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ORIGINAL ARTICLE

Partial Sleep Deprivation Attenuates the Positive Affective System: Effects Across Multiple Measurement Modalities

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Objective: Ample behavioral and neurobiological evidence links sleep and affective functioning. Recent self-report evidence suggests that the affective problems associated with sleep loss may be stronger for positive versus negative affective state and that those effects may be mediated by changes in electroencephalographically measured slow wave sleep (SWS). In the present study, we extend those preliminary findings using multiple measures of affective functioning.
Design: In a within-subject randomized crossover experiment, we tested the effects of one night of sleep continuity disruption via forced awakenings (FA) compared to one night of uninterrupted sleep (US) on three measures of positive and negative affective functioning: self-reported affective state, affective pain modulation, and affect-biased attention.
Setting: The study was set in an inpatient clinical research suite.
Participants: Healthy, good sleeping adults (*N* = 45) were included.
Measurement and Results: Results indicated that a single night of sleep continuity disruption attenuated positive affective state via FA-induced reductions in SWS. Additionally, sleep continuity disruption attenuated the inhibition of pain by positive affect as well as attention bias to positive affective stimuli. Negative affective state, negative affective pain facilitation, nor negative attention bias were altered by sleep continuity disruption.
Conclusions: The present findings, observed across multiple measures of affective function, suggest that sleep continuity disruption has a stronger influence on the positive affective system relative to the negative affective affective system.
Keywords: sleep, affect, emotion, pain, depression, attention.

Statement of Significance
Sleep is a critical regulator of emotional health as evidenced by strong comorbidities between sleep and mood disorders. Our findings, observed through a rigorous within-subject experiment, indicate that disruption of sleep continuity depletes positive affective resources without augmenting negative affective processes. Results suggest that positive affect and its influence on pain and cognition should be critically assessed in patients presenting with sleep complaints. The present experimental findings advance the clinical hypothesis that chronically disrupted sleep may introduce vulnerabilities for the development of mood disorders by eroding the positive emotional resources needed to cope with aversive conditions while decreasing the likelihood that one will recognize and respond to pleasurable stimuli.

INTRODUCTION

Sleep is increasingly recognized as a key contributor to emotional health.[1,2] Until recently, however, relatively little attention has been paid to evaluating the effects of sleep loss on affect valence. This is problematic, given the fact that sleep disorders, such as insomnia and obstructive sleep apnea, substantially increase the risk of development of depression,[3,4] which manifests either as an impairment of primarily positive affective processes (eg, anhedonia),[5] primarily negative affective processes (eg, anger),[6] or both.[7] Without an understanding of how sleep loss impacts positive versus negative affect, diagnostic efforts may be underinformed and treatment plans poorly specified for individuals presenting with a sleep disorder and accompanying affective complaints. Thus, there is a need to systematically interrogate the effects of sleep loss on the affect system, which is characterized by multiple interacting component parts.[8,9]

Although studies have shown that various negative affective states, including anger, hostility, tension, and anxiety, may be altered by sleep loss,[10–12] several recent clinical and experimental studies have demonstrated that the harmful effects of sleep loss on self-reported positive affect may be stronger than those on negative affect.[13–18] For example, sleep restriction attenuated self-reported positive affect, but did not augment negative affect, in adolescents.[18] Poor sleep quality and more spontaneous nocturnal awakenings predicted lower self-reported positive affect, to a greater extent than negative affect, in depressed individuals.[13,17] In an ambulatory monitoring study of young female college students, nightly sleep quality predicted next-day positive affect, joviality, and self-assurance but was not related to negative affect.[14] However, despite the emergence of this trend across diverse sleep study designs, its clinical relevance is limited by the near-exclusive reliance on self-report of affective state in prior studies. Thus, there is a need to comprehensively evaluate the effects of sleep loss on affect through a multimodal assessment of the positive and negative affect systems. Discovery of a possible preferential impact of sleep loss on positive versus negative valence systems, extending beyond basic questionnaires, could critically enhance our understanding of how to assess and treat psychiatric disorders secondary to sleep problems. For example, pharmacological and psychosocial treatments for anhedonia differ substantially from those intended to treat anger, and understanding how sleep differentially influences those processes could help clinicians better anticipate symptom trajectories in the course of treatment. In the present study, we aimed to substantively extend the extant literature by assessing positive and negative affective consequences of sleep continuity disruption across

multiple "nodes" of the affect system. A principal function of affect is to modulate physiological and cognitive states.[8,9] Therefore, we focused the present investigation on two distinct affective modulatory processes: affective pain modulation[19,20] and affect-biased attention.[21] There are myriad tests of affective function, but we selected these processes because they are particularly relevant to aging adults who comprise the fastest growing sector of the global population[22] and are at increased risk of insomnia,[23] attentional control deficits,[24] and chronic pain.[25] Affect reliably modulates both experimental and clinical pain,[20,26] but no study has investigated the acute effect of sleep deprivation (either partial or full) on positive affective pain inhibition or negative affective pain facilitation. Attention bias (AB) to affective stimuli is a cognitive antecedent to the experience of emotionally evocative events and the consequent strategies employed to appraise them.[27] However, the impact of sleep deprivation on positive and negative AB, too, have not been investigated.

Understanding if and to what extent the effects of acute sleep deprivation on positive versus negative affect generalize to affective modulation of pain and/or attention would greatly expand our broader understanding of the interaction of sleep and the affect system, and its consequences for critical psychophysiological processes that undergird an array of chronic disorders, such as chronic pain,[28] insomnia,[29] and depression.[30,31] It would be important, for instance, to know whether sleep disruption-related changes in positive affect interfere with physical activity levels, social engagement, or medication adherence in patients with those disorders. By increasing our understanding of the sleep-affect relationship from a basic experimental standpoint, we will be better equipped to more precisely identify who is at risk, and when risk is greatest, for poor functional outcomes stemming from daily sleep-related affective deficits.

A prior between-subjects study revealed that disrupting sleep continuity through forced awakenings (FA) for three consecutive nights attenuated self-reported positive affect to a greater extent than a sleep restriction control condition and that the group differences were mediated by the larger reductions in slow wave sleep (SWS)—the periods of "deep sleep" associated with cortical oscillations in the delta frequency band (.05 HZ– 2 HZ)—observed through the sleep continuity disruption condition.[10] FA is a novel partial sleep deprivation paradigm that models—albeit to a more severe degree—the type of sleep disruption experienced by people with sleep maintenance insomnia, including patients with chronic pain. Thus, the FA paradigm provides a model of sleep loss that may have greater ecological validity than total sleep deprivation. Here, we extend prior literature by employing a within-subject crossover design that investigates the affective consequences of one night of FA relative to one night of uninterrupted sleep (US) in healthy, good sleeping adults using both subjective and objective measures of affective functioning. We reasoned that if the effect of FA on positive affect is greater than its effect on negative affect, we should also observe larger changes on positive affective pain inhibition and positive AB relative to negative affective pain facilitation and negative AB, respectively. Furthermore, if changes in SWS drive the self-reported perception of positive affect, we expected that SWS would similarly influence positive affective pain inhibition and positive AB.

Our specific hypotheses were (a) FA would attenuate positive affect, positive affective pain inhibition, and positive AB; (b) those effects would be mediated by reductions in SWS; and (c) the effects of FA on corresponding negative affective measures would be smaller in magnitude relative to positive affective measures.

METHODS

Participants

The sample comprised 45 healthy, good-sleeping adults (see Table 1 for demographics). Participants were all enrolled in an ongoing parent study, R01 DA032922 (MTS; MRI), investigating the effects of two nights of sleep continuity disruption on primary and secondary hyperalgesia and the analgesic efficacy of morphine accompanying inflammatory changes. All the daytime tests involved in the parent study were conducted after two nights of normal or disrupted sleep and were therefore completely separate from the daytime testing associated with the present study, which was conducted after one night. Eligibility procedures are presented in Supplemental Material, and inclusion/exclusion criteria are provided in Table S1.

Sample size varied between measures. All subjects completed the self-reported positive and negative affect measure and the emotional dot probe task. Due to computer errors, one subject was missing data on the emotional dot probe task (N = 44). Twelve subjects elected to participate in an optional substudy

Table 1—Participant Demographics.

Sample size	**45**
Age, mean ± *SD*	27.53 ± 7.08
Sex, *n* (%)	
Female	23 (51.1)
Male	22 (48.9)
Race and ethnicity, *n* (%)	
Caucasian	17 (37.8)
African American	17 (37.8)
Asian	7 (15.5)
Hispanic/ latino	4 (8.9)
Employment status, *n* (%)	
Student	17 (37.8)
Employed	20 (44.4)
Unemployed	8 (17.8)
Education, *n* (%)	
High school/ General education diploma	2 (4.4)
Some college / current student	13 (28.9)
College graduate	21 (46.7)
Advanced degree	9 (20)

that prevented their participation in the affective pain modulation task ($N = 33$).

The protocol was approved by the Johns Hopkins University and UCLA institutional review boards, and all subjects completed informed consent prior to participation.

Measures

Positive and Negative Affect Schedule-Expanded Form

The Positive and Negative Affect Schedule-Expanded Form (PANAS-X 32) is a 60-item self-report instrument that comprises a positive affect scale, a negative affect scale, and 11 additional affective subscales (see Supplemental Material). The measure has been well validated and is recommended for use in repeated measure designs.[32] Additional details are available in Supplemental Material.

Affective Pain Modulation

Participants were exposed to a standardized series of positive, negative, and neutral images in a three-block design, whereby each block contained 16 images of a single valence. A random selection of eight images were paired with thermal stimuli of 42°C, 44, 46C°C, or 48°C. Each thermal stimulus was used twice, in randomized order. Pain ratings on a 0–100 numeric rating scale[33] were obtained after each paired image/thermal stimulus presentation. Positive affective pain modulation is inferred if pain ratings decrease under positively valenced stimuli relative to neutral and/or negative valences. Similarly, negative affective pain modulation is inferred if pain ratings increase under negatively valenced stimuli relative neutral and/or positive valences. Additional task details are available in Supplemental Material.

Emotional Dot Probe

The emotional dot probe task measures AB to positive or negative emotional cues, relative to neutral cues, by measuring the reaction time for one's response to a dot presented for a brief duration following the presentation of emotional and neutral cues. A previously validated dot probe task[34] was used to measure AB to positive (pleasure-related) and negative (pain-related) emotional cues. Emotional cues were paired with neutral images on a computer screen for 50, 200, or 2000 ms. Subjects identify the location of a target probe replacing one or the other image via a button press, and reaction times are recorded. Negative values for the AB index indicate deflection of attention away from emotional stimuli, and positive values indicate focusing attention toward emotional stimuli. AB indices were calculated separately for positive and negative stimuli. Additional task details are available in Supplemental Material.

SWS

Minutes of SWS (Stage N3) was assessed through nocturnal polysomnography (PSG), which was conducted according to standard procedures[35] on all experimental nights in private rooms (for a complete description of PSG acquisition, see Supplemental Material). In order to minimize alpha inflation due to multiple testing, we focused on SWS as the primary mediator in our analyses because our prior study demonstrated that SWS, but not rapid eye movement (REM) or other non-rapid eye movement (NREM) stages, mediated the effect of FA, relative to restricted sleep, on positive affect.[10]

Psychomotor Vigilance Test

The psychomotor vigilance test (PVT) is a validated measure of sustained attention shown to be highly sensitive to sleep deprivation.[36] We used the 10-min version of the PVT[37] as a manipulation check. Additional task details can be found in Supplemental Material.

Study Design

The study design layout is featured in Figure 1. The study was within person, and the order of sleep condition was randomly determined and stratified on sex, body mass index (> vs. ≤25), age (18–32 vs. 33–48), and Chinese ethnicity (due to design considerations regarding morphine administration on a different day of the parent study). After an adaptation night in the clinical research unit (CRU), subjects received either one night of US or FA, followed by daytime assessments the next day. There was a minimum 2-week washout period between sleep conditions. At the return visit, subjects did not have an additional adaptation night prior to the sleep manipulation.

Subjects were given a "heart healthy" diet free of fried, high-fat, and high sodium foods during their stay in the CRU, and

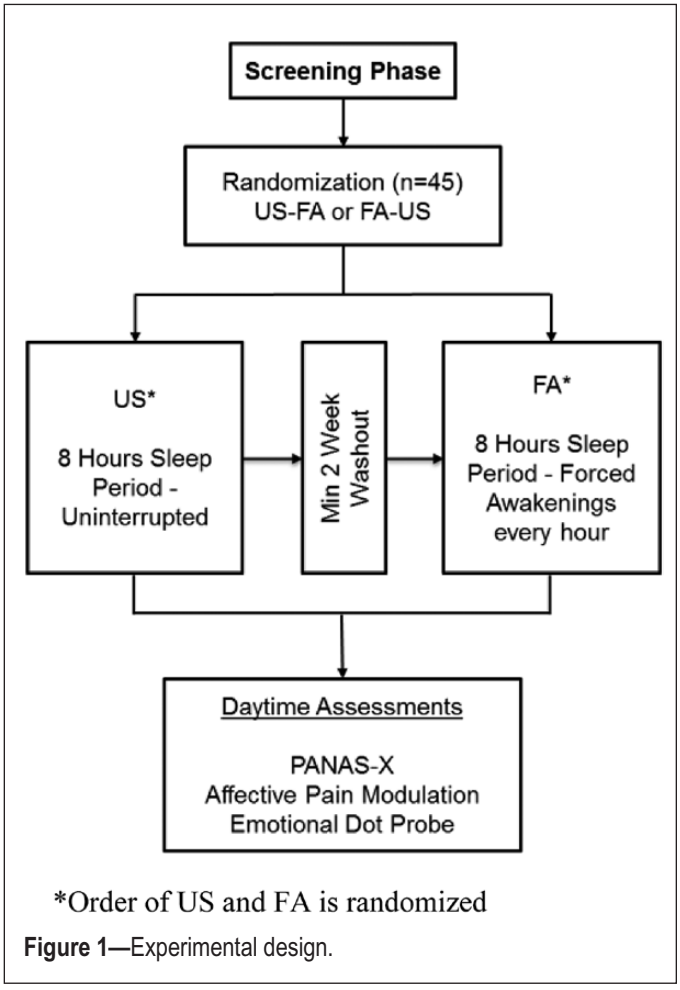

Figure 1—Experimental design.

lunch was typically served around 12 pm. Caffeine, nicotine, and alcohol were prohibited during the stay in the CRU. Subjects were not permitted to nap outside their defined sleep opportunity periods, which was between 23:00 and 07:00. Nursing staff checked on subjects every 30 min throughout the day to verify wakefulness.

US: Subjects slept undisturbed during an 8-hr sleep opportunity.

FA: Subjects underwent partial sleep deprivation via a standardized FA protocol developed by our group,[38] which serves as an experimental model of the type of partial sleep loss experienced by patients with severe insomnia or sleep disruptions associated with chronic pain. The night was divided into eight 1-hr intervals. One of the hour-long intervals was randomly determined to be a 60-min awakening, during which no sleep was permitted. The remaining seven, 1-hr intervals were subdivided into thirds (20-min intervals), and one 20-min interval was randomly selected within each hour as an FA period. During assigned FA periods, nursing staff awakened subjects and kept them awake for the entire interval. Subjects were asked to sit up in bed with the lights on to reduce the chance of microsleep. Although nocturnal light exposure could alter circadian regulation of the sleep–wake cycle, and perhaps influence affect independent of its effect on sleep,[39] we decided to allow lights during awakenings in order to ensure that subjects remain awake throughout the entire period, thereby preserving the integrity of the manipulation. The maximum total sleep time possible was 280 min. PSG monitoring was maintained for the entire sleep opportunity period. Subjects were not permitted to the leave the inpatient unit during sleep deprivation periods and were under continuous nursing supervision/monitoring (day and night) to prevent naps and ensure safety.

Daytime Assessments

Daytime assessments began in the morning (typically between 10:00 and 11:30 am) and lasted approximately 3–3.5 hr. There was variation in assessment start time due to staff availability. Daylight was permitted during testing. Temperature was not controlled due to competing demands of other studies across the CRU.

Data Analytic Plan

Following guidelines and specifications for longitudinal data,[40] mixed-effects modeling was used to evaluate hypotheses regarding self-reported affect and affective pain modulation.

Because the AB index has been shown to have poor test–retest reliability,[41] we determined it was not appropriate to conduct within-person analyses on this measure. As such, for analyses involving positive and negative AB, we isolated the first inpatient visit and conducted between-group analyses of covariance comparing effects of FA to US.

Mediation of the effect of sleep condition on affect and affective pain modulation was evaluated using the MacArthur approach.[42]

For primary analyses, both p value and effect size were used to interpret the significance of study hypotheses. This decision was reached following a recent consensus statement from the American Statistical Association,[43] which called for the evaluation of statistical significance based on a combination of p values and measures of effect size, and encouraged researchers to avoid sole reliance on p values. Additional details of the full data analytic strategy, including model specifications and covariate selection, can be found in Supplemental Material.

RESULTS

Demographics

Demographic data are provided in Table 1. The sample was generally composed of young, educated adults, evenly split between males and females, and racially and ethnically diverse (62% non-Caucasian).

Sleep Manipulation Check

FA was associated with significantly lower total sleep time than US ($p < .001$), which follows the specifications of the manipulation. FA was associated with significantly fewer minutes of Stage N2, N3, and REM compared with US ($p < .001$). FA was associated with a significantly lower reciprocal response time ($p < .001$), indicating decreased vigilance and consistent with PVT effects reported in other sleep deprivation studies.[37] There was a statistical trend for more attention lapses ($p = .10$), defined as periods of >500 ms before a subject responds to the cue. Together, these data suggest that FA successfully lowered total sleep time, altered sleep architecture, and impaired attention. Descriptive statistics for total sleep time, sleep stages, and sustained attention, as a function of sleep condition, are provided in Table S2.

Effects of Forced Awakenings on Positive and Negative Affect

Primary Positive and Negative Affect Scales

Positive affect was significantly lower following FA (22.12 ± 8.42, mean ± *SD*) compared to US (26.44 ± 7.85; $p < .001$), as displayed in Figure 2. Comparison of the residual variance components of the null model ($\sigma = 27.51$) and the conditional model ($\sigma = 16.47$) yielded a pseudo R^2 of 0.40, indicating that 40% of the explainable within-person variance in positive affect was explained by the FA manipulation. Based on Cohen's[44] convention of comparing means, the difference between US and FA was equivalent to $d = -.53$, which is considered a medium-sized effect.

Negative affect was not different between sleep conditions (FA: 11.07 ± 1.87; US: 10.80 ± 2.27; $p = .40$). Variance components of the null ($\sigma = 2.30$) and conditional ($\sigma = 2.34$) models yielded a pseudo R^2 of −0.02, indicating sleep condition poorly characterized variation in negative affect. The measure of mean differences supported the pseudo R^2 estimate, with Cohen's $d = 0.13$, which is considered a very small effect size.[44] All covariates were nonsignificant in both models and therefore were not included in final estimates.

These findings suggest that a single night of sleep continuity disruption is detrimental to positive, but not negative, affect. Post hoc analyses conducted on each of the positive and negative affect subscales revealed similar patterns of effect across discreet positive emotion categories, including joviality, serenity, self-assurance, and attentiveness, which together suggest

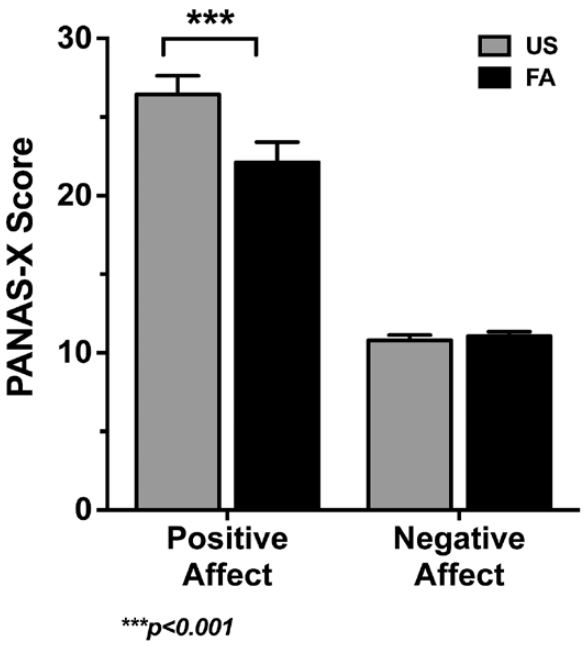

Figure 2—Effects of sleep continuity disruption on positive and negative affects. Means and standard errors of the means are presented for positive affect (PA) and negative affect (NA) reported following one night of uninterrupted sleep (US) and one night of forced awakenings (FA).

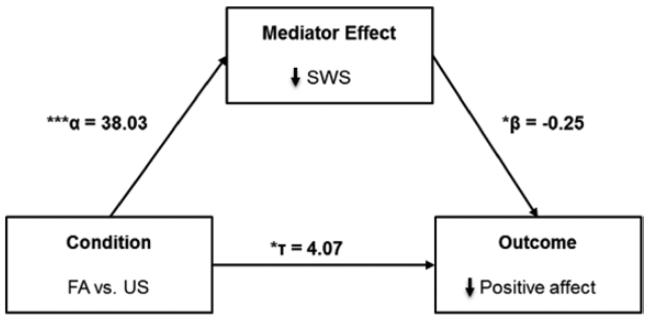

Figure 3—Effect of sleep continuity disruption on positive affect is mediated by loss of slow wave sleep. Regression coefficients for each main effect are presented for each path.

that FA broadly influences positive affect (see Supplemental Material: Supplemental Results).

Test of Mediation by Slow Wave Sleep

According to Kraemer et al.,[42] if the temporal sequencing of predictor (Condition), mediator (ΔSWS), and outcome (Positive Affect) is sequential, and the predictor is significantly associated with the putative mediator, mediation is supported by the presence of either a significant main effect of the mediator or an interaction of predictor and mediator (Condition × ΔSWS). Our design satisfied the temporal sequencing criterion, as the sleep condition caused changes in SWS that temporally preceded the measurement of positive affect. Minutes of SWS were significantly lower in FA (60.01 ± 26.22) compared to US (97.82 ± 43.10; $p < .001$), Cohen's $d = 1.06$, establishing an association of the target variable (ie, sleep condition) and the mediator (ie, SWS). Furthermore, there was a main effect of ΔSWS ($p = .02$) and a Condition × ΔSWS interaction ($p = .02$). Minutes of Stage N2 and minutes of REM sleep, which were both significantly lower in FA compared to US ($ps < .001$; see Supplemental Material), were not significant covariates ($ps > .29$) and were therefore not included in the final model. Together, these findings suggest that the reduction in SWS following FA mediated the effect of FA on positive affect. This effect is graphically displayed in Figure 3.

Post Hoc Tests of Mediation by Stage N2 and REM

Although the a priori hypothesis was that ΔSWS would mediate the loss of positive affect engendered by FA, the significant difference between FA and US in minutes of both Stage N2 and REM led us to test them as mediators in separate post hoc analyses. Neither the main effect of ΔN2 ($p = .75$) nor the interaction of Condition × ΔN2 ($p = .58$) was statistically significant, suggesting that the loss of minutes of Stage N2 did not mediate the effect of FA on positive affect. In contrast, the main effect of ΔREM ($p = .01$) and the interaction of Condition × ΔREM ($p = .03$) were statistically significant, suggesting that ΔREM accounted for a significant proportion of variance in the effect of FA on positive affect.

Effects of FA on Affective Modulation of Pain

A manipulation check revealed that subject ratings of valence and arousal were consistent with published norms (See Supplemental Material: Supplemental Results), indicating a successful manipulation of affect. Additionally, pain intensity increased as a function of stimulus temperature ($p < .001$).

Primary Tests of Affective Pain Modulation

Table S3 presents the means and *SDs* of pain intensity as a function of image valence, temperature, and sleep condition. To establish a baseline, we first modeled the effect of image valence on pain intensity following US. The omnibus main effect of valence on pain intensity ratings (across all temperatures) was significant ($F = 8.55$, df [Satterthwaite] = 753.04, $p < .001$). Contrasts revealed that positive images were associated with significantly lower pain intensity relative to both neutral ($p = .003$) and negative ($p < .001$) images. Negative images were not significantly associated with differences in pain intensity compared with neutral images ($p = .30$).

Next, we used a within-subject, mixed-effects model to test the effects of FA, relative to US, on positive and negative affective pain modulation. The omnibus condition main effect was significant ($F = 53.27$, df [Satterthwaite] = 1566.86, $p < .001$), with participants reporting lower pain across valences in the FA condition compared to US. Condition × Valence interaction was statistically significant ($F = 5.02$, df [Satterthwaite] = 1533.38, $p = .007$), indicating that the effect of valence on pain intensity significantly varied as a function of sleep condition. The effect, averaged across stimulus temperatures, is graphically displayed in Figure 4 and is further supported by a post hoc analysis showing a main effect of valence in the US condition ($p < .001$) that was not present in FA ($p = .92$). Due to the number of parameters in this model, pseudo R^2 is not the optimal choice

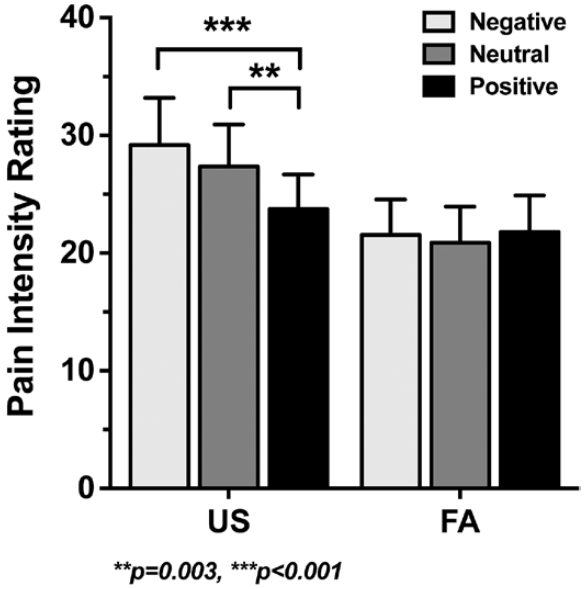

Figure 4—Effects of sleep continuity disruption on affective pain modulation. Means and standard errors of the means are presented for pain intensity associated with positive, negative, and neutral affective conditions, following one night of uninterrupted sleep (US) and one night of forced awakenings (FA).

to evaluate effect size. Rather, we report effect size as a post hoc Cohen's *d* comparing pain intensity reported during positive versus negative and positive versus neutral images within each sleep condition. Table S3 presents each of these effect sizes, which in summary show small- to medium-sized effects of positive valence at 42°C, 44°C, and 46°C following US, and effects in the small to very small range at each temperature following FA. Examination of the effect sizes by temperature suggests that affect did not substantively modulate pain at 48°C following either US or FA. However, the Condition × Valence × Temperature interaction was not significant (p = .83). Overall, these findings suggest that FA attenuated the pain inhibitory effect of positive affective images but did not amplify the pain facilitative effect of negative affective images.

Test of Mediation by Sleep Architecture

Mixed-effects models revealed that neither the main effect of ΔSWS (p = .55) nor the interaction of Condition × Valence × ΔSWS (p = .56) was significant, indicating that ΔSWS did not mediate the effects of FA on affective pain modulation. Similarly, the main effects of ΔREM (p = .74) and ΔN2 (p = .90) and the interaction of Condition × Valence × ΔREM (p = .16) and Condition × Valence × Δ2 (p = .17) were not significant, indicating that neither ΔREM nor ΔN2 mediated the effects of FA on affective pain modulation.

Effects of FA on Affect-Biased Attention

Test of Group Differences in Positive Attentional Bias

Table S4 presents the AB index values at each stimulus duration as a function of sleep condition. Because we did not have a priori hypotheses regarding group differences as a function of stimulus durations, we averaged across them for this analysis. There was a medium-sized[45] main effect of sleep condition on positive AB (p = .09, partial η^2 = .07, Cohen's d = 0.55), reflecting a deflection of attention away from positive emotional stimuli following FA.

Due to the small sample in this between-subject analysis and, consequently, low power, we did not test for mediation by sleep architecture.

Test of Group Differences in Negative Attentional Bias

Sleep conditions did not significantly differ in negative AB at any stimulus duration (p = ..88, partial η^2 = .001, Cohen's d = −0.06).

DISCUSSION

The present within-person sleep continuity disruption experiment yielded three novel findings. First, we demonstrated that a single night of sleep continuity disruption reduced perceived positive affect relative to a night of uninterrupted sleep within-subject and that within-subject reductions in SWS accounted for a significant proportion of variance in that effect. Second, using an affective pain modulation task, we demonstrated that sleep continuity disruption blunted the inhibition of pain by positive affect. Third, we demonstrated that sleep continuity disruption attenuates AB to positive affective stimuli. Across each measurement domain, our results also showed that one night of sleep continuity disruption failed to augment negative affective processes.

The finding that FA-associated changes in SWS mediated the effect of FA on self-reported positive affect meaningfully extends a recent finding that three nights of FA caused larger reductions in positive affect compared to sleep restriction and that the group difference was mediated by differences in the extent of SWS lost.[10] The present findings differ from that study in several respects. First, the present study employed a within-subject crossover design, comparing FA to US in the same subjects. This strengthens the inferences that can be drawn from changes associated with FA. Second, the present study demonstrates that only one night of sleep continuity disruption is necessary to substantially alter positive affect. Third, and perhaps most importantly, the present results show that while positive affect was attenuated following one night of FA, negative affect was generally unchanged. A major distinction in the two studies is that the prior study utilized the Profile of Mood States-Bipolar[46] to assess positive and negative affect, whereas the present study employed the PANAS-X.[32] Although both measures are similar in form (ie, self-reported adjective lists), and have been shown to correlate in validity studies,[32] it is possible that the negative affect scale on the POMS-BI, which incorporates several low activation items reflecting a state of tiredness and confusion is more sensitive to FA than that of the PANAS-X, which primarily indexes high activation items reflecting a state of tension and anxiety. Interestingly, our results support those reported by Talbot et al.[18] who used the PANAS to evaluate affective response to a single night of sleep restriction and found a robust reduction of positive affect and almost no change on negative affect following one night of sleep restriction.

The finding that SWS mediated the effect of sleep continuity disruption on positive affect raises the possibility that SWS could be manipulated for affective therapeutic benefit. Gabaergic medications, such as gaboxodal[47] and tiagabine,[48,49] enhance slow wave activity and transitions to Stage N3 in both experimental models and clinical trials of patients with insomnia. Additionally, neurostimulation therapies, such as transcranial direct current stimulation, can be applied during sleep to entrain brain waves to the slow rhythmic oscillations of an extracranial electrical field, thereby increasing the propensity to achieve SWS.[50,51] To our knowledge, no studies have investigated whether such pharmaceutical or neurostimulation interventions aid in the maintenance of positive affect in insomnia patients. Our present findings warrant further study of these possible therapeutic mechanisms, particularly in patients with comorbid insomnia and depression.

Although the SWS findings support and extend our previous report, we also found in the present study that changes in REM from FA to US mediated the differential effects of sleep condition on positive affect. This additional finding suggests that SWS may not be a unique mechanism of the effect of FA on positive affect and raises a few possibilities that should be investigated in future studies. First, it could be that both SWS and REM changes following FA are capturing overlapping variation in sleep loss, and therefore their respective mediation effects may represent nonspecific findings related more to the loss of sleep caused by the FA manipulation than to specific changes in sleep architecture. However, the absence of Stage N2 mediation argues against this possibility. It is also possible that FA-induced changes in REM and SWS account for independent portions of variance in next day positive affect. To adequately test this hypothesis, future studies should compare the effects of selective REM deprivation versus selective SWS on next day positive affect levels.

Another major goal of the present study was to evaluate the extent to which findings on self-reported affective state generalized to other more implicit measures of affective function. Affective pain modulation reflects the acute changes in pain perception that accompany exposure to emotionally evocative stimuli. Evoked positive affect typically reduces pain sensitivity and evoked negative affect typically increases pain sensitivity.[20,52] Here we observed a pattern of blunted affective pain modulation across valences following FA that not only parallels the self-reported positive and negative affect results in this study but also supports data from other studies that have shown blunted affective pain modulation in patients with fibromyalgia[26] and individuals with severe insomnia symptoms,[29] relative to controls. Notably, frequent nocturnal awakenings are commonly reported in both fibromyalgia and insomnia.[53–56] To our knowledge, ours is the first study to demonstrate alterations in affective pain modulation as a function of experimental sleep continuity disruption.

Although the emphasis of our analyses was to explicate the difference between sleep conditions on positive affective pain inhibition, it is notable that pain ratings across temperatures and valences were significantly lower following FA compared to US. This finding is inconsistent with prior literature, which has demonstrated that sleep deprivation increases pain sensitivity on measures of threshold and tolerance.[57] However, we are mindful not to directly compare our study with others that explicitly set out to investigate the effect of sleep deprivation on pain sensitivity; the pain ratings from our analyses were all obtained in the context of a competing affective stimulus. It is possible that the lower pain ratings following FA were driven by a reduction in vigilant attention, suggesting a possible role of distraction in modulating pain ratings across valence conditions. The possibility of a transvalence distraction effect does not preclude the interpretation that FA was associated with attenuated positive affective pain inhibition.

Notably, changes in sleep architecture did not significantly mediate the effect of FA on positive affective pain inhibition. It is not clear why SWS and REM influenced the perceived positive affective response to sleep continuity disruption but failed to engage the process of affective pain modulation. One possibility is that SWS and REM are more closely linked to the individual's ability to generate positive emotions than the ability to react to positive valenced stimuli; this could be explicitly tested in future studies. Furthermore, the positive stimulus content in the affective pain modulation task was limited to high activation erotic slides. It is possible that SWS and REM are more important for reactivity to other types of positive stimuli, such as humor or music.

Findings on measures of affect-biased attention were remarkably similar to those observed with the other two affective measures in that sleep continuity disruption attenuated the degree of attention toward positive affective stimuli without augmenting the degree of attention toward negative affective stimuli. Given known effects of attention on emotion regulation,[27] deflection of attention away from positive emotional information might explicate the FA-induced erosion of positive affect observed in this study. This finding stands in contrast to that reported by Anderson and Platten,[58] in which a single night of total sleep deprivation resulted in enhanced reactivity to negative emotional stimuli in a Go/No-Go task. Future studies may expand upon this finding by employing eye-tracking and event-related potentials to assess the subtle effects of FA on affect-biased attention. These psychophysiological measures may have comparatively greater sensitivity to assess the cognitive impacts of sleep disruption than the reaction time measures used in the present study.

Additional studies are needed to understand whether the present findings are specific to sleep continuity disruption or instead represent a more general phenomenon following sleep loss. Talbot et al.[18] conducted a partial sleep deprivation study by delaying sleep onset and found results on the PANAS very similar those of the present study. However, a previous study from our group[10] found results that slightly deviated from the present findings, with the restricted sleep group (via delayed onset bedtime) showing both a decrease in positive mood and an increase in negative mood, assessed via the POMS-Bi, after one night. The literature is not yet mature enough to confidently conclude whether simply changing the structure of sleep deprivation reliably leads to different outcomes. More likely, the effects of sleep deprivation on affect are driven by a combination of the structure of sleep deprivation and the types of measures used to assess affect. More studies are needed to understand these nuances.

This study had several limitations that should be weighed against the evidence. First, due to the fact that this study was

embedded in a parent study that included a second night of each sleep condition and a different battery of daytime testing measures, we could not assess the effects of recovery sleep on the present findings. Second, because we allowed light exposure during the FA procedure to promote feasibility, the sleep–wake cycle may have been artificially influenced by study procedures, thereby limiting generalizability to settings in which individuals experience awakenings in the absence of light. Future studies should determine whether nocturnal light exposure during wake after sleep onset periods influences next day affect experiences independent of the awakenings themselves. Third, generalizability may be limited by a lack of clinical comparison group and a relatively young, well-educated sample. Sleep quality and architecture are known to change with age, so it is possible that the findings could differ with a substantially older sample. These limitations are balanced by several strengths, including a rigorous within-subject design, a multimodal assessment strategy, and a racially and ethnically diverse sample.

SUMMARY AND CONCLUSIONS

The present study is, to our knowledge, the first to systematically evaluate the effects of sleep continuity disruption on positive affect via a multimodal assessment of affective processes. The results indicate that a single night of sleep continuity disruption lowers positive affect levels, blunts the inhibition of pain by positive affect, and deflects attention away from positive affective stimuli. Furthermore, the results demonstrate that reductions in both SWS and REM accounted for a significant proportion of variance in the FA-induced reduction in self-reported positive affect.

We believe the present findings could advance the field of behavioral medicine in several ways. Our results make it clear that the positive affect system is threatened by sleep continuity disruption. Therefore, a greater clinical focus should be placed on assessing the positive affective states of patients with sleep disorders, such as insomnia or sleep apnea. Bolstering patients' positive affective resources could be a clear target of treatment[59] that may otherwise be overlooked if not properly assessed.

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SUPPLEMENTARY MATERIAL

Supplementary Material is available at *SLEEP* online.

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DISCLOSURE STATEMENT

None declared.