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Nocturnal cognitive arousal is associated with objective sleep disturbance and indicators of physiologic hyperarousal in good sleepers and individuals with insomnia disorder.

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Abstract

Background.—Cognitive arousal is central to models of sleep disturbance and insomnia, but findings are mixed regarding whether cognitive arousal is associated with objective sleep disturbance and physiologic hyperarousal. This study explored associations of objective nocturnal wakefulness and indicators of physiologic hyperarousal with cognitive arousal in healthy sleepers and individuals with insomnia.

Methods.—Fifty-two adults (51.9% women; 18 with insomnia disorder, 34 healthy sleepers) underwent two overnight polysomnography (PSG) studies (adaptation + baseline nights) and a multiple sleep latency test (MSLT). Baseline depression was assessed and presleep cognitive arousal and somatic arousal were recorded for each night. Multivariate regression was used to evaluate associations of PSG sleep parameters with insomnia, cognitive arousal, and somatic arousal.

Results.—Analyses showed that high levels of nocturnal cognitive arousal were associated with prolonged sleep latency, lower sleep efficiency, and shorter total sleep time by PSG on both nights. An association between nocturnal cognitive arousal and wake after sleep onset was observed on night one only. Moreover, greater nocturnal cognitive arousal was associated with greater likelihood of obtaining short sleep and with longer MSLT sleep latencies. Insomnia diagnosis, depression, and somatic arousal were not associated with PSG sleep parameters or MSLT latency.

Conclusions.—Heightened cognitive arousal at night is linked to objective sleep disturbances and indicators of physiologic hyperarousal at night and during the day. For patients with insomnia,

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cognitive arousal may contribute to the 24-hr physiologic hyperarousal. Cognitive arousal may be a critical therapeutic target for severe or treatment-resistant sleep disturbance.

Keywords

rumination; worry; cognitive arousal; mslt; psg; polysomnography

INTRODUCTION

Cognitive arousal is a central feature of sleep disturbance and insomnia.[1–9] Various forms of cognitive arousal, such as rumination and worry, have been implicated in the etiology and maintenance of sleep disturbances, and have been linked to elevated risk for common insomnia comorbidities including anxiety,[10–13] depression[10,11,14–17] and cardiovascular disease.[18–21]

Much of what we know about cognitive arousal in sleep disturbance is based on studies that have relied on self-reported sleep symptoms. These studies led to critical discoveries in the field including the identification of the robust relationship between cognitive arousal and reported sleep quality,[22–26] determining that ruminative thinking is common in the late evening and night,[27,28] that cognitive arousal is closely linked to difficulty *falling* asleep,[17,29–31] and that cognitive arousal facilitates or even exacerbates the effects of sleep disturbance/insomnia on future depression.[16,32–33] As insomnia disorder is diagnosed based on patient reports of sleep disturbance, these studies offer critical insight into the cognitions and behaviors contributing to the clinical presentation of insomnia. However, self-reported sleep latency and wake after sleep onset are often inconsistent with objective sleep data.[34,35] Individuals with insomnia, by definition, report long periods of nocturnal wakefulness and/or insufficient sleep. Yet, an estimated one-third to one-half of insomnia patients overestimate their nocturnal wakefulness relative to objective measurement methods.[35,36] Thus, some insomnia researchers have proposed phenotyping insomnia into at least two categories: insomnia with or without objective sleep disturbance.[34,36] This is an important distinction as insomnia patients with objective sleep disturbance have poorer prognosis, are more resistant to treatment, and are at greater risk for psychiatric and medical morbidities as compared to insomnia patients with normal objective sleep.[34,37–39] Identifying factors associated with objective sleep disturbance is necessary to inform conceptual models specific to the etiology and maintenance of severe insomnia phenotypes.

Emerging evidence suggests that cognitive arousal, particularly when active in the presleep period, is associated with objective nocturnal wakefulness. Inducing rumination prolongs PSG and actigraphy-defined sleep latency,[31,40–43] and naturally occurring rumination at night is associated with actigraphy-based sleep latency.[30] Although cognitive arousal appears most closely associated with sleep latency, some PSG studies show that high cognitive arousal also corresponds to longer wake after sleep onset, lower sleep efficiency, and shorter sleep duration.[40,42,44,45]

Despite the burgeoning literature in cognitive arousal and insomnia, important gaps remain in our current understanding. PSG studies have typically measured *trait* cognitive characteristics (rumination, worry) or induced *state* rumination and worry. Given daily

variations in stress and nightly variability in sleep,[46] assessing state rumination preceding the sleep period across multiple nights may be important for understanding its association with nightly objective sleep symptoms. While inducing state rumination is necessary to determine causal relations, rumination inductions often directly influence mood itself (e.g., speech threats increase fear and reduce positive affect), thereby confounding results. Studies that assess naturally occurring rumination during the presleep period and involve multiple overnight assessments are needed to best identify objective sleep symptoms associated with nocturnal cognitive arousal. In addition, no studies have explored the associations of state nocturnal rumination with overnight PSG parameters and daytime multiple sleep latency test (MSLT) latencies. Given that objective short sleep duration (< 6 hrs) is a nocturnal indicator of physiologic hyperarousal (particularly within the context of insomnia)[34] and that longer latencies on MSLT indicate daytime physiologic hyperarousal,[47,48] then assessing overnight PSG and MSLT within the same participants will offer important insights into the potential 24-hr physiologic hyperarousal associated with nocturnal cognitive arousal.

The present study examined the associations of state-level nocturnal cognitive arousal with objective sleep disturbance and indicators of hyperarousal. Healthy sleepers and patients with insomnia disorder slept in a sleep center for two nights of PSG monitoring, and then underwent an MSLT. We hypothesized that higher cognitive arousal at night would be associated with longer sleep latency, greater wake after sleep onset, lower sleep efficiency, and shorter sleep duration on PSG. We also predicted that higher nocturnal cognitive arousal would be associated with objective short sleep duration of < 6 hrs on both PSG nights and with longer sleep latencies on MSLT.

METHODS

Participant recruitment and screening

The present study was conducted in a large multiple-hospital health system in Metro Detroit, Michigan, USA. All procedures were approved by the institutional review board. Participants were recruited from the community via media advertising (online, newspapers, radio) from June 2004 through July 2007. Inclusion criteria included age 18-64 years. Exclusion criteria included: self-reported loud snoring, hypnotic use within past month, > 250 mg of caffeine per day (based on self-reported average consumption of caffeinated beverages), current or past diagnosis of Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition[49] (DSM-IV) major depression or other major psychiatric illness (per structured clinical interview for DSM-IV [SCID]), alcohol or substance use disorders (per SCID), major medical conditions (renal, cardiovascular, hepatic, metabolic, gastrointestinal, or neurological disorders), diagnosis of any sleep disorder other than insomnia (per patient report in a clinical interview or via PSG adaptation night in laboratory, see below), and habitual bedtime that deviated by ± 2 hours from 23:00. These criteria were evaluated in a 3-step process involving phone pre-screening, in-person clinical interview, and finally the overnight PSG adaptation night. Phone pre-screening was used to assess sociodemographic characteristics, age, hypnotic use, caffeine and other substances, and sleep disorder diagnoses. Individuals who passed pre-screening then underwent an in-person sleep and psychiatric clinical interview conducted by a clinical psychologist board certified

in sleep medicine, which involved confirming pre-screening data, ruling out a lifetime history of DSM-IV major depression, and assessing for current DSM-IV insomnia disorder.

PSG screening & study procedures

Participants who passed the in-person screening interview then underwent a PSG adaptation/screening night with a montage routinely used to detect sleep disorders other than insomnia. Apnea events were defined as an interruption in airflow of ≥ 10 seconds and hypopnea events were defined as airflow decrease of ≥ 50% with blood oxygen desaturation of ≥ 4%. Having an apnea-hypopnea index score > 10 was an exclusion criterion. Only periodic limb movements in sleep associated with arousals (arousal must have begun after leg movement and unexplained by other factors such as respiratory events) were considered for exclusionary purposes. Having periodic limb movement with arousal index scores > 10 was an exclusion criterion (we had no criteria related to periodic limb movements without arousals). No participants were disqualified according to these criteria. The second PSG night was a baseline night. For all participants, bedtime was 23:00 and rise time was 07:00, which was implemented to ensure that all subjects had a uniform waketime before their MSLT. Upon waking after both nights, participants completed surveys prior to being unhooked from PSG. After the baseline night, participants underwent a 4-nap MSLT testing starting at 09:00 with 2 hours between naps.

Measures

Baseline measures—DSM-IV Insomnia Disorder was diagnosed via in-person interview with a clinical psychologist board certified in sleep medicine. Diagnostic criteria included: (A) difficulty falling or staying asleep or nonrestorative at least three times per week sleep for ≥ 1 month; (B) sleep problems and associated daytime fatigue causes significant distress or impairment in daytime function; (C) sleep disturbance is not better explained by another sleep disorder, mental illness, substance use, or medical condition.

Self-reported sleep parameters were determined by participants retrospectively reporting their habitual sleep latency (in minutes), nighttime awakenings (average nightly occurrences), wake after sleep onset (in minutes), and total sleep time (in hours and minutes).

Insomnia severity was measured using the *Insomnia Severity Index* (ISI),[50] which assesses symptoms over the previous 2 weeks. Total scores on the ISI range from 0 to 28 with higher scores indicating greater severity. A cut-point of ISI ≥ 10 suggests clinical insomnia.[51]

Global sleep quality was measured using the *Pittsburgh Sleep Quality Index* (PSQI),[52] an 18-item questionnaire that assesses multiple facets of sleep quality over the prior month. These areas of sleep include sleep duration, sleep latency, sleep efficiency, subjective sleep quality, sleep interfering-behaviors, and daytime impairment. Scores range from 0 to 21 with higher scores indicating poorer sleep quality. A cut-point of PSQI > 5 indicates significantly poor sleep.

Depression.: *Depressive symptoms* were measured using the *Patient Health Questionnaire-9* (PHQ-9),[53] which is a 9-item self-report measure of depression that assess symptoms over

the previous 2 weeks. PHQ-9 scores range from 0 to 27, with higher scores indicating greater severity. A cut-point of PHQ-9 ≥ 10 suggests probable major depression.

Objective overnight sleep and daytime assessments

Polysomnography (PSG) sleep parameters/nocturnal wakefulness.: PSG data were scored by a certified sleep technician according to standard procedures by American Academy of Sleep Medicine's (AASM) 2007 manual for the scoring of sleep and associated events.[54] Sleep studies were interpreted by a clinical psychologist board certified in sleep medicine. Sleep measures included sleep latency (minutes from lights out to first epoch of sleep), latency to persistent sleep (minutes from lights out to first 10 minutes of continuous sleep), wake after sleep onset (minutes awake post onset and pre-final awakening), sleep efficiency (% of time in bed asleep), and total sleep time (sleep duration).

Multiple sleep latency test (MSLT).: MSLT measurements were performed based on standard procedures by Carskadon et al.[55] On the morning after the PSG baseline night, MSLT naps began at 09:00 and continued at 2-hour intervals for a total of 4 nap trials. The dependent measure for the MSLT was mean sleep latency (minutes) to the first epoch of any stage of sleep. Longer MSLT sleep latency is a common indicator of daytime physiologic hyperarousal in insomnia (see reviews by Bonnet and Arand and Roehrs et al.).[48,56]

Nocturnal cognitive arousal.: The *Presleep Arousal Scale Cognitive* factor (PSAS-C)[57] was administered each morning upon waking from overnight PSG. The PSAS-C measures habitual, trait-like tendency to experience cognitive arousal and rumination during the presleep period at night. For this study, we modified the instructions to ask about experiences specific to while they were trying to fall asleep in laboratory the night before. The PSAS-C consists of 8 items (e.g., 'review or ponder events of the day' and 'can't shut off your thoughts') and scores range from 8 to 40 with higher scores indicating greater presleep cognitive arousal. We are not aware of any clinical cutoffs for the PSAS-C, but multiple untreated insomnia samples have reported mean PSAS-C scores > 16 (i.e., mean item response > 2 indicating 'moderately' to 'extremely'), whereas average PSAS-C scores for good sleepers is < 13.[58–61] In the present study, we classified participants who scored > 16 on the PSAS-C as having *high cognitive arousal*, whereas those who scored PSAS-C ≤ were classified as having *low cognitive arousal*. Notably, PSAS-C scores > 16 indicate a mean item-level response higher than 'slightly.'

Nocturnal somatic arousal.: The *Presleep Arousal Scale Somatic* factor (PSAS-S)[57] was administered in the morning. The PSAS-S measures trait-like experiences of somatic arousal during the presleep period at night (e.g., heart racing or muscle tension while trying to fall asleep). For this study, we modified the instructions to ask about experiences specific to while they were trying to fall asleep in laboratory the night before. The PSAS-S consists of 8 items and scores range from 8 to 40 with higher scores indicating greater presleep somatic arousal.

Analyses—All analyses were conducted in SPSS version 25 (IBM Corporation, Armonk, NY). We first characterized sociodemographics, sleep symptoms, depressive symptoms,

nocturnal cognitive arousal, and nocturnal somatic arousal for the sample. Next, we conducted a series of independent samples t-tests to characterize differences between insomnia participants and good sleepers on nocturnal cognitive arousal, nocturnal somatic arousal, and objective sleep disturbance parameters for both overnight sleep studies. We then conducted a series of multivariate linear regression models. We regressed PSG sleep parameters on insomnia diagnosis and depressive symptoms to investigate associations of objective sleep disturbance with insomnia diagnosis, depressive symptoms, prior night's cognitive arousal, and prior night's somatic arousal. In addition, we ran logistic regression models predicting objective short sleep, which we operationalized as < 6 hrs of nightly sleep (coded: 1 = < 6 hrs; 0 = ≥ 6 hrs). These regression models were first run with data from the *adaptation night*, then rerun with data from the *baseline night* to allow for evaluation across multiple nights. We then ran a multivariate linear regression model estimating average sleep latency on MSLT as predicted by the prior night's cognitive arousal, prior night's somatic arousal, and PSG-defined total sleep time, while controlling for higher order insomnia (diagnosis) and depression (symptoms). Notably, age, gender, and BMI were considered as covariates in regression models, but were not significantly associated with the outcome variables and thus not included in the models presented below. Lastly, because multivariate regression models identified nocturnal cognitive arousal as robust predictor of objective sleep, we conducted a series of *posthoc* independent samples t-tests to describe differences between participants with high vs low cognitive arousal (irrespective of insomnia diagnosis) on PSG sleep parameters on the baseline night only for descriptive purposes.

RESULTS

Sample characteristics (Table 1)

Fifty-two adults (51.9% women; 18 participants with DSM-IV insomnia, 34 healthy sleepers) participated in our study. Participants' ages ranged from 18 to 51 years (30.2±9.9). Most of the sample self-identified racially as non-Hispanic white (61.5%) or non-Hispanic black (23.1%). Eighteen participants were diagnosed with DSM-IV insomnia disorder (34.6%). Participants with vs without insomnia did not differ on gender (p=.70), age (p=.06), or BMI (p=.88). As expected, participants diagnosed with insomnia reported longer habitual sleep latency and wake after sleep onset, more frequent nighttime awakenings, and shorter nightly sleep duration/total sleep time relative to those without insomnia (Table 1). Similarly, insomnia participants reported poorer sleep quality on the PSQI (Cohen's d=2.50), greater global insomnia symptoms on the ISI (Cohen's d=3.34), and greater depressive symptoms on the PHQ-9 (Cohen's d=1.57; Table 1).

Differences in nocturnal cognitive arousal, somatic arousal, and objective sleep based on insomnia diagnosis

Participants with insomnia disorder, relative to healthy sleepers, reported higher levels of nocturnal cognitive arousal while trying to fall asleep on the adaptation night (Cohen's d=.95) and baseline night (Cohen's d=1.07; Table 2). Similarly, insomnia participants reported greater somatic arousal on the adaptation night (Cohen's d=.64) and the baseline night (Cohen's d=.94; Table 2).

PSG-defined sleep parameters are reported for the full sample and by insomnia status in Table 2. After conducting independent samples t-tests, we observed no significant differences in sleep latency, wake after sleep onset, sleep efficiency, or total sleep time on the adaptation night. On the baseline night, we again observed no significant group differences, although several group differences approached significance on the baseline night in the direction suggestive of objective sleep disturbance being greater among insomnia participants than healthy sleepers.

Objective sleep disturbance, nocturnal cognitive arousal, and somatic arousal.

We next conducted a series of multivariate linear regression models to evaluate associations of objective sleep disturbance with nocturnal cognitive arousal and somatic arousal while controlling for insomnia diagnosis and depressive symptoms for both the adaptation and baseline nights.

Sleep latency.—Nocturnal cognitive arousal was associated with longer sleep latency on the adaptation night (β=.37, p=.04) and the baseline night (β=.52, p=.003), whereas insomnia diagnosis, depressive symptoms, and somatic arousal were not significant (Table 3). Multivariate linear regressions predicting latency to persistent sleep revealed a similar pattern of results such that nocturnal cognitive arousal was associated with longer latency to persistent sleep (Adaptation: β=.51, p=.004; Baseline: β=.51, p=.003), whereas insomnia diagnosis, depression, and somatic arousal were again non-significant.

Wake after sleep onset.—On the adaptation night, nocturnal cognitive arousal was associated with longer wake after sleep onset (β=.48, p.007), whereas this association was not significant on the baseline night (β=.23, p=.19). Insomnia diagnosis, depression, and somatic arousal were not associated with wake after sleep onset on either night (Table 3).

Sleep efficiency.—Greater nocturnal cognitive arousal was significantly associated with lower sleep efficiency on both nights (Adaptation: β=−52, p=.004; Baseline: β=−.38, p=.03), whereas somatic arousal and insomnia diagnosis were non-significant (Table 3). The association between sleep efficiency and depression was curious. In the multivariate linear regression model, higher depressive symptoms were associated with *greater* sleep efficiency on the baseline night. We then ran *posthoc* bivariate correlations for depressive symptoms and sleep efficiency to examine their relationship when not accounting for the effects of cognitive arousal. We found that bivariate correlations between depression and sleep efficiency were non-significant on both nights (Adaptation: r=.06, p=.70; Baseline: r=−.07, p=.63). Considering that bivariate associations between sleep efficiency and depression did not even approach significance, coupled with moderate collinearity among our predictors (insomnia, depression, rumination), the positive association between depression and sleep efficiency was likely a type III error.

Total sleep time.—Because time-in-bed was fixed to 8 hours for all participants, the pattern of results here are the same as the above models predicting sleep efficiency (Table 3). Higher levels of nocturnal cognitive arousal were associated with shorter total sleep time on both nights (Adaptation: β=−.52, p=.004; Baseline: β=−.36, p=.03). We conducted two

logistic regressions to examine whether greater nocturnal cognitive arousal was associated with short sleep, which was operationalized as < 6 hrs of total sleep time.[34] Higher nocturnal cognitive arousal was associated with increased odds of short sleep on both nights (Adaptation: b=. 13, OR=1.14 [95% CI=1.01, 1.29], p=03; Baseline: b=20, OR=1.23 [95% CI=1.03, 1.46], p=02).

***Posthoc* group comparisons on PSG parameters: High vs low cognitive arousal.—**As multivariate regression models identified nocturnal cognitive arousal as the only robust predictor of objective wakefulness at night, we ran a series of *posthoc* independent samples t-tests to describe group differences related to cognitive arousal on objective disturbance for the baseline night (Table 4). Participants with high nocturnal cognitive arousal took 37 minutes longer to fall asleep and 45 minutes longer to reach persistent sleep than those with low cognitive arousal. Similarly, high cognitive arousal participants spent 44 more minutes awake after initial sleep onset than those with low cognitive arousal. In accordance with these differences in sleep latency and wake after sleep onset, we observed large differences in sleep efficiency (86.97% vs 71.06%; Table 4) and total sleep time (6.95 vs 5.70 hrs; Table 4). We also explored differences in sleep stages and nocturnal awakenings between groups, which revealed that participants with cognitive arousal spent a greater proportion of sleep time in NREM1 and less time in REM than those with low cognitive arousal (Supplementary Table 1).

Nocturnal cognitive arousal predicts daytime hyperarousal

Finally, we sought to identify factors associated with MSLT latencies. An independent samples t-test showed that insomnia participants had longer MSLT latencies than healthy sleepers (14.67±3.87 vs 11.66±4.38 minutes, t[50]=2.45, p=.02, Cohen's d=.73). We then regressed MSLT latencies on nocturnal cognitive arousal and nocturnal somatic arousal while controlling for insomnia diagnosis, depressive symptoms, and prior night's PSG-defined total sleep time (to control for differences in prior night's sleep duration). Nocturnal cognitive arousal was significantly associated with daytime sleep latency such that a 1-SD increase in PSAS-C was associated with a 2-minute increase in mean sleep latency on MSLT (b=.40, β=.40, p=.04). No other predictors in the model were significant: insomnia (p=.42), depression (p=.47), nocturnal somatic arousal (p=.25), and prior PSG night's total sleep time (p=.81). A *posthoc* independent samples t-test showed that participants with MSLT latency ≥ 15 minutes (high daytime hyperarousal), relative to those with MSLT latency < 15 minutes (not hyperaroused), reported substantially more cognitive arousal the prior night which amounted to a medium-large effect (t[50]=2.60, p=.01, Cohen's d=.72). Similarly, participants with high cognitive arousal took over 3 minutes longer to fall asleep on the MSLT than those with low cognitive arousal (15.51 vs 12.12 mins; Table 4).

DISCUSSION

Heightened cognitive arousal while trying to fall asleep at night was associated with greater objective sleep disturbance as measured across two nights of PSG in a combined sample of good sleepers and insomnia patients. Nocturnal cognitive arousal was also associated with objective short sleep at night and prolonged daytime sleep latencies on the MSLT. These

findings add to the literature on cognitive arousal and sleep disturbance by showing that ruminative thinking is not only associated with patient perceptions of nocturnal wakefulness,[1,35,62,63] but also with objective indicators of nocturnal wakefulness and hyperarousal.[30,31,42–44] A critical finding from our study is that nocturnal cognitive arousal was more robustly associated with nocturnal wake and indicators of hyperarousal than insomnia diagnosis, depressive symptoms, or even self-reported presleep somatic arousal. As insomnia patients endorse high levels of cognitive arousal, our data suggest that ruminative thinking could potentially be an important active ingredient in insomnia with regard to objective sleep disturbance and 24-hr hyperarousal.

Comparing participants with vs without insomnia disorder on nocturnal cognitive arousal, somatic arousal, and objective insomnia symptoms.

Consistent with prior reports,[29,64,65] participants with insomnia disorder reported substantially more cognitive arousal and somatic arousal while trying to fall asleep, relative to good sleepers. But when comparing good sleepers and insomniacs on nocturnal PSG, insomnia participants did not exhibit substantially poorer objective sleep relative to healthy sleepers. On both the adaptation and baseline nights, no group differences were observed for objective sleep, although differences approached significance in the expected direction on the baseline night. Even so, these results are consistent with the common finding of discrepancy between subjective reports and objective indicators of sleep in insomnia disorder.[34,35,66] Insomnia patients with objectively measured *normal* sleep duration tend to underestimate sleep duration.[35] Thus, as insomnia populations include those with and without objective sleep disturbance, it is unsurprising that insomnia diagnosis alone did not identify those with objective sleep disturbance per PSG.

Nocturnal cognitive arousal and objective sleep disturbance

Unlike insomnia diagnosis, nocturnal cognitive arousal was robustly associated with prolonged sleep latency, low sleep efficiency, and short sleep. These findings are consistent with prior reports on cognitive arousal and objective sleep,[30,31,42,44] but also adds to the literature by showing that (1) these results replicate across multiple nights and are thus unlikely to be influenced by daily variations in stress or nightly variations in objective sleep[46] and (2) objective sleep disturbance is associated with naturally occurring state-level rumination in the presleep period, which is unconfounded by induced and potential mood-altering effects of prior laboratory studies that have utilized negatively-valenced rumination manipulations.

Important to highlight here is that cognitive arousal at night was most robustly associated with prolonged sleep latency on PSG, which is a well-documented finding in the literature from studies using other methodologies including self-reported and actigraphy-defined sleep.[17,29–31] On the baseline night in our study, participants with high cognitive arousal took 37 minutes longer to fall asleep and 45 minutes longer to reach persistent sleep relative to those with low cognitive arousal. However, whether presleep cognitive arousal is directly linked to wake after sleep onset is less clear. In multivariate models, cognitive arousal was significantly associated with wake after sleep onset on the adaptation night, but not the baseline night. But a simple group comparison showed that participants with high cognitive

arousal had 44 more minutes of wake after sleep onset than those with low cognitive arousal. It is possible that cognitive arousal while trying to fall asleep is associated with wake after sleep onset only to the extent to which presleep ruminative thoughts reappear during wake bouts in the middle of the night and early morning. Research is needed to investigate cognitive processes during bouts of sleep maintenance insomnia.

When considering our findings within the extant literature, two potential and non-mutually exclusive phenomena may be at play. On one hand, prior experimental laboratory studies inducing rumination/cognitive arousal suggest that rumination can cause objective sleep disturbance.[30,31,41] This suggests that ruminating in bed at night can cause and/or exacerbate objectively measured nocturnal wakefulness, especially early in the sleep period. In other words, *faulty cognitive processing* prevents and interferes with physiological sleep processes, rather than merely skewing perceptions of sleep. What is unclear from our data, however, is whether objective wakefulness at night is related to cognitive hyperarousal, cognitive de-arousal, or both. Cognitive hyperarousal refers to aberrant excitation of cognitive and emotional systems, whereas cognitive de-arousal refers to inhibited deactivation of normal cognitive processes at bedtime that naturally de-arouse an awake mind even in the absence of *hyper*arousal.[9] Cognitive arousal may exist on a continuum of de-arousal to hyperarousal, but ultimately the inability to regulate and de-arouse cognitive-emotional systems in the presleep period may inhibit the expression of normal sleep, thereby manifesting as insomnia.[9]

Consideration for a cognitive catalyst model of sleep disturbance and insomnia

Cognitive arousal as a form of faulty cognitive processing may only reflect part of the cognitive influence on sleep disturbance. Poor sleepers often harbor unrealistic and harmful beliefs about sleep (e.g., catastrophizing fallout of a poor night's sleep), which reflect selective attentional bias and negative *cognitive content*.[67–69] Research on cognitive arousal in sleep disturbance and insomnia suggests that ruminating on negatively-valenced sleep-related content is especially disruptive to sleep as indicated by self-report and PSG. [45,70] Relevant here is the cognitive catalyst model of depression. [71–73] This empirically supported model indicates that faulty cognitive processing (cognitive arousal) intensifies the impact of existing negative cognitive content (e.g., dysfunctional beliefs) on mood to fuel depression. In other words, cognitive arousal and negative cognitive content have a synergistic effect on mood, which is consistent with insomnia studies by Spiegelhalder[45] and Carney[70] showing that ruminating on sleep-related content is especially germane to insomnia.

Importantly, the relationship between nocturnal cognitive arousal and nocturnal wakefulness is not likely unidirectional. The inability to fall asleep and/or stay asleep may trigger cognitive arousal in two critical ways: Being awake at night is more conducive to rumination (i.e., faulty cognitive processing) than other times of the day,[27,28] and the inability to sleep can trigger selective attention toward sleep difficulties (i.e., negative cognitive content) that provide content for rumination.[74,75] To our knowledge, however, we know of no experimental studies inducing wakefulness (with neutral valence) to test effects on subsequent nocturnal rumination. Research is needed to determine whether unintended wakefulness at night can trigger ruminative processes. It is possible that sleep disturbance

and rumination can create a toxic positive feedback loop that destabilizes psychoneurobiological systems regulating sleep and stress, thus leading to insomnia and other common comorbidities like depression, particularly when experienced nightly for many weeks or months or even longer.

Potential implications and future directions for insomnia etiology research

Our results and findings in the literature on cognitive arousal and sleep suggest that severe insomnia phenotypes marked by objective sleep disturbance could, in part, have psychological roots in cognitive-emotional dysregulation. Indeed, insomnia with objective short sleep has been most closely linked to physiologic hyperarousal (e.g., HPA axis), but not to ruminative thinking.[34] Yet, our results here suggest that cognitive-emotional hyperarousal or de-arousal is robustly related to objective short sleep, which we replicated across two nights. Further, cognitive arousal before sleep at night also predicted objective indicators of hyperarousal during the day. Despite most participants with high cognitive arousal sleeping < 6 hrs on the baseline night (vs 7 hrs for the low cognitive arousal group), these participants still had longer latencies on the MSLT relative to the low cognitive arousal group. Our data as well as evidence from experimental studies suggest that rumination may contribute to 24-hr physiologic hyperarousal.[31,43] This evidence supports further investigation into the potential role of rumination in insomnia-related physiologic mechanisms (e.g., elevated cerebral glucose metabolism, cortical and subcortical activity during sleep) and medical sequelae (e.g., cardiometabolic disorders) that have been historically considered more closely associated with physiologic hyperarousal, per se', rather than cognitive-emotional hyperarousal.

Notably, cognitive-behavioral therapy for insomnia (CBTI), which is first-line treatment for insomnia disorder,[76] has limited efficacy for insomnia cases with objective sleep disturbance. [38,77] Unfortunately, despite devoting 1–2 sessions to cognitions, CBTI also has limited effects on cognitive arousal and ruminative thinking.[78] If severe cognitive-emotional dysregulation contributes to objective sleep disturbance, then augmenting CBTI with interventions that better reduce rumination may be more effective in this patient population. Indeed, treatments improving cognitive de-arousal may enhance insomnia treatment outcomes,[9] and evidence suggests that mindfulness-based augmentations to CBTI have potential in this area.[5,60,79]

Limitations

Our study findings should be interpreted in the context of methodological limitations. Reports of nocturnal cognitive and somatic arousal were retrospective such that participants reported these experiences upon waking in the morning. Thus, we cannot determine directionality between cognitive arousal and nocturnal wakefulness in this study, especially within the context that rumination was most strongly related to difficulty falling asleep. Rather, interpretations regarding directionality described above are based on prior laboratory studies that have induced various forms of cognitive arousal like rumination and worry, which produced objective sleep disturbances. Although inducing stress and state rumination prolongs objective sleep latency,[31] it is possible that difficulty falling asleep leads to rumination as the inability to sleep can trigger negative schema that provide content for

rumination.[74,75] Future experimental studies are needed to clarify causal associations between nocturnal cognitive arousal and nocturnal wakefulness. In addition, sleeping in a novel environment may have triggered cognitive arousal when trying to fall asleep for some participants. Although we do not anticipate that any such potential effects would change the nature of the relationship between nocturnal cognitive arousal and objective sleep, we cannot rule it out.

Our nocturnal cognitive arousal measure also represents a methodological limitation. Our data suggested that nocturnal cognitive arousal was most closely associated with sleep latency, but not consistently with wakefulness in the middle of the night. Specifically, nocturnal cognitive arousal was only associated with wakefulness in the middle of the night on the adaptation night. However, our nocturnal cognitive arousal measure specifically assesses cognitive activity in the presleep period (but *not* cognitions in the middle of the night). Our assessment window of the presleep period may have favored identification of associations between cognitive arousal and presleep wakefulness over middle of the night wakefulness. However, it is also important to highlight that individuals tend to ruminate most in the late evening and night, particularly when trying to fall asleep.[27,28] Even so, it is likely the case that presleep cognitive activity is associated with middle of the night wakefulness to the extent that presleep cognitions corresponds to cognitions that reappear during wake bouts in the middle of the night. Evidence suggests that presleep cognitive content extends into the sleep period and that individuals recall these cognitions when woken up during dreaming.[80] Thus, if the sleeping mind continues processing presleep ruminative content, then it is likely that rumination during wake bouts in the middle of the night share content with presleep cognitions. Future research in this area should account for rumination that occurs during wake bouts in the middle of the night and investigate how presleep cognitions potentially affect sleep continuity.

Our sample was largely comprised of young US adults identifying as non-Hispanic white and non-Hispanic black, which may limit generalizability to individuals outside of these sociodemographic groups. Along these lines, this was a single site study in Detroit, Michigan, thus our results may not generalize to populations with different sociodemographic and cultural compositions. Further, as the sample was comprised of good sleepers and insomnia participants, research is needed to replicate these findings in a sample of individuals with DSM-5 insomnia disorder. Lastly, the present study involved multiple statistical analyses. Despite the heightened risk for type I errors for multiple analyses, it is important to emphasize that our findings are consistent with findings of studies examining rumination and sleep disturbance using different methodologies. Further, replication in our own study across nights likely protects against erroneous findings.

Conclusions

Reports of cognitive hyperarousal in bed at night were consistently associated with objective sleep disturbance across multiple nights in a combined sample of individuals with and without insomnia. Among all objective PSG parameters, nocturnal cognitive arousal was most strongly related to difficulty falling asleep. Importantly, nocturnal cognitive arousal was more robustly linked to objective disturbed sleep than was clinical insomnia diagnosis,

depressive symptoms, or self-reported nocturnal somatic arousal. Further, nocturnal cognitive arousal was linked to nocturnal and daytime indices of physiologic arousal, which suggests that ruminating at night extends emotion dysregulation into the sleep period and beyond after waking the next day. Taken together, these results emphasize the importance of cognitive arousal to the disruption of nightly sleep and its close association with physiologic hyperarousal. Future prospective research is needed to better characterize potential etiological and perpetuating influences of rumination in the development of insomnia disorder with objective sleep disturbance and in the destabilization of stress-regulation systems that regulate sleep.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Highlights

- Good sleepers and insomniacs do not differ on objective sleep disturbance
- Individuals with insomnia have higher cognitive arousal than good sleepers
- Nocturnal cognitive arousal is associated with objective sleep disturbance
- Participants with high cognitive arousal take an hour to fall asleep
- Participants with high cognitive arousal sleep less than 6 hours

Table 1.

Sample demographic information, self-reported sleep parameters, and depressive symptoms.

	Full sample n=52	**No Insomnia n=34/52; 65.4%**	**Insomnia Disorder n=18/52; 34.6%**	
Gender, female	n=27; 51.9%	n=17/34; 50.0%	n=10; 55.6%	χ^2=.15, p=.70
Age	30.2±9.9, 18-51	28.29±8.8, 19-48	33.8±11.2, 18-51	t(50)=1.96, p=.06
BMI	24.7±3.9	24.7±3.7	24.6±4.4	t(50=−.16, p=.88
Race				
White (non-Hispanic)	n=32; 61.5%	n=20; 58.8%	n=12; 66.7%	
Black (non-Hispanic)	n=12; 23.1%	n=7; 20.6%	n=5; 27.8%	
Hispanic	n=2; 3.8%	n=2; 5.9%	n=0; 0.0%	
Asian	n=4; 7.7%	n=4; 11.8%	n-0; 0.0%	
Other	n=2; 3.8%	n=1; 2.9%	n=1; 5.6%	
Marital Status				
Single	n=33; 63.5%	n=23; 67.6%	n=10; 55.6%	
Married	n=18; 34.6%	n=11; 32.4%	n=7; 38.9%	
Divorced	n=1; 1.9%	n=0; 0.0%	n=1; 5.6%	
Self-Reported Sleep				
Bedtime	23:11 (±49 mins)	23:06 (±49 mins)	23:19 (±49 mins)	t(50)=.17, p=.87
Wake-time	07:38 (±56 mins)	07:43 (±57 mins)	07:31 (±55 mins)	t(50)=.57, p=.57
Sleep latency	30.62±29.30 mins	15.88±15.70 mins	58.44±29.01 mins	t(50)=6.89***, d=1.82
Nighttime awakenings	.97±1.33	.25±.74	2.33±1.12	t(50)=8.03***, d=2.19
Wake after sleep onset	17.10±31.14 mins	.41±1.48 mins	48.61±36.01 mins	t(50)=7.86***, d=1.88
Total sleep time	6.92±1.35 hrs	7.65±.57 hrs	5.56±1.34 hrs	t(50)=−7.90***, d=2.03
PSQI	6.13±4.73; 40.4%	3.41±2.16; 11.8%	11.28±3.89; 94.4%	t(50)=9.40***, d=2.50
ISI	8.26±7.56; 40.0%	3.44±3.69; 6.2%	16.83±4.30; 100.0%	t(50)=11.61***, d=3.34
PHQ-9	4.98±5.28; 11.5%	2.53±2.93; 0.0%	9.61±5.67; 33.3%	t(50)=5.97***, d=1.57

Note: No Insomnia = negative for DSM-IV insomnia disorder. Insomnia Disorder = Diagnosed with acute or chronic insomnia disorder per DSM-IV criteria. BMI = body mass index (kg/m^2). PSQI = Pittsburgh sleep quality index. ISI = insomnia severity index. PHQ-9 = patient health questionnaire – 9. χ^2 = chi-square analysis to compare proportions between participants with vs without insomnia. t = t-statistic for comparison of means between participants with vs without insomnia. p = significance value.

*** p<.001.

d = Cohen's d effect size.

Table 2.

PSG sleep parameters, nocturnal cognitive arousal and somatic arousal on the adaptation nights and baseline night for full sample and by insomnia status.

	Full sample n=52	**No Insomnia n=34/52; 65.4%**	**Insomnia Disorder n=18/52; 34.6%**	
Adaptation night				
Nocturnal cognitive arousal	14.44±5.76	12.56±3.79	18.00±7.15	t(50)=3.60 **, d=.95
Nocturnal somatic arousal	12.75±11.50	11.94±2.95	14.28±4.27	t(5)=2.32 *, d=.64
Sleep latency	22.70±18.13 mins	21.68±17.48 mins	24.64±19.66 mins	t(50)=.56, p=.58
Latency to persistent sleep	32.89±24.97 mins	33.25±26.12 mins	32.19±23.37 mins	t(50)=−.14, p=.89
Wake after sleep onset	61.09±54.94 mins	56.75±58.47 mins	69.28±48.07 mins	t(50)=.78,p=.44
Sleep efficiency	82.40±13.36	83.59±14.04	80.15±12.03	t(50)=−.88, p=.38
Total sleep time	6.59±1.07 hrs	6.71±1.12 hrs	6.41±0.96 hrs	t(50)=−.85, p=.40
Baseline night				
Nocturnal cognitive arousal	13.37±54.40	11.85±3.53	16.22±4.55	t(50)=3.84 ***, d=1.07
Nocturnal somatic arousal	12.75±11.50	10.74±2.76	13.67±3.45	t(50)=3.34 **, d=.94
Sleep latency	27.12±28.91 mins	22.22±27.15 mins	36.36±30.63 mins	t(50)=1.71, p=.09
Latency to persistent sleep	34.98±31.19 mins	31.03±31.31 mins	42.44±30.41 mins	t(50)=1.26, p=.21
Wake after sleep onset	44.53±53.46 mins	34.71±45.86 mins	63.06±62.73 mins	t(50)=1.86, p=.07
Sleep efficiency	84.21±14.58	86.92±13.76	79.11±15.09	t(50)=−1.88, p=.07
Total sleep time	6.73±1.16 hrs	6.96±1.10 hrs	6.30±1.18 hrs	t(50)=−2.00, p=.05

Note: No Insomnia = did not endorse DSM-IV insomnia disorder. Insomnia disorder = endorsed DSM-IV insomnia disorder. Nocturnal cognitive arousal measured by the Pre-Sleep Arousal Scale, Cognitive factor. Nocturnal somatic arousal measured by the Pre-Sleep Arousal Scale, Somatic factor. Sleep latency, latency to persistent sleep, wake after sleep onset, sleep efficiency, and total sleep time assessed via overnight polysomnography. t = t-statistic for comparison of means between participants with vs without insomnia. p = significance value.

* p<.05

** p<.01

*** p<.001.

d = Cohen's d effect size.

Table 3.

PSG Adaptation Night: Regressing PSG-based insomnia symptoms on insomnia diagnosis and presleep cognitive and somatic arousal.

	Adaptation Night				Baseline Night			
Outcome	**Predictors**	**b**	**β**	**p**	**Predictors**	**b**	**β**	**p**
Sleep Latency					Sleep Latency			
Adaptation: F=. 18, p=84	Insomnia diagnosis	−3.00	−.08	.68	Insomnia diagnosis	9.80	.16	.33
Baseline: F=1.99, p=.11	Depressive symptoms	−.20	−.06	.77	Depressive symptoms	−1.84	−.34	.06
	Nocturnal cognitive arousal	1.15	.37	.04	Nocturnal cognitive arousal	3.44	.52	.003
	Nocturnal somatic arousal	.47	.09	.61	Nocturnal somatic arousal	.78	.09	.61
Latency to persistent sleep					Sleep Latency, Persistent			
Adaptation: F=.32, p=.73	Insomnia diagnosis	−15.63	−.30	.11	Insomnia diagnosis	4.54	.07	.67
Baseline: F=2.98, p=.03	Depressive symptoms	.45	.10	.61	Depressive symptoms	−2.09	−.35	.05
	Nocturnal cognitive arousal	2.22	.51	.004	Nocturnal cognitive arousal	3.60	.51	.003
	Nocturnal somatic arousal	−.31	−.05	.80	Nocturnal somatic arousal	2.03	.21	.22
Wake after sleep onset					Wake after sleep onset			
Adaptation: F=1.55, p=.22	Insomnia diagnosis	12.98	.11	.54	Insomnia diagnosis	22.70	.20	.25
Baseline: F=2.89, p=.03	Depressive symptoms	−2.06	−.20	.30	Depressive symptoms	−2.98	−.29	.12
	Nocturnal cognitive arousal	4.61	.48	.007	Nocturnal cognitive arousal	2.82	.23	.19
	Nocturnal somatic arousal	−.4.67	−.31	.09	Nocturnal somatic arousal	4.93	.30	.11
Sleep efficiency					Sleep efficiency			
Adaptation: F=1.20, p=.31	Insomnia diagnosis	−2.28	−.08	.65	Insomnia diagnosis	−6.48	−.21	.20
Baseline: F=2.98, p=.03	Depressive symptoms	.47	.18	.33	Depressive symptoms	1.09	.39	.03
	Nocturnal cognitive arousal	−1.20	−.52	.004	Nocturnal cognitive arousal	−1.26	−.38	.03
	Nocturnal somatic arousal	.88	.24	.18	Nocturnal somatic arousal	−1.19	−.27	.13
Total sleep time					Total sleep time			
Adaptation: F=1.15, p=.33	Insomnia diagnosis	−10.17	−.08	.68	Insomnia diagnosis	−34.89	−.24	.16
Baseline: F=2.92, p=.03	Depressive symptoms	2.14	.18	.36	Depressive symptoms	5.07	.38	.04
	Nocturnal cognitive arousal	−5.72	−.52	.004	Nocturnal cognitive arousal	−5.74	−.36	.03
	Nocturnal somatic arousal	4.37	.25	.17	Nocturnal somatic arousal	−5.46	−.26	−.15

Note: Sleep latency, Sleep latency (persistent), Wake after sleep onset, Sleep efficiency (Total Sleep Time/Time in Bed), and Total Sleep Time were assessed via polysomnography. Insomnia diagnosis based on DSM-IV criteria per clinical interview. F = F-ratio to determining model significance. b = unstandardized beta coefficient. β = standardized beta coefficient. p = significance value for model or parameter estimate.

Table 4.

Group differences between subjects high and low on nocturnal cognitive arousal on objective sleep disturbances on the baseline night.

	Low Cognitive Arousal n=43/52	**High Cognitive Arousal n=9/52**	
Sleep latency	20.74±19.89	57.56±44.64	t(50)=3.94***, d=1.07
Latency to persistent sleep	27.14±21.11	72.44±44.17	t(50)=4.72***, d=1.31
Wake after sleep onset	36.83±43.91	81.28±79.10	t(50)=2.37**, d=.69
Sleep efficiency	86.97±12.38	71.06±17.74	t(50)=−3.24**, d=1.04
Total sleep time	6.95±.98 hrs	5.70±1.44 hrs	t(50)=−3.17**, d=1.01
MSLT latency	12.12±4.28 mins	15.51±4.18 mins	t(50)=2.18*, d=.80

Note: Cognitive arousal was assessed via the Presleep Arousal Scale – Cognitive factor (PSAS-C). Low Cognitive Arousal was defined as baseline PSAS-C scores ≤ 16. High Cognitive Arousal was defined as baseline PSAS-C scores > 16. Sleep latency, Sleep latency (persistent), Wake after sleep onset, Sleep efficiency (Total Sleep Time/Time in Bed), and Total Sleep Time were assessed via polysomnography. Means and Standard Deviations are reported for objective findings. t = t-statistics for independent samples t-tests.

* p<.05.

** p<.01.

*** p<.001.

p = significance value for non-significant results. d = Cohen's d effect size for independent samples t-tests.