

The impact of experimental sleep restriction on affective functioning in social and nonsocial contexts among adolescents

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Background: Short sleep duration is highly prevalent in adolescence, and it prospectively predicts problems with emotional adjustment and psychiatric health. To move beyond epidemiological associations and inform models of developmental psychopathology, we experimentally restricted sleep to observe impacts on affective functioning. Based on the importance of social contexts to adolescent emotional experiences, we also examined the impact of restricted sleep on socioaffective functioning in an ecologically valid peer interaction task. **Methods:** In Study 1, adolescents (ages 11.5–15.0, $n = 48$) were randomly assigned to two nights of polysomnography-monitored sleep restriction (4 hr in bed) or extension (10 hr in bed). One week later, they completed the other sleep manipulation. Affective functioning was assessed by self-report and pupil response to standardized affective sounds. Study 2 used a similar protocol and invited adolescents (ages 12–15.0, $n = 16$) to the sleep laboratory along with 2–4 friends to observe affective behavior in a social context primed for peer conflict. Mixed effects models were used to evaluate the effect of sleep condition on affective outcomes. **Results:** Study 1 demonstrated increased negative affect following sleep restriction, relative to extension, on self-report ($p = .02$) and pupil measures ($p = .01$). Study 2 replicated these effects (both $p = .04$) and demonstrated greater negative affective behavior in a peer social context ($p = .01$). Exploratory analyses for positive affect showed reductions as assessed by self-report ($p = .005$), but not pupil ($p = .81$), in Study 1; and no significant effects in Study 2 (self-report, $p = .14$; pupil, $p = .29$; positive affective behavior, $p = .43$). **Conclusions:** Experimental sleep restriction in adolescence impacts negative affective functioning as evidenced by self-report and pupil reactivity, as well as observed behavior in a social context primed for peer conflict. Implications for the impact of short sleep on developmental trajectories of emotional adjustment and psychiatric health, and opportunities for early intervention, are briefly discussed. **Keywords:** Sleep; sleep restriction; adolescence; affect; emotion; emotional reactivity; emotion regulation; pupillography; social behavior.

Introduction

Short sleep duration in adolescence is a well-documented public health issue with notable consistency across continents and cultures (Gradisar, Gardner, & Dohnt, 2011). There are important ongoing debates regarding how much sleep is optimal across the life span (Yetish et al., 2015), and specifically in adolescence (e.g., Eide & Showalter, 2012). However, based on available empirical research spanning various functional outcomes, the National Sleep Foundation's most recent recommendation for youth in the adolescent age range (ages 14–17) is around 8–10 hr for optimal sleep, while <7 hr is not recommended (Hirshkowitz et al., 2015). Yet, in the United States, where the current studies were conducted, one third of 12-year-olds report fewer than 7 hr of sleep, which accelerates to two thirds by age 17 (Keyes, Maslowsky, Hamilton, & Schulenberg, 2015).

The high prevalence of short sleep is relevant to psychiatry and psychology because there are

prospective and bidirectional relations between sleep and emotional adjustment, as well as nearly every psychiatric health problem across the life span (Kouros & El-Sheikh, 2015; Sadeh, Tikotzky, & Kahn, 2014). However, intervening variables in longitudinal designs can obscure causality. Complementary experimental designs that more directly assess causal influences (Hill, 1965) can inform models of developmental psychopathology and strengthen the evidence base for public health initiatives (e.g., school start times) or clinic-based sleep interventions. In this study, we experimentally restrict sleep to observe impacts on affective functioning, which we define as the adaptive modulation of emotions – including behavioral and motivational features of emotions – in the pursuit of short- and long-term goals.

Why examine affective functioning in adolescence?

Developmental changes in affective functioning in adolescence contribute to a twofold increase in morbidity and mortality, including suicide, depression,

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substance abuse, and risky behavior (Ozer, Macdonald, & Irwin, 2002) – problems for which insufficient sleep are also risk factors (Harvey, Murray, Chandler, & Soehner, 2011). Around the onset of puberty and peaking in mid-adolescence, there is increasing sensitivity in a network of neural regions (e.g., striatum and amygdala) involved in emotional and motivational processing, as well as valuing systems involved in learning from rewarding and threatening information in the environment (for review, Crone & Dahl, 2012). Increased reactivity to affective information can influence and challenge regulatory control of emotion and behavior (involving regions in the prefrontal cortex), which leads to some of the seemingly irrational or emotionally driven decisions and behaviors that typify adolescence (e.g., Galván, Hare, Voss, Glover, & Casey, 2007; Teslovich et al., 2014).

There is growing evidence that changes in affective functioning are inextricably tied to social changes in adolescence (Crone & Dahl, 2012). Gaining social competence and independence from family are central developmental milestones of adolescence. Accordingly, there are developmental changes in neural networks underpinning social functioning that drive a selective orientation toward the emotional salience of social information; for example, the threat of peer rejection or the reward of acceptance or status (Nelson, Leibenluft, McClure, & Pine, 2005). This increased neural sensitivity to social information has been shown to affect emotion regulation and related behavioral outcomes (e.g., risky behavior in a simulated driving game) in peer contexts (Chein, Albert, O'Brien, Uckert, & Steinberg, 2011).

An important set of questions, and the focus of this investigation, is whether short sleep alters the threshold at which adolescents can adaptively navigate normative developmental challenges to affective functioning, and particularly in the salient social contexts that create additional challenges to emotion regulation in adolescence. Such daily effects of short sleep on affective functioning could tip vulnerable youth toward negative health trajectories.

Impact of sleep restriction on affective functioning in adolescence

Two studies have examined the impact of sleep restriction on affective functioning in adolescence. The first showed that two nights of sleep restriction (~6.5 hr at home and 2 hr in the laboratory, respectively), versus sleep at home (7–8 hr), was associated with self-reported increases in negative affect and decreases in positive affect, as well as greater anxious responses to an interview about worries and catastrophic thinking (Talbot, McGlinchey, Kaplan, Dahl, & Harvey, 2010). Additionally, open-ended interviews on the morning following sleep restriction, relative to the evening prior to restriction, indicated

increases in negative, and decreases in positive, affective expressions and vocalizations (McGlinchey et al., 2011). The second study used a 3-week home protocol of sleep restriction (6.5 hr for each of five weeknights) relative to extension (10 hr in bed for each of five weeknights) and showed greater self-reported negative affect, and poorer affect regulation by parent report, following restriction (Baum et al., 2014).

We are unaware of any sleep restriction study to examine the impacts on the neural underpinnings of affective functioning in adolescents. One experimental total sleep deprivation study in adults showed increased activity in neural regions involved in affective processing (e.g., amygdala) in response to negative affective stimuli, as well as reduced functional connectivity between the amygdala and regions involved in regulatory control (e.g., prefrontal cortex; Yoo, Gujar, Hu, Jolesz, & Walker, 2007). Another study demonstrated potentiated pupillary reactivity to negative affective stimuli following sleep restriction (Franzen, Buysse, Dahl, Thompson, & Siegle, 2009). The pupil is innervated by cognitive and affective processing brain regions and has been shown to dilate when activity in these regions is high (Koikegami & Yoshida, 1953; Siegle, Steinhauer, Stenger, Konecky, & Carter, 2003). As such, pupil reactivity can be used as a general, but nonspecific, physiological index of task-related brain activity (for more on rationale, see Franzen et al., 2009). If similar findings following acute sleep restriction are demonstrated in adolescence, it would lend support to the testable theory that chronically short sleep may negatively impact developing circuitry that underpins adaptive affective functioning.

Several adult studies have examined the impact of sleep deprivation on neural underpinnings of positive affective functioning. At least one fMRI study indicated increased activation in regions involved in affect (amygdala) and reward processing (striatum) in response to positive emotional pictures (Gujar, Yoo, Hu, & Walker, 2011), and several studies have shown enhanced striatal activation in anticipation or receipt of reward (Mullin et al., 2013; Venkatraman, Huettel, Chuah, Payne, & Chee, 2011). These findings are interesting given evidence for reduced subjective experience of positive affect, suggesting different levels of assessment may reveal unique insights (Franzen, Siegle, & Buysse, 2008). Moreover, one study using pupil methodology failed to demonstrate the effects of sleep deprivation on reactivity to positive affective pictures in adults (Franzen et al., 2009). More research is needed to closely examine the effects of sleep restriction on positive affective functioning across assessment levels (neural response, self-report, behavior, etc.), and particularly in adolescence when there are profound changes in positive affect and reward processing that contribute to health risks (for review, Galván, 2010).

Finally, a notable omission in the literature is the impact of sleep restriction on affective functioning in social contexts of adolescence. Several studies with adults have documented such effects (Anderson & Dickinson, 2010; Kahn-Greene, Lipizzi, Conrad, Kamimori, & Killgore, 2006; Murray, Schein, Erikson, Hill, & Cohen, 1959). One study demonstrated that emotion played a greater role in social decision-making on a bargaining and trust game following sleep deprivation (Anderson & Dickinson, 2010). Examining such effects in adolescence is an important next step to understanding the impact of short sleep on affective functioning and development.

Current studies

In Studies 1 and 2, we used a multimethod design to examine the impact of sleep restriction not only on self-reported affect but also on pupillary responses to affective stimuli. We hypothesized that sleep restriction, relative to extension, would amplify negative affective responding, as evidenced by subjective report of negative affect and pupillary response to negatively valenced stimuli. Additionally, in Study 2, we used an innovative protocol in which youth came to the sleep laboratory with their friends to examine affective behavior in a natural (ecologically valid) and emotionally salient social context. We hypothesized that sleep restriction, relative to extension, would be associated with increases in observed negative affective behavior when peers were asked to resolve a conflict. In both studies, we also examined positive affective functioning: subjective report, pupillary response to positive stimuli, and observed positive affective behavior in a social context. However, analyses were exploratory given less consistency across prior studies and levels of assessment following sleep restriction.

Study 1

Methods

Participants. Adolescents who are 11.5–15.0 years [$n = 48$; mean age = 13.33, $SD = .99$; 21 girls and 24 boys; five African-American, three biracial (African-American and Caucasian), one Asian, 36 Caucasian, and three undisclosed race] participated in this study. An additional nine participants began the experimental protocol and dropped out due to illness ($n = 1$), inability to tolerate study procedures ($n = 2$), noncompliance with study procedures ($n = 1$), or declination to return for the second visit ($n = 5$). Exclusion criteria included the following: current or past Axis I psychiatric diagnosis, assessed by Schedule for Affective Disorders and Schizophrenia for School-Age Children-Present and Lifetime Version (K-SADS-PL)(Kaufman et al., 1997) or the Diagnostic Interview Schedule for Children-IV (Shaffer, Fisher, Lucas, Dulcan, & Schwab-Stone, 2000); sleep

disorder assessed by the locally developed Structured Clinical Interview for DSM-IV Sleep Disorders; other major medical problem, including epileptiform disorders or history of a seizure; previous adverse neuropsychiatric reaction to sleep deprivation; first-degree relative diagnosed or treated for bipolar disorder; currently taking medications known to affect sleep/wake function; pregnant; colorblind; and inability to read or write English.

Procedure. Study procedures were approved by the University of Pittsburgh Institutional Review Board (IRB). Parental informed consent and adolescent assent were obtained. Participants were remunerated for their time. Eligible adolescents participated in two 48-hr laboratory visits during summer months. They were randomly assigned to two nights of sleep restriction or extension in the laboratory and then returned 1 week later for the other condition. Participants wore an Actiwatch 2 (Philips Respironics, Murrysville, PA) beginning about a week prior to the first laboratory through the second visit. Sleep restriction included 4 hr in bed (1:00–5:00 am) for two consecutive nights. Sleep extension included 10 hr in bed (10:00 pm to 8:00 am) for two consecutive nights. Participants were under constant behavioral monitoring to ensure they remained awake at all other times. Participants completed structured and unstructured activities and completed questionnaires (visual analog scales) of subjective affect three times throughout Day 1 (following Night 1). On Day 2, participants completed the same questionnaires and a battery of tasks, including the auditory valence identification task in the morning.

Our rationale to use two consecutive nights in the laboratory for each sleep manipulation included the following: to maximize experimental control; to ensure that sleep debt was accumulated and not counteracted by the novelty of the experience or ‘rally effects’; and to reduce first-night effects of sleeping in a laboratory environment. The 1-week period in between sleep manipulations was used as a wash-out period to bring sleep back to baseline for participants randomized to sleep restriction first. We included 10-hr time in bed in the sleep extension condition to ensure participants would be as well-rested as possible and to discharge any potential sleep debt participants built up prior to sleeping in the laboratory. On average, participants slept less than this at home according to actigraphy-derived total sleep time ($M = 7.26$, $SD = .8$ hr, range = 5.1–8.7 hr). We selected the timing of sleep restriction to have the same midpoint as the sleep extension nights and to total up to 8 hr of sleep over the 48-hr laboratory visit. We followed the rationale of the study by Talbot et al. (2010), which used the same dose of sleep loss (though in that study, it was spread across a night at home and a night in the laboratory) – to more closely approximate the types of sleep loss experienced by adolescents and to reduce homeostatic sleep pressure by

providing some nighttime sleep to help ensure participants would remain awake during daytime testing activities.

Measures

Polysomnography. Sleep was scored by polysomnographic technicians in 30-s epochs using standard criteria (Iber, Ancoli-Israel, Chesson, & Quan, 2007). Sleep onset was defined by 10 consecutive minutes of contiguous sleep. Sleep apnea (i.e., 10 or more apnea/hypopnea events per hour) was ruled out by an at-home ApneaLink (ResMed, San Diego, CA) study ($n = 38$) or clinical screening.

Visual analog scales. Each item consisted of a 100-mm line from very little (0) to very much (100; Buysse et al., 2007). Negative affect was the mean rating of sad, tense, anxious, stressed, and irritable. Positive affect was the mean rating of happy, calm, and relaxed.

Auditory valence identification task. Forty-two sound clips (14 per category¹: positive, negative, neutral valence) from International Affective Digitized Sounds (Bradley & Lang, 1999) were presented for 6 s, in random order, preceded by a 1-s orientation cue and followed by an 8-s intertrial interval. Stimuli were presented using E-prime 2 (Psychology Software Tools, Pittsburgh, PA). A fixation was presented in the center of the screen consisting of 13 X's at 16-point font size in gray on a silver background. Two vertical hash marks above and below the center fixation shifted from two to one space apart at the beginning of each trial. After the stimulus terminated, the hash marks returned to two spaces apart, cueing participants to rate the valence of the stimulus by pressing one of three buttons (the order of which was counterbalanced), indicating whether the valence of the stimulus was positive, negative, or neutral. Valence recognition accuracy was coded as 'correct' (perceived valence was consistent with intended valence) or 'incorrect' (perceived valence was inconsistent with intended valence). Overall valence recognition accuracy was >86%.

To collect pupil dilation data, a video camera and infrared light source were pointed at the participants' left eye and the video image was digitized to calculate pupil diameter using an Applied Science Laboratories EYE-TRAC 6 (Bedford, MA). Pupil diameter was continuously recorded at 60 Hz (every 16.7 ms) and passed digitally to a storage computer. Signals were transmitted from the computer presenting the stimulus to the storage computer to mark trial length, fixation, and stimulus onset. Data were cleaned using standard methodology derived from Siegle, Ichikawa, and Steinhauer (2008), including eye blink identification and artifact rejection. Blinks and rejected segments were corrected

using linear interpolation, and data were smoothed by applying a five-point moving average. Data from one participant were lost due to equipment failure, and another eight were lost due to >50% of trials with excessive blinking or eyelid closures for more than half of a trial, leaving a final sample of $n = 39$. The time course of responses during the stimulus presentation and subsequent intertrial interval were established by subtracting baseline pupil diameter (6 ms prior to trial onset) from the pupillary waveform for each affective category. Data were down-sampled into 1-second averages for analysis.

Analytic plan

Mixed effects models evaluated the effect of sleep condition on self-reported affect subjected to a natural log transformation. Individuals were included as a random effect. Fixed effects included sleep condition (restriction, extension) and study day (Day 1, Day 2) as repeated measures. For the auditory valence identification task, we calculated the average pupil response during the peak window of activity (seconds 4–6) based on visual inspection of the waveforms across the entire sample. Mixed effects models evaluated the effect of sleep condition on pupil response to affective sounds; models separately contrasted positive and negative, relative to neutral sounds, to control for physiological responsiveness.

Results

Manipulation checks. Polysomnographically assessed total sleep time supported a successful sleep manipulation (Table 1). To ensure that sleep restriction did not differentially impact the ability to recognize valence categories in the auditory valence task, we conducted a mixed model in which sleep

Table 1 Means and standard deviations of primary Study 1 outcome variables

Assessments	Sleep extension		Sleep restriction	
	Mean	SD	Mean	SD
Total sleep time	08:51	00:51	03:57	00:30
Night #1 (hours:minutes)				
Total sleep time	09:11	00:28	03:54	00:07
Night #2 (hours:minutes)				
Subjective negative affect Day 1	19.28	12.58	22.29	16.10
Subjective negative affect Day 2	18.11	13.11	21.68	12.34
Subjective positive affect Day 1	67.42	17.01	61.53	18.37
Subjective positive affect Day 2	70.01	17.84	59.27	16.57
Peak pupil negative (mm)	.24	.21	.36	.25
Peak pupil positive (mm)	.13	.18	.14	.18
Peak pupil neutral (mm)	.16	.18	.18	.19

Subjective negative affect and positive affect are reported as the original, nontransformed data.

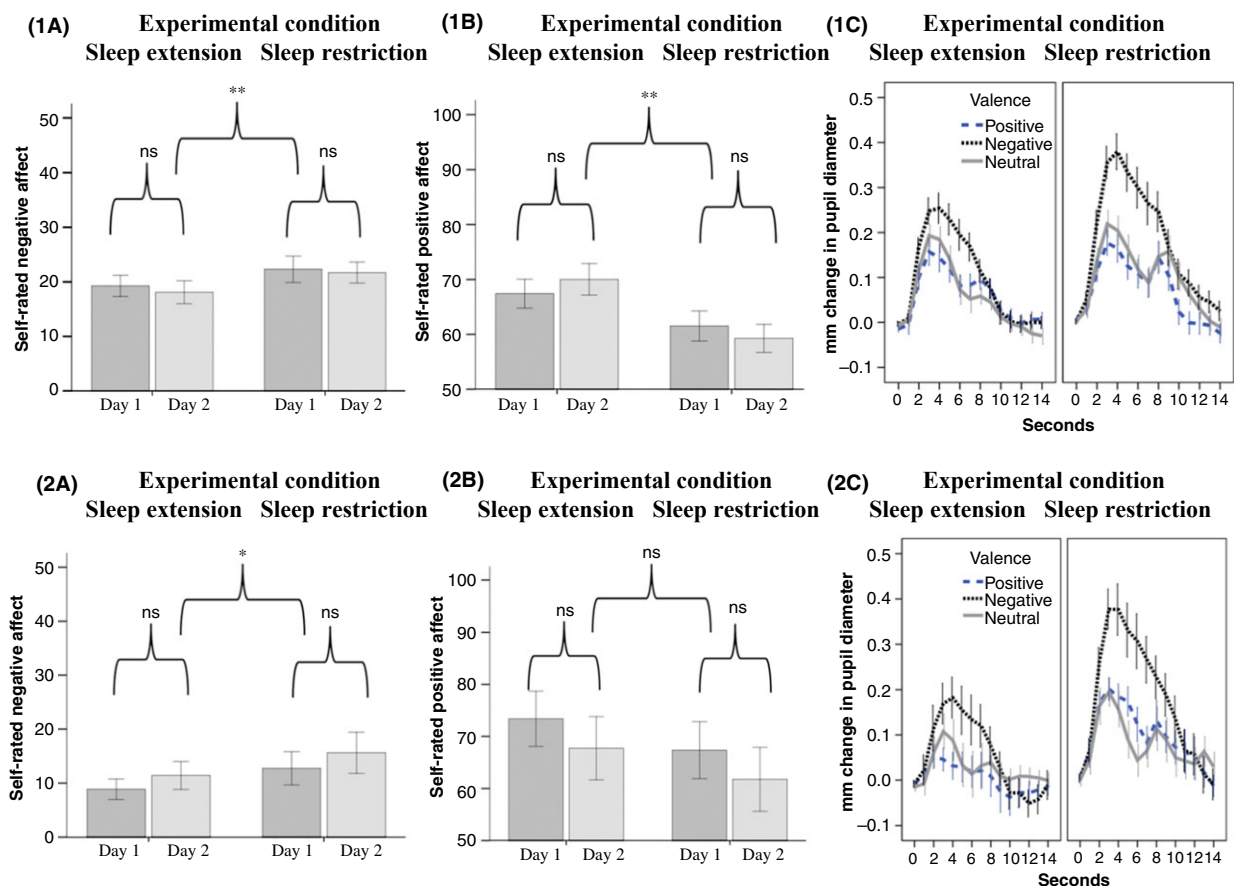
manipulation and valence category predicted valence recognition accuracy. There was no main effect of sleep condition ($F(1, 14) = 1.51, p = .22$) or sleep condition by valence interaction ($F(2, 14) = .46, p = .63$) on valence recognition accuracy.

Self-report. There was a main effect for sleep condition on negative affect, $F(1, 46.25) = 6.11, p < .02$; negative affect was higher following restriction (Table 1 and Figure 1). Neither the main effect of day nor the sleep condition by day interaction was significant ($F(1, 40.97) = 0.188, p = .67$ and $F(1, 44.43) = .212, p = .152$, respectively). There was a main effect for sleep condition on positive affect, $F(1, 47.03) = 10.29, p < .005$; positive affect was lower following sleep restriction. Neither the main effect for day nor the sleep condition by day interaction was significant ($F(1, 46.76) = .00, p = .983$ and $F(1, 46.79) = 2.84, p = .099$, respectively).

Pupillary response. There was a significant effect of sleep condition on the contrast of pupillary

response to negative relative to neutral stimuli, $t(38) = 2.58, p = .01$; pupil response was higher following sleep restriction (Table 1 and Figure 1). There was no effect of sleep condition on the contrast of positive relative to neutral stimuli, $t(38) = -.24, p = .81$.

Post hoc analyses. To ensure that the impact of sleep manipulation on pupil response to affective sounds was not driven by differences in arousal, we conducted mixed effects models while covarying for normative arousal ratings on the average pupil response from seconds 4–6 on a per trial basis. Similar to primary analyses, the effect of sleep condition on negative relative to neutral stimuli remained, $t(2,888) = 2.29, p = .02$, and there was no effect of sleep condition on the contrast of positive relative to neutral stimuli, $t(2,877) = -.05, p = .96$. Online supplementary Figure S1 shows the average pupil dilation waveforms on a median split of high and low arousing positive and negative stimuli, and it indicates no differences in the



Abbreviations: * $p < .05$; ** $p < .01$; ns = non-significant; error bars ± 1 standard error. **Notes.** Self-rated affect scales were 0–100.

Half of each scale is represented in graphs, due to restricted range—data were log transformed for analyses.

Figure 1 Self-rated affect and pupil response to affective sounds across sleep conditions. 1A/2A) Self-reported ratings of negative affect show significantly elevated negative affect following sleep restriction, relative to sleep extension in Study 1 (top panel, 1A) and Study 2 (bottom panel, 2A). 1B/2B) Self-reported ratings of positive affect show significantly reduced positive affect following sleep restriction, relative sleep extension in Study 1 (top panel, 1B), but not Study 2 (bottom panel, 2B). 1C/2C) Time course of pupil dilation in response to positive, negative, and neutral sounds clips shows significantly elevated pupil response to negative sounds during the peak window of activity (seconds 4–6) following sleep restriction, relative to sleep extension in both Study 1 (top panel, 1C) and Study 2 (bottom panel, 2C)

pattern of findings across different arousal categories.

We also explored potential age and gender effects on pupillary response and self-reported negative affect by including gender and age as simple effect and interaction terms in primary analysis models. The only significant finding was a main effect of gender ($F = 6.23$, $p = .017$) on pupillary response, with larger pupillary responses to negative relative to neutral sounds in females compared with males.

Study 2

Methods

Participants. Adolescents who are 12–15.0 years ($n = 16$, mean age = 14.46, $SD = 1.19$; six African-American and 10 Caucasian youth; six girls and 10 boys) completed the protocol in groups of same-sex friends (groups included 2, 3, or 4 friends) in a within-subject cross-over design. Exclusion criteria were identical to Study 1, with the addition of obesity (defined by BMI-for-age >95%) and loud snoring, as apnea diagnostics were not performed. An additional four participants were not included because family scheduling issues prevented completion of the second sleep condition.

Procedure. Procedures were similar to Study 1 with two noteworthy exceptions. Sleep restriction included 6 hr of time in bed (2:00–8:00 am) on Night 1 and 2 hr of time in bed (2:00–4:00 am) on Night 2. Although nightly schedules differed from Study 1, the total sleep opportunity (eight of 48 hr) was the

same. The peer interaction task was completed in the afternoon of Day 2.

Measures

Polysomnography, visual analog scales, and auditory valence identification task. These were identical to Study 1.

Peer conflict task. Participants listed five recent disagreements they had with their friend and rated the severity of the conflict (5-point Likert scale). Youth were studied with one friend, and at each of the experimental visits, they were asked to discuss one of the two conflicts identified and rated most highly by both members of the pair during a 5-min videotaped discussion (Hetherington & Clingempeel, 1992). Separate conflicts were discussed at each visit; for occasions in which conflict severity was not rated as equivalently high, the more highly rated conflict was discussed during the sleep extension visit to bias against hypotheses. A cue card reminded them to address the following: how disagreement started; who else was involved; how the issue ended (if it had); and how to deal with the issue in the future. Following this conflict discussion, a 5-min cool-down task transitioned youth to discuss something pleasant. Safeguarding procedures (debriefing or intervention by study staff) were also in place in the event that youth had difficulty de-escalating conflict or negative emotions, but there was never a need for this to be implemented in the current sample.

In total, four participants were missing a time point due to scheduling constraints. Videotapes were coded using the Interactional Dimensions Coding System-Revised (IDCS-R), which is a macro-analytic system (i.e., ‘big picture’ view of behavior) designed to assess couples’ communication (Julien, Markman, & Lindahl, 1989) and modified for adolescents (Furman & Shomaker, 2008). Coders rated behavior, facial expressions, and verbal content on a five-point Likert scale with half-point intervals (1, extremely uncharacteristic to 5, extremely characteristic). By standard procedure, each of the ratings were made for two, 2.5-min segments, and averaged to create summary scores. To minimize the number of statistical tests, two composite scores were calculated by averaging summary scores that best represented negative affective behaviors (negative affect, conflict, withdrawal, and dominance) and positive affective behaviors (positive affect, support validation, and problem solving). Tapes were randomly assigned to coders who were blind to participants’ sleep condition. The coding team was trained by the first author as part of a prior study for which strong reliability was established (McMakin et al., 2011), and 50% of segments from the current study were double coded. Intraclass correlation coefficients for positive and negative composite variables were .72 and .75, respectively.

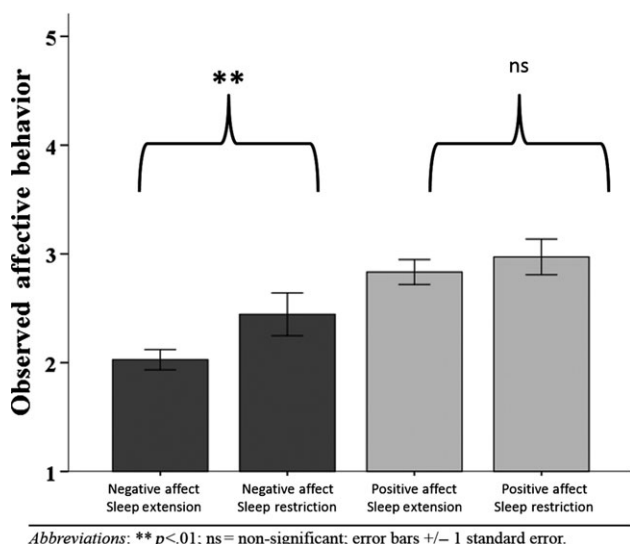


Figure 2 Observed affective behavior in a social context across sleep conditions in Study 2. Observed negative affective behavior was significantly elevated following sleep restriction, relative to sleep extension. Positive affective behavior did not significantly differ across sleep conditions

Analytic plan

The analytic plan was identical to Study 1, with two exceptions. First, additional mixed effects models were included to evaluate the effect of sleep condition on observed affective behaviors (negative and positive) during the peer interaction task. Individuals were included as a random effect and fixed effects included sleep condition. Second, for the auditory valence identification task, three outliers (two neutral and one negative data points) $>1.5 \times \text{IRQ}$ from the 25th or 75th percentile were Winsorized.

Results

Manipulation check. Sleep time, as assessed by polysomnography, supported a successful sleep manipulation (Table 2).

Self-report. There was a main effect for sleep condition on negative affect, $F(1, 12.4) = 5.51, p = .04$; negative affect was higher following sleep restriction (Table 2, Figure 1). Neither the main effect of day nor the sleep condition by day interaction was significant ($F(1, 14.91) = 3.16, p = .10$ and $F(1, 14.81) = .62, p = .45$, respectively). There was no main effect for sleep condition on positive affect, $F(1, 14.5) = 2.41, p = .14$. There was a trend for a main effect for day; self-reported positive affect was lower on Day 2, $F(1, 15.10) = 4.28, p = .06$; however, there was no sleep condition by day interaction, $F(1, 15.08) = .02, p = .88$.

Pupillary response. There was a significant effect of sleep condition on the contrast of pupillary response to negative relative to neutral stimuli, $t(14) = 2.22, p = .04$; pupil response was higher following restriction. There was no effect of sleep

condition on the contrast of positive relative to neutral stimuli, $t(14) = -1.26, p = .29$.

Observed behavior. There was a main effect for sleep condition on negative affective behaviors, $F(1, 14.6) = 7.77, p = .01$; negative affective behavior was higher following restriction (Figure 2). There was no main effect of sleep condition on positive affective behaviors, $F(1, 14.1) = .67, p = .43$. Please see Supplementary section for analyses of specific affective behaviors that comprised negative and positive composites.

Post hoc analyses. To examine if self-report and pupil responses were associated with real-world behavior, we explored bivariate correlations between outcome variables that were significant in primary analyses. Pupillary response to negative sounds was positively associated with negative affective behaviors in social contexts following sleep restriction ($r = .64, p = .01$; Figure 3), but there was no evidence of an association following sleep extension ($r = -.06, p = .85$). A one-tailed Fischer's r - z transformation supported a significant difference between these correlations ($z = -1.78; p = .04$). There was no support for a significant association (p 's $> .10$) between self-reported negative affect and observed negative affective behaviors in either sleep condition.

Discussion

These studies demonstrated an impact of sleep restriction on affective functioning in adolescents. Specifically, negative affect was amplified following sleep restriction, relative to extension, both in terms of subjective affective experience and pupillary response

Table 2 Means and standard deviations of primary Study 2 outcome variables

Assessments	Sleep extension		Sleep restriction	
	Mean	SD	Mean	SD
Total sleep time	09:01	00:39	05:44	00:10
Night #1 (hours:minutes)				
Total sleep time	09:13	00:40	01:57	00:05
Night #2 (hours:minutes)				
Subjective negative affect Day 1	18.60	12.28	20.86	14.17
Subjective negative affect Day 2	18.20	13.27	21.20	12.09
Subjective positive affect Day 1	69.38	15.65	63.80	16.01
Subjective positive affect Day 2	71.29	16.91	61.74	15.27
Negative affective behavior	1.52	.24	1.86	.47
Positive affective behavior	2.83	.40	2.94	.51
Peak pupil negative (mm)	.16	.15	.34	.17
Peak pupil positive (mm)	.04	.10	.18	.10
Peak pupil neutral (mm)	.08	.13	.15	.13

Subjective negative affect and positive affect are reported as the original, nontransformed data.

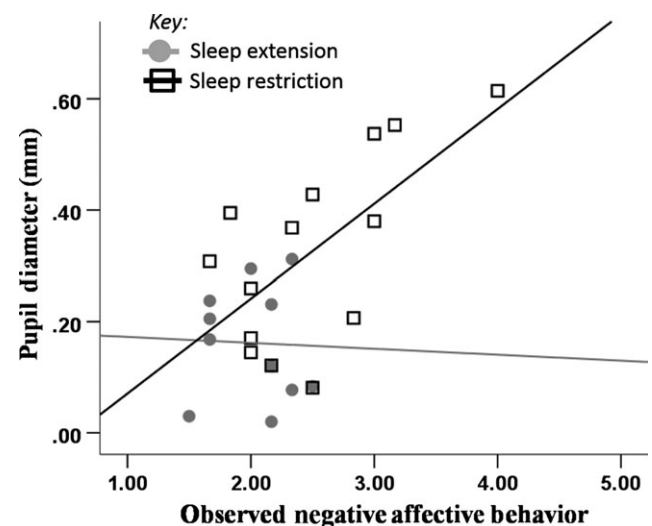


Figure 3 Study 2 pupil reactivity in response to negative sounds and negative affective behaviors in social contexts primed for conflict. Pupil dilation (average response during peak window of activity, seconds 4–6) in response to negative sounds was significantly correlated with negative affective behavior while trying to resolve a conflict with a friend following sleep restriction

to negative affective sounds. Results were replicated across two independent samples. In addition, Study 2 demonstrated effects on emotion regulation in real-world social contexts – specifically, sleep restricted youth showed greater negative affective behavior when trying to resolve a conflict with a friend.

The impact of sleep restriction on self-reported negative affect replicates prior work (Baum et al., 2014; Talbot et al., 2010). To our knowledge, Study 2 was the first to extend findings to affective behavior in social contexts in adolescents. This is an important extension given that essential developmental milestones of adolescence include gaining social competence and peer group belonging. Affective information in social contexts (e.g., peer rejection or acceptance) is, therefore, uniquely salient, and can challenge emotion regulation. These data suggest that short sleep may amplify these challenges, with potential for disrupting key developmental processes.

Pupil responses to affective stimuli provide an inexpensive and indirect physiological index of neural activity related to cognitive and affective processing. Therefore, the effects on pupil responses suggest possible impacts of sleep restriction on neural underpinnings of affective functioning that are reflected in subjective experience and behavior. This notion is further supported by post hoc analyses, showing that greater pupil responses to negative affective sounds in the laboratory were positively associated with negative affective behaviors in real-world social contexts. An important direction for future work is to include assessments of subjective report, pupil reactivity, and affective behavior in a single task to directly compare and contrast responses across these levels. Finally, it is important to note that these results do not reveal whether amplified pupil reactivity was a function of increased sensitivity of affective processing, decreased regulatory control, or some combination. However, taken together, these data support a testable theory that chronically short sleep in adolescence could feasibly impact the development of key circuitry required for adaptive affective functioning.

Exploratory analyses evidenced disruptions in self-reported positive affect in Study 1, but not Study 2. Results from Study 1 are consistent with one prior sleep restriction study with adolescents (McGlinchey et al., 2011). Pupillary response to positive sounds did not evidence disruptions following sleep restriction in Study 1 or 2. However, there were no increases in pupillary reactivity to positive sounds under either sleep condition. This contrasts with (nonsleep) studies in adults that found pupil responses to both positive and negative auditory stimuli (Partala & Surakka, 2003). Our positive stimulus set may have been less powerful because we eliminated sounds with sexual content for this sample of minors. Sleep restriction dose, developmental stage, assessment type (self-report, neural circuitry, behavior), affective stimulus type (emotional, reward, high arousal, low

arousal, etc.), context (social, nonsocial), and/or time course of responses are important factors to continue to parse in future studies.

Post hoc analyses revealed that girls had larger pupil responses to negative affective stimuli overall, but there were no gender or age-related effects on primary affective outcomes associated with the sleep manipulation. This was somewhat surprising given the large changes occurring across both sleep and affective systems, as well as gender-related differences, during this sensitive developmental period. It is possible that examining a larger sample size across a broad age range and/or assessing pubertal stage would reveal developmental differences in outcomes.

A major strength of this set of studies was conducting sleep manipulations in a standardized laboratory environment to observe causal impacts on affective functioning. The rigorous protocol was also a strength. For example, participants spent two nights in the laboratory in both sleep conditions in a randomized cross-over design, which allowed maximal experimental control and minimized novelty or first night effects associated with being in the laboratory. Participants were thoroughly screened for psychiatric and sleep disorders with a clinician administered interview. Multiple methodologies were included to assess affective functioning – this was the first set of studies to examine pupillary responses to affective stimuli in sleep-restricted adolescents, and Study 2 was the first to examine the impact of sleep restriction on affective behavior in real-world social contexts in adolescence. Finally, Studies 1 and 2 provided a replication of key self-report and pupil results.

Limitations include a homogeneous sample of healthy, mostly Caucasian, adolescents, which limits generalizability of findings. The attrition rate of Study 1 (16%) was also relatively high, likely a result of this being a difficult and time-intensive protocol for participants. Also, the sample size for Study 2 was small. Relatedly, we did not conduct post hoc analyses (e.g., arousal, gender, and age as covariates) in Study 2 due to smaller sample size. Instead, we relied on post hoc checks from Study 1 to rule out a significant impact of arousal, gender, or age on interpretation of findings. It will be critical to examine these processes in larger and more diverse samples and among youth who are vulnerable to problems with psychiatric health. Additionally, although adolescents are on average chronically sleep restricted, self-reported sleep duration is typically longer than 4 hr (except in cases of slumber parties, etc.). The threshold of sleep loss necessary to produce the impacts observed in this study is unknown. Finally, this study was the first to examine the impact of sleep loss on social contexts, and we focused specifically on an interaction primed for conflict. This may have contributed to a floor effect for observing differences in positive affective

behavior. However, in our prior work using this same task and coding system among youth at high versus low risk for depression, we did observe differences in positive affective behavior during conflict (e.g., supportive statements, open body position; McMakin et al., 2011), which provides validation for the current approach. Nevertheless, it is important to expand the types of contexts beyond those primed for conflict to fully ascertain how sleep impacts both negative and positive affective functioning across social contexts that reflect daily life for teens.

Moving forward, fMRI methodology can examine more specifically how sleep impacts neural circuitry involved in affective functioning, and in what contexts. Longitudinal designs with high-risk populations that integrate neuroscience and developmental psychopathology can reveal how effects on neural functioning relate to the developing ability to manage emotion and behavior, and particularly in social contexts, and how this contributes to increasing problems with psychiatric health. By closely tracking development (e.g., age and hormonal changes), it may be possible to identify sensitive periods when short sleep and affective functioning most powerfully interact to influence health trajectories. Grounded in this evidence base, healthy sleep promotion could be applied as a public health priority (i.e., school start times, sleep health education) or as an early intervention target for high-risk youth during sensitive windows of development, to offset the twofold rise in rates of morbidity and mortality in adolescence.

Supporting information

Additional Supporting Information may be found in the online version of this article:

Appendix S1. Analyses of specific affective behavioral codes on the peer interaction task from Study 2.

Figure S1. Study 1 pupil dilation waveforms based on a median split of high and low arousing positive and negative stimuli.

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Key points

- Short sleep duration is highly prevalent in adolescence and is associated with emotional adjustment and psychiatric health.
- Experimental sleep restriction can clarify causal impacts of short sleep on affective functioning to inform models of developmental psychopathology.
- Two independent studies demonstrated effects of sleep restriction on affective functioning at the level of self-report and pupil response to standardized affective sounds.
- We also examined the impact of sleep restriction on behavior while resolving a conflict with a friend and observed increased negative affective behavior.
- Sleep duration may be an important target for public health initiatives (later school start times) and early interventions for youth at high risk for problems with emotional adjustment and psychiatric health in adolescence.

Note

1. The IADS stimulus numbers for negative stimuli included 244, 255, 261, 277, 278, 279, 286, 290, 292, 420, 424, 600, 625, 711; normative valence and arousal ratings via Self-Assessment Mankin (SAM) were 2.14 and 7.28, respectively (Bradley & Lang, 1999). Valence SAM ratings ranged from 1 (most negative) to 9 (most positive) and arousal SAM

ratings ranged from 1 (least arousing) to 9 (most arousing). The IADS neutral stimuli included 171, 246, 320, 322, 358, 361, 374, 376, 425, 627, 701, 705, 708, 722; normative valence and arousal ratings were 4.96 and 4.58, respectively. The IADS positive stimuli included 109, 110, 112, 220, 226, 230, 311, 352, 353, 365, 367, 810, 811, 820; normative valence and arousal ratings were 7.24 and 5.80, respectively. All three valences differed

significantly from the others for both normative valence and arousal ratings (all p 's=0.001 or less).

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