates}_{0ij} + \gamma_{02j}\text{Health Covariates}_{0ij} + \gamma_{03j}\text{Affect} + \gamma_{04j}\text{Affect}^2 + \upsilon_{00j}$$

Longitudinal Models

All participants in the first wave of cortisol sampling (Time 1) were asked to complete another wave of cortisol sampling data collection using identical procedures 5 years after their baseline measures (Time 2). Therefore, a subset of youth ($n = 166$; 53%) provided ESM and cortisol data at two time points over 5 years. To bolster our cross-sectional findings with longitudinal models of within-person change, in addition to the multilevel growth models described earlier, we also measured within-person changes in cortisol (e.g., cortisol slope and CAR) as a function of within-person changes in high and low arousal PA and NA over the 5-year period, using an econometric approach called fixed-effects regression. The fixed-effect model for panel data uses the following equation:

$$(2.0)\ \Delta\text{Cort}_{it} = \beta_0 + \beta 1\,\Delta\text{PA}_{it} + \beta_2\Delta\text{NA}_{it} + \beta_3\Delta\text{Wake Time}_{it} + \beta_4\Delta\text{Sleep}_{it} + \beta_5\Delta\text{Health Behaviors}_{it} + \mu_i + \varepsilon_{it}$$

where Δ indicates the change in variables taking place between Time 1 and Time 2 and μ_i represents person-level characteristics that are constant

over time. Time-varying unexplained variation is represented by ε. In the estimation stage, all stable characteristics drop out of the model (i.e., a time-invariant characteristic cannot cause such a change because it is constant for each person). As a result, we can estimate the effect of PA and NA on cortisol only because affect changes over time.

This econometric model strengthens causal claims because it controls for the effects of time-invariant variables, reducing bias from potential selection effects driven by individual characteristics (56). In other words, the fixed-effects model controls for all time-invariant differences between the individuals, both observed (e.g., race/ethnicity or sex) and unobserved (e.g., culture or early childhood factors), so the estimated coefficients of the fixed-effects models are not biased because of omitted time-invariant characteristics. The only omitted variables that threaten a fixed-effect design are those variables that change within individuals over time. In an attempt to reduce this bias, our analyses included key time-varying predictors of cortisol including wake time, typical sleep duration, and nicotine use.

RESULTS

At Time 1, youth, on average (SD), woke up at 6:49 AM (6.81 minutes) and reported 7.15 (0.90) hours of sleep per night. Fourteen youth in the sample met the criteria for recent MDD, 17 students reported being a regular smoker,

TABLE 1. Descriptive Statistics for Sample, Presented by High and Low Arousal Positive Affect and Negative Affect

Characteristics	Overall (*n* = 315)	PA High Arousal (*n* = 79)	PA Low Arousal (*n* = 79)	NA High Arousal (*n* = 79)	NA Low Arousal (*n* = 80)
Male, *n* (%)	85 (27.0)	24 (30.4)	24 (30.4)	12 (15.2)	18 (22.)5
Race/Ethnicity, *n* (%)					
White	150 (47.6)	34 (43.0)	40 (50.6)	48 (60.8)	36 (45.0)
Black	33 (10.5)	11 (13.9)	10 (12.7)	7 (8.9)	10 (12.5)
Asian/Pacific Islander	18 (5.7)	5 (6.3)	2 (2.5)	2 (2.5)	4 (5.0)
Latino	59 (18.7)	19 (24.1)	17 (21.5)	6 (7.6)	9 (11.3)
Multiethnic/Multi-racial	38 (12.1)	8 (10.1)	8 (10.1)	11 (13.9)	12 (15.0)
Other race/ethnicity	17 (5.4)	2 (2.5)	2 (2.5)	5 (6.3)	9 (11.3)
Regular nicotine use, *n* (%)	17 (5.4)	1 (1.3)	2 (2.5)	4 (5.1)	6 (7.5)
Oral contraceptive use, *n* (%)	26 (8.3)	7 (8.9)	5 (6.3)	15 (19.0)	13 (16.3)
Average hours of sleep, M (SD)	7.150 (0.90)	7.125 (1.0)1	7.316 (0.94)	6.998 (0.83)	6.954 (0.93)
Neuroticism score, M (SD)	11.840 (61.0)	11.34 (4.44)	9.843 (5.18)	13.700 (3.22)	13.984 (2.95)
PA, M (SD)					
Active	1.045 (0.47)	1.599 (0.35)	1.372 (0.49)	.983 (0.42)	0.984 (0.45)
Alert	1.352 (0.61)	2.068 (0.31)	1.768 (0.53)	1.382 (0.54)	1.239 (0.61)
Happy	1.612 (0.56)	1.982 (0.51)	2.249 (0.34)	1.456 (0.41)	1.417 (0.48)
Cheerful	1.106 (0.62)	1.612 (0.59)	1.799 (0.49)	.972 (0.51)	0.966 (0.54)
Relaxed	1.432 (0.57)	1.714 (0.58)	2.052 (0.44)	1.241 (0.46)	1.217 (0.52)
NA, M (SD)					
Nervous	0.422 (0.47)	0.377 (0.45)	0.308 (0.43)	0.993 (0.53)	0.737 (0.62)
Frustrated	0.777 (0.51)	0.688 (0.55)	0.533 (0.43)	1.375 (0.40)	1.148 (0.53)
Worried	0.623 (0.54)	0.501 (0.53)	0.402 (0.49)	1.328 (0.44)	1.040 (0.59)
Irritable	0.705 (0.53)	0.593 (0.53)	0.494 (0.44)	1.294 (0.48)	1.055 (0.55)
Stressed	0.928 (0.68)	0.803 (0.68)	0.581 (0.57)	1.710 (0.52)	1.478 (0.65)
Sad	0.368 (0.44)	0.306 (0.44)	0.234 (0.37)	0.721 (0.51)	0.955 (0.44)
Lonely	0.382 (0.51)	0.301 (0.44)	0.249 (0.46)	0.755 (0.62)	1.053 (0.54)
Cortisol parameters, M (SD)					
Wakeup cortisol	0.417 (0.21)	0.435 (0.23)	0.414 (0.25)	0.402 (0.19)	0.399 (0.18)
Bedtime cortisol	0.107 (0.15)	0.097 (0.13)	0.118 (0.23)	0.133 (0.23)	0.144 (0.22)
CAR	0.160 (0.29)	0.103 (0.31)	0.157 (0.29)	0.164 (0.30)	0.170 (0.30)
Cortisol slope	−0.107 (0.05)	−0.115 (0.05)	−0.107 (0.05)	−0.104 (0.06)	−0.094 (0.06)

PA = positive affect; NA = negative affect; CAR = cortisol awakening response.

Raw cortisol values (in micrograms per deciliter) and high/low PA and NA groups representing the highest quartile are presented for descriptive purposes only. Log-transformed cortisol values and continuous affect measures are used in all analyses.

and 26 females in the sample were using oral contraceptives. Details on cortisol parameters and demographic covariates are presented in Table 1, both for the overall sample and, for illustrative purposes, those at the extreme ends of each affect variable (i.e., those in the highest quartile). On average, the diurnal cortisol slopes across the day are negative (with a higher waking value and a lower bedtime value), such that a positive coefficient is indicative of "flatter" slopes and a negative coefficient indicates steeper slopes (see Fig. 1).

Diurnal Cortisol Analyses

We observed a number of findings based on the health and sociodemographic covariates, as reported in previous research on this sample (38,39,50). In particular, male sex was associated with lower wakeup levels of cortisol ($\gamma = -0.195$, $p = .001$) and a smaller CAR ($\gamma = -0.253$, $p = .003$). Black race was associated with lower wakeup levels of cortisol ($\gamma = -0.176$, $p = .036$), higher bedtime levels of cortisol ($\gamma = 0.433$, $p = .006$), and a flatter slope across the waking day ($\gamma = 0.038$, $p < .001$). Greater total hours of sleep, on the other hand, were associated with higher cortisol at waking ($\gamma = 0.064$, $p = .025$) and a steeper slope across the day ($\gamma = -0.007$, $p = .032$).

Next, we examined whether high and low arousal PA and/or NA was associated with diurnal cortisol parameters in this three-level growth curve analysis (Table 2). There was a marginally significant relationship between high arousal PA and lower levels of cortisol at bedtime ($\gamma = -0.067$, $p = .10$), with a significant, negative quadratic term ($\gamma = -0.090$, $p = .007$), illustrating that the effect of high arousal PA on lower bedtime cortisol increased at higher intensity levels (i.e., for stronger positive emotions). For the diurnal cortisol slope, the quadratic term was also significant and negative ($\gamma = -0.004$ $p = .037$); the linear term was not significant ($\gamma = -0.002$, $p = .270$), suggesting that only values at the high end of the high arousal PA scale were associated with a steeper cortisol slope. High arousal PA was not significantly associated with waking levels of cortisol or the CAR. There were also no statistically significant relationships between low arousal PA with any of the cortisol parameters.

High arousal NA was marginally associated with lower cortisol at waking ($\gamma = -0.055$, $p = .091$), with a significant, positive quadratic term ($\gamma = 0.042$, $p = .030$), suggesting that at very high levels of high arousal NA, the relationship between high arousal NA and waking cortisol reverses. Because these scales are standardized, it can be interpreted that starting at high-intensity levels of the scale (at least 1.3 SDs above the mean), high arousal NA is associated with higher levels of waking cortisol. Low arousal NA (sad, lonely) was associated with lower cortisol upon waking ($\gamma = -0.075$, $p = .038$), a flatter slope across the waking day ($\gamma = 0.011$, $p = .007$), and marginally associated with higher levels of cortisol at bedtime ($\gamma = 0.106$, $p = .079$).

Within-Person Change for 5 Years

Overall, most participants showed a decrease in morning cortisol level (average within-person decrease of 0.163 μg/dl) across 5 years, from Time 1 to Time 2. However, changes in bedtime cortisol level (0.016 μg/dl lower at Time 2), CAR (0.023 μg/dl lower at Time 2), and cortisol slope (0.005 μg/dl flatter at Time 2) were modest. There were also small mean within-person changes in affect—with overall increases in both high and low arousal PA and overall decreases in both high and low arousal NA—from Time

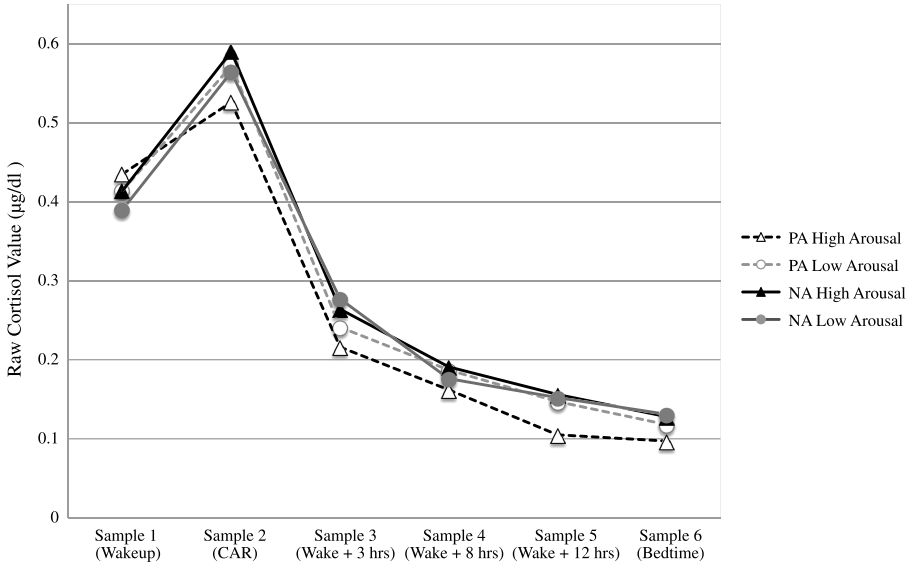

FIGURE 1. Average cortisol rhythms across the waking day by affect and arousal. CAR = cortisol awakening response; PA = positive affect; NA = negative affect.

TABLE 2. Three-Level Growth Model Examining the Effect of Positive Affect and Negative Affect on Log-Transformed Cortisol (in micrograms per deciliter; $n = 315$)

Estimation of Fixed Effects	Coefficient	SE
Waking cortisol		
Intercept	−1.160**	0.027
High arousal PA	−0.030	0.023
High arousal PA^2	−0.017	0.017
Low arousal PA	0.007	0.026
Low arousal PA^2	−0.016	0.020
High arousal NA	−0.055†	0.034
High arousal NA^2	0.042*	0.019
Low arousal NA	−0.075*	0.041
Low arousal NA^2	0.024	0.014
Cortisol slope		
Intercept	−0.147**	0.008
High arousal PA	−0.002	0.003
High arousal PA^2	−0.004*	0.002
Low arousal PA	0.004	0.003
Low arousal PA^2	−0.001	0.002
High arousal NA	0.003	0.004
High arousal NA^2	0.001	0.002
Low arousal NA	0.011**	0.004
Low arousal NA^2	−0.002	0.002
Bedtime cortisol		
Intercept	−2.939**	0.041
High arousal PA	−0.067†	0.041
High arousal PA^2	−0.090**	0.030
Low arousal PA	0.073	0.041
Low arousal PA^2	−0.027	0.031
High arousal NA	−0.017	0.053
High arousal NA^2	0.052	0.031
Low arousal NA	0.106†	0.059
Low arousal NA^2	−0.019	0.024

Variance explained	Pseudo R^2
Wakeup cortisol	0.267
Cortisol slope	0.252
Bedtime cortisol	0.194

PA = positive affect; NA = negative affect; SE = standard error.

Covariates include a time since waking squared variable to capture the quadratic curvilinear components of change in cortisol across the day and a dummy variable indicating the cortisol awakening response (Level 1), daily wake time (Level 2), and race/ethnicity, average hours sleep, oral contraceptives use, nicotine use, recent major depressive disorder, and neuroticism risk (Level 3). The estimates for bedtime cortisol were measured in a separate hierarchical linear model with time centered at bedtime.

Multiply cortisol values (in micrograms per deciliter) by 27.59 to obtain nanomoles per liter.

* $p < .05$, ** $p < .01$, † $p < .1$.

1 to Time 2 (Table S2, Supplemental Digital Content 2, http://links.lww.com/PSYMED/A205).

The fixed-effects models, examining change in affect and cortisol over a 5-year period, are presented in Table 3. The estimated effect is interpreted as the effect of a within-person change in high and low arousal PA or NA on change in cortisol rhythms over time, controlling for both measured and unmeasured time-invariant covariates. High arousal PA was the only affect factor to predict within-person changes in cortisol. Estimates of the coefficients in Models 1 and 2 show a significant association between high arousal PA and the diurnal cortisol slope ($\beta = -0.038$, $p = .009$) and bedtime cortisol ($\beta = -0.661$, $p = .027$) over the 5-year period. That is, an increase in high arousal PA was associated with a steepening of the diurnal cortisol slope and lowering of evening cortisol over time, controlling for all time-invariant covariates.

DISCUSSION

Past research has focused on HPA *dys*regulation (e.g., hypocortisolemia or hypercortisolemia) as markers of poor mental and physical health outcomes. For instance, many researchers have examined changes in normal HPA axis activity as possible mechanisms to explain the sharp rise in clinical depression and anxiety disorders during adolescence (30,31). Because cortisol is commonly known as a "stress hormone," few studies have attempted to link variations in diurnal cortisol to positive psychological experiences. The current study addresses this gap in the literature by examining the relationship between multiple measures of PA and salubrious HPA functioning in a diverse sample of high school students.

The most novel finding from the current study was that high arousal PA was associated with healthy patterns of cortisol secretion across the day. Youth with elevated, high-intensity active and alert moods had a steeper decline in cortisol than did their less energetic peers. Additional analyses uncovered that the steeper decline was primarily driven by lower evening cortisol, which could be a reflection of more efficient negative feedback control of the HPA axis. The inability to suppress cortisol at night, leading to prolonged glucocorticoid exposure, is a hypothesized mechanism linking flat cortisol slopes to a variety of poor health outcomes including obesity (57), depression (58), and all-cause mortality (12). On the other hand, a steep diurnal pattern of cortisol secretion across the day (high on awakening, moderate CAR with rapid recovery, and low in the evening, defining a steep slope across the day from waking to bedtime) could prove to be a useful indicator of good health (3,55,59). High arousal PA may therefore promote or sustain healthy psychobiological trajectories across adolescent development.

Importantly, our analyses control for a number of potentially confounding variables, including wake time and sleep duration. Previous research has reported a reciprocal

TABLE 3. Fixed-Effect Regression Examining Within-Person Change in Average Positive Affect and Negative Affect on Within-Person Change in Log-Transformed Cortisol (in micrograms per deciliter) from Time 1 to Time 2 ($n = 166$)

	Model 1 Diurnal Cortisol Slope		Model 2 Bedtime Cortisol	
	β	SE	β	SE
Affect variables				
PA–high arousal	−0.038**	0.015	−0.661*	0.295
PA–low arousal	0.021	0.015	0.111	0.305
NA–high arousal	−0.009	0.105	−0.387	0.380
NA–low arousal	0.021	0.019	0.508	0.347
Health covariates				
Mean wake time	−0.009*	0.004	−0.209**	0.077
Average hours of sleep	−0.005	0.005	−0.153	0.105
Nicotine use	0.053**	0.019	1.131**	0.358
Oral contraceptive use	0.021*	0.010	0.213	0.173
	R^2 within = 0.160		R^2 within = 0.208	

PA = positive affect; NA = negative affect; SE = standard error.

Multiply cortisol values (in micrograms per deciliter) by 27.59 to obtain nanomoles per liter.

* $p < .05$, ** $p < .01$.

relationship between sleep and basal patterns of cortisol across the day, with more hours of sleep predicting a steeper cortisol slope, and higher waking cortisol predicting more sleep and lower fatigue the following day (2,50). Furthermore, both laboratory and epidemiological data have shown that shorter sleep duration and poor sleep quality are associated with a flatter cortisol slope and increased evening levels (60–62). The current study suggests that active and alert moods predict diurnal cortisol across the day, above and beyond these sleep-specific predictors of cortisol activity.

An additional methodological contribution of the study is the use of fixed-effects estimation. This technique is commonly used in economics and has more recently been applied in developmental and health research to reduce the impact of confounding by time-invariant factors, such as the unmeasured characteristics of the individual. In these within-person analyses, youth who experienced increases in high arousal PA between adolescence and young adulthood also demonstrated steepening cortisol slope and lowering bedtime cortisol across the same period.

Given that both high arousal (e.g., anger and stress) and low arousal (e.g., sad and depressed) negative emotions have been linked to measures of cortisol activity in the past (2,4), it is often assumed that valence, rather than arousal, is the most important emotional predictor of cortisol activity. The current study, however, highlights the importance of arousal, particularly high-intensity feelings of arousal, when studying the physiological mechanisms linking PA and HPA axis activity in adolescents. Strong emotions may be particularly salient for youth, relative to both younger children and adults, due to neurobiological changes that occur at the beginning of adolescence. Research on adolescent brain development suggests differential development of bottom-up limbic systems, implicated in incentive and emotional processing, to prefrontal top-down control systems during this developmental period. This imbalance model proposes that differential developmental timing of these regions (i.e., less top-down regulation) accounts for heightened emotionality during adolescence ((63,64); see also Ref. (65) for a critical review of this hypothesis).

Although high and low arousal positive emotions often co-occur, these different expressions of emotion may have distinct psychobiological profiles. We also found that low arousal NA (but not high arousal NA) during adolescence was associated with lower waking cortisol and a flatter cortisol slope across the day, a finding that is consistent with past research (4). Furthermore, although moderate levels of high arousal NA during adolescence may be associated with lower cortisol upon waking, youth at the extreme levels of high arousal NA show a reversal in this pattern (i.e., higher waking cortisol). The associations between NA and basal cortisol, however, were not replicated in the longitudinal analyses. Future research is needed to uncover the role of emotional arousal and intensity in diurnal cortisol in adult samples, which may help elucidate inconsistencies across past research studies.

Because measuring PA was not an original goal of the larger study from which these data were drawn, we were limited by the type and quantity of arousal-specific emotions we could measure. Future research should seek to examine a wider range of arousal-specific emotions such as excitement, vigor, pep, strength, and exuberance. The

current study is also limited in terms of generalizability (i.e., higher proportion of female and neurotic youth) and causality (i.e., unknown causal direction). The greater proportion of females over males is accounted for by the fact that females usually score higher than do males on neuroticism, the personality trait that was oversampled at baseline (44), and females were more likely to agree to participate in the study if invited. A few previous studies have found differences in diurnal cortisol rhythms in adult participants high in neuroticism as compared with those with low neuroticism (66,67), whereas others have not (68,69). Importantly, neuroticism risk did not significantly predict any diurnal cortisol parameters in the current analyses, nor did it change point estimates when it was included as a covariate. Finally, although many studies conclude that stressful or emotional experiences are responsible for alterations in diurnal cortisol activity, causal directions are not yet firmly established (70). Although emotional experience may influence cortisol levels, it is also possible that cortisol could influence perceived arousal level and perceived emotional experience (3).

Despite these limitations, the current study underlines the importance of examining both valence and arousal to uncover the complex psychobiology of youth emotions, and highlights the role of positive affective experiences in diurnal cortisol activity. High arousal PA may be a particularly salient influence of the HPA axis during adolescence, a developmental period characterized by periodic heightened emotional intensity and a proclivity toward excitement and activity.

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