Emotion reactivity in chronic primary insomnia: Neural correlates and relationship with responses to cognitive-behavioural therapy for insomnia

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ABSTRACT

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Kirsten Ka Mon Gong

Individuals with chronic insomnia have trouble falling or staying asleep, wake up earlier than desired, and have daytime complaints. Some individuals also have difficulty regulating their emotions, especially in response to sleep-related stimuli. Cognitive-behavioural therapy for insomnia (CBTi), the gold-standard treatment for insomnia, has been shown to improve sleep. But, it is still unclear if CBTi impacts the neural activations underlying emotional reactivity in adults with insomnia and if emotion reactivity plays a role in CBTi treatment response. Thus, the first aim of this study was to investigate brain activity, using functional magnetic resonance imaging, during an emotion reactivity task in adults with insomnia and healthy sleepers. Secondly, we examined the effects of CBTi on brain activity during this emotion task in adults with insomnia. Lastly, we determined if brain activity at baseline was predictive of CBTi treatment response. While no differences in brain activity in response to sleep relative to neutral images were found between adults with insomnia and healthy sleepers at baseline, different patterns of activation in response to negative relative to neutral images were observed. Furthermore, after CBTi, adults with insomnia had reduced activity within the left insula and superior temporal gyrus in response to sleep relative to neutral images. But, there were no changes in affect. Lastly, brain activity in response to sleep relative to neutral images at baseline did not predict improvements in sleep. Overall, these findings contribute to the growing literature about sleep and emotions in adults with insomnia.

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Contribution of Authors

Kirsten Ka Mon Gong did the literature review, performed statistical analyses in R, created tables and figures in R, wrote the drafts of the manuscript, created the survey on Amazon MTurk, trained participants on the emotion reactivity task, helped acquire the MRI data, checked the quality as well as preprocessed the MRI data, wrote second-level analysis scripts, and cleaned all measures acquired from sleep diaries and questionnaires. Dr. Thien Thanh Dang-Vu and Dr. Florence Pomares assisted with revisions of the thesis by providing feedback as well as editing or proofreading the manuscript. Dr. Florence Pomares and Dr. Aurore Perrault created the emotion reactivity task on PsychoPy, wrote scripts on MATLAB to preprocess and analyze the MRI data, and contributed to data collection. Lukia Tarelli was involved in coordinating the data collection and screening participants.

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Introduction

Adequate amount of sleep and good sleep quality are crucial for numerous reasons, including maintenance of cognitive and emotional health. Emotions are important because they impact our perception, attention, learning, memory, reasoning, and problem-solving (Tyng et al., 2017). Emotions include different components, such as subjective experiences as well as behavioural and physiological responses (American Psychological Association, 2007). Subjective experiences can be evaluated through self-reported feelings and behavioral responses can be evaluated with facial expressions, body movements, or posture (Dael et al., 2012, as cited in Tyng et al., 2017; Jack & Schyns, 2015, as cited in Tyng et al., 2017). Physiological responses, such as electrical or hemodynamic responses of the central nervous system, can be measured using neuroimaging techniques (Vytal & Hamann, 2010) among other techniques. For example, responses of the autonomic nervous system (e.g., heart rate) can be measured with biosensors (Li & Chen, 2006, as cited in Tyng et al., 2017). As it is more challenging for individuals to consciously hide or alter their physiological responses, these responses may be more representative of an individual's emotional state compared to the subjective or behavioural responses (Tyng et al., 2017).

Insomnia and Emotion

Although sleep deprivation can temporarily affect one's cognitive functioning or mood, this can become a bigger issue when sleep problems are prolonged (Toschi et al., 2021). Therefore, sleep disorders, such as insomnia disorder, may be a model to examine emotion regulation difficulties. Chronic primary insomnia is a sleep disorder that is characterized by difficulty falling

asleep, difficulty staying asleep, and/or early awakenings with daytime complaints that occur at least three times per week for a minimum duration of three months. Some of the daytime complaints that are associated with nighttime sleep are mood disturbances and irritability (American Academy of Sleep Medicine [AASM], 2014). These characteristics are also not caused by substances nor medical or psychiatric conditions (Riemann et al., 2010).

Mood disturbances as well as emotion regulation difficulties are prevalent in individuals with insomnia. For instance, Buysse and colleagues (2007) found that individuals with primary insomnia reported having more negative mood in comparison to good sleepers. Adults with insomnia also had more emotion regulation difficulties than sex- and age-matched good sleepers (Galbiati et al., 2020). Although adults with insomnia did not have difficulty recognizing their emotions, their confidence in emotion regulation skills was remarkedly lower compared to good sleepers. For instance, patients with insomnia who had poorer subjective sleep quality (as measured using the Pittsburgh Sleep Quality Index (PSQI), which is a self-reported measure to assess subjective sleep parameters) tended to have more negative affect (assessed using the Positive Affect and Negative Affect Schedule (PANAS), which is a self-reported measure to examine positive and negative emotions; Dong et al., 2017; Watson et al., 1988). Similarly, patients with insomnia who had greater self-reported insomnia severity also tended to have more self-reported emotion dysregulation (Palagini et al., 2017). Additionally, emotion dysregulation was a mediator in the relation between low resilience, a predisposing factor of psychopathology, and cognitive hyperarousal prior to sleep, a perpetuating factor of insomnia, in individuals with insomnia (Palagini et al., 2018). This provides additional evidence that there is a relation between emotion regulation difficulties and sleep disturbances in patients with insomnia.

Although there is a link between emotion dysregulation and sleep disturbances, researchers have also studied the direction of this relation. Indeed, several studies provided support that the relation between emotions and sleep is bidirectional (Altena et al., 2016; Kahn et al., 2013; Talbot et al., 2012; Zakiei et al., 2020). But, there are few studies that have shown an unidirectional effect or a bidirectional effect whereby one direction was stronger than the other. For instance, in a study with depressed and healthy participants, better sleep quality was predictive of better positive affect the following day. Poorer sleep quality was also predictive of greater negative affect the following day. However, fluctuations in positive or negative affect did not predict fluctuations in sleep quality the following day (Bouwmans et al., 2017). Narmandakh et al. (2021) showed that the effect of daytime affect on sleep the following night was not as strong as the effect of sleep on affect the following day. In a longitudinal study, Jansson-Fröjmark and colleagues (2016) found that greater emotion dysregulation over time increased one's risk of experiencing insomnia in the future. Yet, there is evidence that chronic insomnia more often precedes mood disorders or co-occurs rather than appears following the onset of a mood disorder (Johnson et al., 2006; Ohayon & Roth, 2003).

Models of Insomnia

A prominent model of insomnia, which was developed by Spielman and colleagues (1987), is the 3P model of insomnia. They proposed that three factors contribute to the onset and maintenance of insomnia. Firstly, there are predisposing factors (e.g., genetic predisposition), which increase one's risk for experiencing sleep disturbances. Then, there are precipitating factors (e.g., acute, stressful events or situations, such as losing a job or a loved one), which lead to the onset of insomnia. Lastly, there are perpetuating factors (e.g., unhealthy sleep habits, such as longer time spent in bed, irregular sleep-wake schedules, or frequent napping, or even excessive

worrying about daytime impairments), which are involved in maintaining this disorder over an extended period of time even after the stressful event or situation dissipates (Spielman et al., 1987; Wright et al., 2019).

There are other models that exist to explain how insomnia is developed or maintained and how emotions may specifically act as a precipitating or perpetuating factor. For instance, Kales and colleagues (1976) proposed the internalization of conflicts model of insomnia to explain the role that emotions can have on the onset and maintenance of insomnia. In this model, it is thought that individuals with insomnia develop this fear of sleeplessness over time due to both enhanced emotional and physiological arousal. This fear can give rise to heightened emotional arousal, which then leads to difficulty falling or staying asleep. Thus, emotional arousal appears to play a role in the onset and maintenance of insomnia disorder. Moreover, Harvey (2002) proposed the cognitive model of insomnia to explain how primary insomnia was maintained. Throughout the day or in bed, individuals may engage in excessive worrying or rumination about sleep and the effects that it can have on their health or level of daytime functioning. These negative thoughts can generate feelings of anxiety or emotional distress. Those feelings can promote selective attention to sleep-related threat cues. Individuals can also automatically monitor their body sensations or the environment for sleep-related threats. If they identify a sleep-related threat, this can lead to excessive worrying or rumination once again. Although emotional arousal may play a role in the onset and maintenance of insomnia, cognitive processes should also be taken into consideration to better understand patients' sensitivity toward sleep-related stimuli.

Self-Reported Ratings of Stimuli

Individuals with insomnia tend to have greater arousal and may be more sensitive to negative sleep-related images compared to good sleepers. Baglioni and colleagues (2010a) looked

at how individuals with primary insomnia and good sleepers rated images that were sleep-related or unrelated to sleep. In terms of valence ratings, all participants provided different valence ratings for the neutral images than the negative images. Furthermore, individuals with insomnia subjectively reported that negative sleep-related images were just as unpleasant as negative images that were unrelated to sleep. However, individuals with normal sleep subjectively reported that the negative images that were unrelated to sleep were more unpleasant than the negative sleep-related images. With respect to arousal, all participants provided different arousal ratings for the neutral images than the negative images. Nevertheless, images were overall rated as more arousing in individuals with insomnia compared to good sleepers. Additionally, individuals with insomnia provided similar arousal ratings for negative images that were not sleep-related and negative images that were sleep-related. On the contrary, good sleepers found the negative images that were not sleep-related to be more arousing than the negative images that were sleep-related. Although subjective experiences can be used to evaluate how individuals with insomnia respond to emotional stimuli, hemodynamic responses associated with the neural circuitry underlying emotions can also be examined objectively.

Emotional Brain Network

There are two neural systems that are associated with recognizing the emotional significance of a stimulus, producing an emotional state, and regulating the emotional state. The ventral system is important for recognizing the emotional significance of a stimulus, producing an emotional state in response to the stimulus, and regulating autonomic responses to emotional stimuli. It is comprised of the amygdala, insula, ventral striatum, ventral part of the anterior cingulate cortex, and ventral portion of the prefrontal cortex. In particular, the amygdala is involved in identifying the emotional significance of a stimulus as well as in producing emotional

states and behaviours (Phillips et al., 2003). David and Whalen (2001, as cited in Phillips et al., 2003) also stated that the amygdala is involved in modulating attention to emotionally salient stimuli. Based on functional neuroimaging studies, the insula is activated while individuals anticipate aversive stimuli (Phelps et al., 2001). Its activity has also been linked to emotional states while individuals encounter emotional stimuli (Casey et al., 1996, as cited in Altena et al., 2016; Charney & Drevets, 2002, as cited in Altena et al., 2016). The ventral striatum has been involved in craving (Breiter et al., 1996), reward processing (Pagnoni et al., 2002), and reward anticipation (Knutson et al., 2001). Based on functional neuroimaging studies, the ventral portion of the anterior cingulate cortex has been suggested to be activated during positive and negative mood induction (Mayberg et al., 1999; Shin et al., 2000) or in the presence of rewards (Elliott et al., 2000). Ventral portions of the prefrontal cortex, such as the ventrolateral prefrontal cortex, have been activated during tasks that involve negative mood induction (Shin et al., 2000), emotional stimuli (Reiman et al., 1997), or negative facial expressions (Sprengelmeyer et al., 1996).

The dorsal system is critical for regulating emotional states or behaviours. This system includes the hippocampus, dorsal part of the anterior cingulate cortex, and the dorsal portion of the prefrontal cortex (Phillips et al., 2003). The hippocampus is involved in the promotion as well as prevention of defensive behaviours or anxiety in contexts that may be considered threatening (Gray & McNaughton, 2000). The dorsal regions of the anterior cingulate cortex are involved in attention to emotional states (Lane et al., 1998) as well as effortful control of arousal that is related to emotional states (Raichle et al., 1994). The dorsal regions of the prefrontal cortex are activated during tasks that involve directing attention away from emotional aspects of stimuli or toward the context that the stimuli are presented in (Tucker et al., 1995).

Brain Responses to Emotional Stimuli

Based on functional neuroimaging studies, individuals with insomnia seem to have elevated emotion reactivity for images that were negative and especially those that are insomnia-related compared to good sleepers. Although difficulties in processing emotional information, as reflected by elevated amygdala activity in response to emotional stimuli, have been implicated in social anxiety (Hattingh et al., 2013), posttraumatic stress (El Khoury-Malhame et al., 2011), and borderline personality disorders (Hazlett et al., 2012), the study by Baglioni and colleagues (2014) was the first one that used functional magnetic resonance imaging (fMRI) to investigate whether amygdala activity in response to visual stimuli that were either related or unrelated to insomnia differed between participants with insomnia and good sleepers. In terms of arousal, individuals with insomnia and good sleepers had greater amygdala activity while viewing negative images with high arousal levels than negative images with moderate arousal levels. In terms of valence, individuals with insomnia had similar amygdala activity while viewing negative images and neutral images with the same arousal levels. However, good sleepers had greater amygdala activity while viewing negative images than neutral images with the same arousal levels. In terms of sleep content, individuals with insomnia had greater amygdala activity while viewing negative images related to insomnia compared to negative images that were not sleep-related. However, good sleepers had more amygdala activity while viewing negative images that were not sleep-related compared to insomnia-related images. Although some individuals with insomnia seem to have this selective attention to sleep-related stimuli, which may be implicated in the maintenance of the sleep disorder (Espie et al., 2006, as cited in Spiegelhalder et al., 2018), there was no difference in brain activity in response to sleep-related words between adults with primary insomnia and good sleepers (Spiegelhalder et al., 2018). Therefore, there is greater attentional bias as well as enhanced

brain activity in response to negatively valenced sleep images rather than sleep-related words in adults with primary insomnia compared to good sleepers. Nevertheless, different techniques can be implemented to help these individuals regulate their emotions.

Emotion Regulation in Insomnia

Emotion regulation is the ability to modify the valence, intensity, and duration of one's emotion (Gross, 1998, as cited in Gross, 2014). Emotion regulation is essential because it enables individuals to acquire a desired emotional state to think or behave in a beneficial way (Tamir, 2016). Adaptive emotion regulation strategies consist of cognitive reappraisal, which involves changing how one views a situation in order to modify their emotions (Samson & Gross, 2012, as cited in Gross, 2014). The use of cognitive reappraisal has been linked to positive outcomes, such as the tendency to have and to express more positive emotions as well as to have better well-being (Gross & John, 2003). Maladaptive emotion regulation strategies include expressive suppression (Aldao et al., 2010). As expressive suppression does not minimize the experiential response, individuals will still experience the negative emotion. This negative emotion can last for a while or continue to arise if it is not dealt with (Gross & John, 2003). Indeed, negative emotions can play a role in sleep disturbances, especially when they are experienced before bedtime (Cerolini et al., 2015). The use of expressive suppression has been associated with other negative outcomes, such as the tendency to have and to express fewer positive emotions as well as have a decline in well-being (Gross & John, 2003). Furthermore, the inability to properly regulate emotions (i.e., emotion dysregulation) can lead to abnormal emotional reactivity (i.e., low or high emotional reactivity). In other words, it can negatively impact how individuals respond to emotional information (Derryberry & Reed, 2003, as cited in Baglioni et al., 2010b). After encountering a stressful stimulus, emotion dysregulation can make it difficult for cognitive or physiological responses to

return to baseline (Galbiati et al., 2020). In the context of insomnia, this is problematic because these maladaptive strategies have consequences on health and well-being (Carver et al., 1989, as cited in Aldao et al., 2010; Folkman & Lazarus, 1980, as cited in Aldao et al., 2010).

Given that mood disturbances or emotion regulation difficulties associated with sleep are common in patients with insomnia, it is important to identify effective interventions that may not only work to improve their sleep but may also indirectly enhance their mood through improvements in sleep. Although sleep medications can be prescribed to help individuals with their sleep difficulties, they often have side effects, especially when taken long term (van der Zweerde et al., 2019). Therefore, alternative interventions are more favourable and safer for individuals who have chronic primary insomnia (Sharma & Andrade, 2012).

Cognitive-Behavioural Therapy for Insomnia

Given the importance of targeting the cognitive and behavioural components that play a role in the maintenance of insomnia, the first line of the treatment for insomnia is cognitive-behavioural therapy for insomnia (CBTi). CBTi is a psychological intervention that targets maladaptive behaviours and beliefs about sleep that are prevalent in individuals with insomnia. In order to optimize their sleep using behavioural techniques, these individuals learn how to practice sleep restriction. Sleep restriction helps to optimize sleep efficiency, which is computed as the ratio between the total time spent asleep and the total time spent in bed multiplied by 100 and gives some indication as to how well an individual has slept. This particular technique consists of reducing the overall amount of time spent in bed as much as possible to the amount of time spent sleeping by consistently following a fixed sleep schedule. There is also stimulus-control therapy whereby individuals are instructed to select a fixed bed and wake time, go to bed only when they are tired, leave the bed if they cannot sleep, or refrain from taking naps during the day (Anderson,

2017; Morin, 2006; Pigeon, 2010; Shrivastava et al., 2014). To deal with their maladaptive thoughts or beliefs about sleep, they also receive cognitive therapy. For instance, with the help of a trained expert, individuals can properly address and restructure their beliefs. Thus, their excessive worrying about sleep is minimized prior to bedtime. CBTi also consists of information about sleep hygiene through psychoeducation: information about environmental factors that are suitable or harmful for sleep, such as light, noise, temperature, and bedding; about substance use; and about healthy habits that can ameliorate sleep, such as dieting and exercise (Sharma & Andrade, 2012). CBTi also includes muscle relaxation or imagery techniques to ease their body tension and racing thoughts, respectively. CBTi can be administered to one individual at a time or to a group (Anderson, 2017). Approximately 50% of chronic insomniacs are CBTi responders, i.e., they show clinically significant decreases in insomnia symptoms after the intervention, while roughly one-third of insomniacs achieve remission after CBTi (Morin et al., 1999).

Although the purpose of CBTi is to improve sleep in individuals with insomnia, there are components of CBTi that seem to affect emotions or neural circuits underlying emotions. For example, as cognitive therapy can help individuals to manage their excessive worries about sleep prior to bedtime, this method is expected to diminish their thoughts that are filled with negative emotions (e.g., intrusive thoughts about sleep) during bedtime (Baglioni et al., 2010b). CBTi decreases resting-state functional connectivity between the left amygdala and left lingual gyrus (Lee et al., 2018). This suggests that these changes in the neural circuit underlying emotions following CBTi may be linked to heightened arousal of sensory information, as the lingual gyrus is a brain region that has been associated with processing visual information. Based on the cognitive model of insomnia that was proposed by Harvey (2002), Dong and colleagues (2017) suggested that negative affect may be reduced in patients with insomnia if their sleep quality

improves. Thus, CBTi may have additional benefits aside from contributing to sleep improvements in individuals with insomnia.

There is evidence that suggests that, following CBTi, there are changes in brain activity associated with the visualization of sleep-related versus neutral images. In a study by Kim and colleagues (2017), participants with insomnia and healthy sleepers were presented with a total of 28 sleep-related or neutral, non-sleep related images within a magnetic resonance imaging (MRI) scanner. They were then instructed to press a button on the left with their left thumb if a given image was not sleep-related or to press a button on the right with their right thumb if it was sleep-related. Kim et al. (2017) reported that, prior to CBTi, participants with insomnia had enhanced blood oxygen level dependent (BOLD) response in regions, such as the precentral cortex, left prefrontal cortex, and posterior cingulate cortex while identifying that a stimulus was sleep-related compared to good sleepers. After receiving CBTi, the BOLD response in the precentral and prefrontal cortices decreased while selecting that a stimulus was sleep-related (i.e., BOLD response to sleep stimuli minus BOLD response to neutral stimuli) in individuals with insomnia. However, Kim et al. (2017) did not evaluate subjective responses (i.e., valence ratings) to neutral, negative, and sleep-related stimuli in conjunction with fMRI brain responses toward these stimuli, which is one of the main focuses of the current study. Perhaps if insomnia is targeted with CBTi, this will be followed by improvements in emotion reactivity both subjectively and objectively, especially toward sleep-related stimuli.

Predictors of CBTi Treatment Response

CBTi is usually provided over four or more one-hour long sessions that occur weekly or bi-weekly. Thus, some individuals with sleep disturbances view this therapy as more time-consuming and less practical than taking medications to improve sleep. Indeed, the dropout rate

for CBTi was reported to be around 10 to 40% (Ong et al., 2008). In addition to the cost of the intervention, another downside that limits its accessibility is the long wait times before the intervention is delivered by a trained CBTi practitioner (Koffel et al., 2018). As previously stated, even after completing the sessions, around 50% of them will be CBTi responders, while roughly one-third will be in remission (Morin et al., 1999).

As the degree of CBTi treatment response is variable in patients with insomnia and as it is clinically useful to identify individuals who may or may not benefit from CBTi given its time commitment, cost, accessibility, and effectiveness, different predictors of CBTi treatment response have been proposed or identified (Morin et al., 1999). For instance, subtypes of insomnia may be indicative of CBTi treatment response. Blanken and colleagues (2019) used latent class analysis to identify subtypes of insomnia. These subtypes were derived from participants' response patterns on questionnaires about sleep, life history, fatigue and arousal, personality, mood, and happiness. They found that individuals in the moderately distressed but reward sensitive group, meaning that they had intact responses to positive emotions, subjectively reported having less difficulty falling asleep after receiving CBTi compared to a wait-list control group. Those in the moderately distressed but reward sensitive group also had decreased total Insomnia Severity Index (ISI) scores, reflecting subjectively fewer or less severe insomnia symptoms, compared with the wait-list control group. Similarly, the slightly distressed with high reactivity to their environment and life events group had decreased total ISI scores after receiving CBTi compared with the wait-list control group. But, the slightly distressed with high reactivity to their environment and life events group did not subjectively report having less difficulty falling asleep after receiving CBTi compared to the wait-list control group (Blanken et al., 2019). Based on Blanken and colleagues' (2019) study, this may indicate that individuals with insomnia who are highly reactive to their

environment, perhaps even their sleep environment, may still have difficulty initiating sleep just as much as individuals with insomnia who are still awaiting CBTi treatment. On the contrary, individuals with insomnia who are less reactive to their environment may have less insomnia symptoms after CBTi. Furthermore, following CBTi, patients with chronic primary insomnia who had less positive affect, as measured by the PANAS, had greater improvements in sleep, as assessed by the PSQI, than patients with more positive affect (Van Houdenhove et al., 2011). Although there are mixed findings regarding affect or emotions, they may have an influence on treatment response in individuals with insomnia and should be further investigated.

Despite the growing recognition of the importance of the relation between emotions and sleep, it is still unclear if CBTi may beneficially affect emotion regulation and consequently impact the neural activations underlying emotion reactivity in adults with chronic primary insomnia. As prior studies did not integrate subjective responses of negative, neutral, and sleep-related stimuli and brain responses toward these stimuli, further research is needed to better understand the effectiveness of CBTi on emotion reactivity subjectively and objectively using fMRI. Additionally, the role that emotion reactivity plays for treatment response in individuals with chronic primary insomnia is unclear.

Objectives and Hypotheses

The three main aims of the current study are the following:

1. To examine fMRI brain responses to negatively valenced images in adults with chronic primary insomnia and healthy sleepers.
2. To investigate the effects of CBTi on brain activation during the emotion reactivity task in adults with chronic primary insomnia.

3. To determine if brain activation while viewing sleep-related images during the emotion task at baseline was predictive of CBTi treatment response (i.e., improvement in sleep, as assessed by the ISI) in adults with chronic primary insomnia.

Based on past literature, we hypothesized the following:

1. At baseline, adults with chronic primary insomnia would have greater amygdala activity while rating sleep-related images during an emotion reaction task compared to the healthy sleepers. On the other hand, adults with chronic primary insomnia would have less amygdala activity while rating negative images compared to the healthy sleepers.
2. Following CBTi, participants with chronic primary insomnia would have decreased amygdala activity relative to baseline while rating sleep-related images during the emotion reaction task. Participants who received immediate treatment would also have decreased amygdala activity while rating sleep-related images during the emotion task compared to the participants with insomnia in the wait-list control condition.
3. Lastly, based on Blanken et al.'s (2019) study, less amygdala activity (reflecting less emotion reactivity) while rating sleep-related pictures during the emotion task at baseline would predict better treatment response (i.e., improvement in sleep, as assessed by the ISI) among individuals with chronic primary insomnia.

Methods

Study Design

This study is part of a larger research project in which a randomized controlled trial design was implemented to investigate neuroimaging biomarkers of treatment response to cognitive-behavioural therapy for chronic insomnia. However, the first aim of this current study was to examine brain activity during an emotion reactivity task in adults with chronic primary insomnia (i.e., an immediate treatment group and a wait-list control group) relative to a group that was composed of good sleepers. The second aim was to investigate the effects of CBTi on brain activity during an emotion reactivity task in adults with chronic primary insomnia. The third aim was to examine if brain activity could predict sleep improvements in these adults following CBTi.

As shown in Figure 1, participants with chronic primary insomnia were screened for eligibility at the different steps of the study (phone screening, intake interview). All eligible participants were then scheduled for their first overnight visit at the sleep laboratory, which was called V0. During this visit, they completed online self-report questionnaires about their sleep, mood, and health habits. In order to screen participants out for periodic limb movement and sleep apnea, polysomnography was performed. After this visit, they completed sleep diaries in the morning as well as wore a Philips Actiwatch 2 device for 14 days in order to assess their sleep objectively and to be able to make comparisons with their subjective sleep parameters.

Eligible participants were scheduled for their second overnight visit (i.e., V1), which consisted of neuroimaging and sleep assessments. They first received training on the emotion reactivity task on a computer and then performed one version of the emotion reactivity task within an MRI scanner. In the evening after the MRI, they completed online self-report questionnaires about their sleep and psychological state. Additionally, polysomnography was performed. The

following morning, participants were then randomized to either the immediate treatment group (TX) or the wait-list control (WL) group. The individualized CBTi consisted of eight sessions with a trained psychologist or expert over a 12-week period. After completing the CBTi sessions, participants in the immediate treatment group returned to the laboratory for their second neuroimaging assessment (i.e., V2). However, participants in the wait-list control group returned to the laboratory for their second neuroimaging assessment after a 12-week wait period. The protocol for V2 was identical to V1 (i.e., neuroimaging, online questionnaires, polysomnography, actigraphy, sleep diaries). After V2, participants in the wait-list control group then received individualized CBTi. In order to assess the efficacy of the CBTi treatment, the wait-list control group completed self-report questionnaires at home after completing their CBTi sessions (i.e., V3). They also completed sleep diaries and wore a Philips Actiwatch 2 device for 14 days. A year after receiving CBTi (i.e., one-year follow-up), all participants with insomnia then completed online questionnaires. They also filled out sleep diaries for 14 days. The protocol for the good sleepers was identical, except that they were only scheduled for the first two overnight visits (i.e., V0 and V1) and did not complete sleep diaries nor wear the Actiwatch device following V1. All participants signed an informed consent form. Upon completion, the participants were financially compensated for participating in the study. This project received ethical approval from the Comité central d'éthique de la recherche du ministère de la santé et des services sociaux.

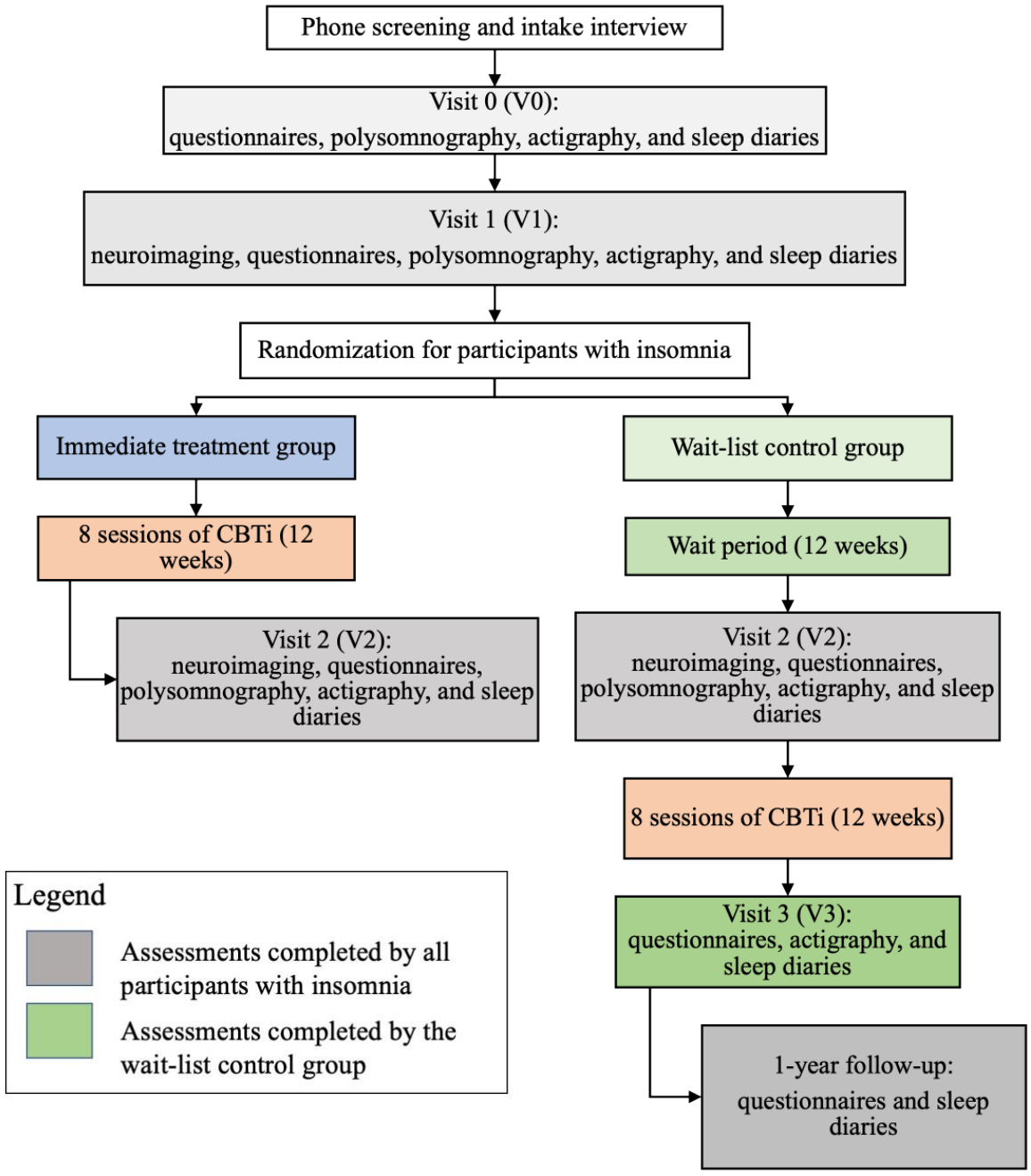
Participants

Targeted Sample Size

The overall targeted sample size for the larger research project entails 80 adults with chronic primary insomnia and 40 good sleepers. However, the targeted sample size for the current

Figure 1

Protocol of the Study



project was at least 10 participants per group. Hence, we aimed to have at least 20 participants with insomnia and at least 10 good sleepers.

Inclusion Criteria

Adults with insomnia were eligible to participate in the study if they met the following diagnostic criteria for insomnia disorder from the third edition of the International Classification of Sleep Disorders (AASM, 2014): difficulty falling asleep, difficulty staying asleep, and/or experienced early awakenings at least three times per week for at least three months. They had to have complaints of daytime impairment that were associated with their sleep difficulties, such as mood disturbances or impaired attention.

Adults with normal sleep were eligible to participate as control participants if they did not have any sleep complaints and if they did not meet the aforementioned criteria for insomnia.

Exclusion Criteria

Individuals were not eligible to participate in the study if they were younger than 25 years old or older than 65 years old. They could not have any medical conditions that can affect their sleep, major cardiovascular events or interventions, or other sleep disorders. Individuals were not eligible if they had night shifts or rotating shift for more than two weeks in the past three months. They were excluded if they had cognitive impairment (i.e., dementia) or severe mental disorder (i.e., psychotic disorders, anxiety disorder, major depressive disorder). They were not eligible if they consumed more than 10 glasses of alcohol per week or smoked more than 10 cigarettes per day. Women were not eligible if they were pregnant or breastfeeding. If individuals were receiving psychotherapy or were using hypnosedatives or medication for depression or anxiety, they were excluded because these treatments can be confounding variables with the CBTi intervention and

the fMRI responses. Adults were also ineligible if they had any counterindications to MRI, such as metallic implants or claustrophobia.

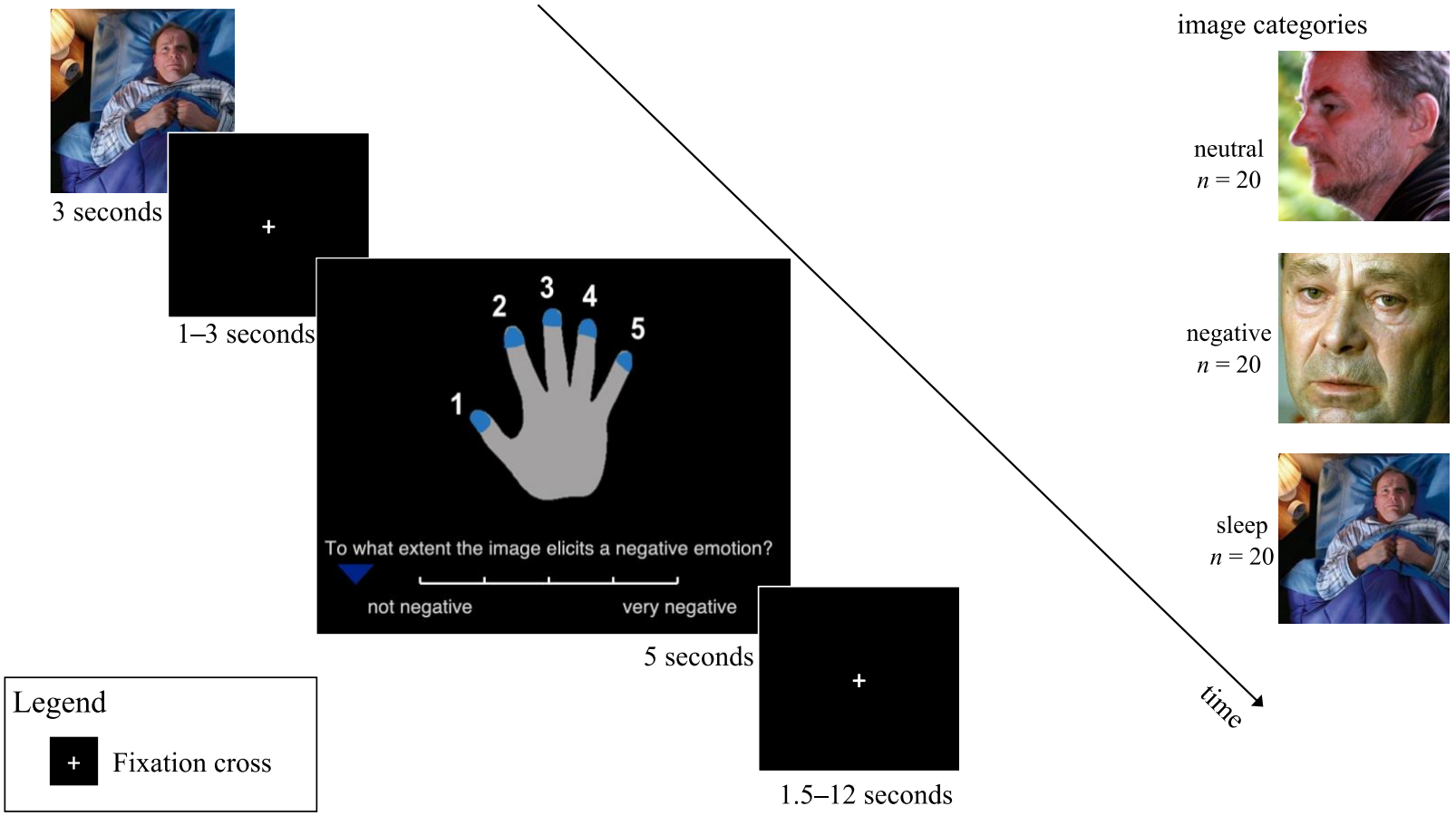
Measures

Emotion Reactivity Task

To assess emotion reactivity and its neural correlates in insomnia, participants completed an emotion reactivity task in the MRI scanner. Emotion reactivity can be measured by presenting an individual with an emotional stimulus and then evaluating threshold, peak intensity, or duration of one's emotional response (Derryberry & Reed, 2003, as cited in Baglioni et al., 2010b). Nevertheless, in this study, the average intensity was the only component of interest. As presented in Figure 2, participants viewed images and provided valence ratings of images that varied, in terms of their valence and arousal, while changes in brain activity in response to these images was measured using fMRI. The task was presented using the standalone PsychoPy version 3.0.7 and lasted 13 minutes. To avoid any practice effects when the task was repeated at V2, two versions of the emotion reactivity task were used. Each version included different images. To minimize order effects, the order was also counterbalanced across participants. Overall, there were 20 trials for each category: neutral, negative, or sleep-related. For each trial, an image belonging to one of the three categories was presented for three seconds. This was followed by a fixation cross for 1.7 seconds on average, ranging from 1 to 3 seconds. A rating scale then appeared for five seconds along with the following question: "To what extent the image elicits a negative emotion?" Response options ranged from 1 (not negative) to 5 (very negative). Another fixation cross was displayed for 3.2 seconds on average, ranging from 1.5 to 12 seconds (Baglioni et al., 2014).

Figure 2

Schematic of the Emotion Reactivity Task



Note. Examples of each image category are presented on the right.

Visual Stimuli

The 40 negative and 40 neutral images were acquired from the Nencki Affective Picture System (NAPS) database (Marchewka et al., 2014). All neutral images depicted faces or people. A total of 40 sleep-related images were used in the emotion reactivity task: 10 came from the database that was used by Baglioni and colleagues (2014) and 30 were obtained from the Internet. The Baglioni images along with the 30 images from a set of 100 new visual stimuli were validated by good sleepers and individuals with insomnia along three different dimensions on the Amazon Mechanical Turk platform. Respondents first completed the ISI. If they obtained an overall score greater than 14, they were classified as individuals with insomnia (n = 29). On the contrary, if they had an overall score below seven, they were classified as good sleepers (n = 30). All respondents were then presented with 100 images. There were 29 images that did not include any people. Respondents had to provide valence ratings for each image (1 = very negative to 9 = very positive, with 5 = neutral). They also assessed how sleep-related each image was (1 = not sleep-related to 9 = very sleep-related, with 5 = ambivalent). Lastly, they indicated the arousal intensity of each image (1 = relaxed to 9 = aroused, with 5 = neutral/ambivalent). The participants were compensated 12 dollars per hour. For these 100 new visual stimuli, the average arousal, valence, and sleep ratings for individuals with insomnia were, respectively, 5.20 (SD = 1.80), 4.60 (SD = 2.10), and 7.10 (SD = 0.80). The average arousal, valence, and sleep ratings for good sleepers were, respectively, 4.60 (SD = 1.50), 4.90 (SD = 1.90), and 6.60 (SD = 0.80). The valence, arousal, and sleep ratings for the images that were included in each version of the emotion reactivity task are presented in Table 1. The valence, arousal, and sleep ratings for sleep-related images in each version specifically of participants with insomnia and good sleepers are presented in Table A1.

Table 1

Valence, arousal, and sleep ratings for images in each version of the emotion reactivity task

Rating Type	Image Category	Version 1		Version 2	
		M	*SD*	*M*	*SD*
Valence					
	Neutral	5.17	0.25	5.29	0.39
	Negative	3.42	0.41	3.41	0.35
	Sleep-related	2.85	0.72	3.11	0.56
Arousal					
	Neutral	5.11	0.54	5.29	0.59
	Negative	5.98	0.45	6.03	0.35
	Sleep-related	6.34	0.66	6.23	0.43
Sleep					
	Neutral	0.00	0.00	0.00	0.00
	Negative	0.00	0.00	0.00	0.00
	Sleep-related	6.72	0.51	6.51	0.46

Note. Data are from good sleepers and participants with insomnia who completed the survey on the Amazon Mechanical Turk platform

Insomnia Severity Index

The Insomnia Severity Index (ISI) is a reliable and valid tool to examine treatment response in individuals with insomnia, which was used to assess insomnia severity at baseline and treatment response. It is a seven-item measure on a 5-point Likert scale that assesses the severity of insomnia symptoms over the last two weeks. The overall score ranged from 0 to 28 (0–7: no insomnia; 8–14: sub-threshold insomnia; 15–21: moderate insomnia; 22–28 severe insomnia). The internal consistency of the ISI scores was .91 for a sample who had insomnia. Responders of CBTi were defined as participants whose total ISI score dropped by seven or more following CBTi. If participants obtained a total ISI score lower than eight after CBTi, they were in remission (Morin et al., 2011).

Sleep Diaries

To evaluate treatment efficacy on sleep in participants with insomnia, we used the consensus sleep diary. Daily sleep diaries consist of 19 questions about daily activities (e.g., "Yesterday, at what time did you start work, school, housework, volunteer activities, child or family care?") and sleep (e.g., "Last night, at what time did you get into bed?"). Participants either completed them online every morning or filled out a paper version. The subjective sleep parameters of interest, which were averaged over two weeks, were total sleep time, sleep onset latency, wake after sleep onset, and sleep efficiency. More specifically, total sleep time (TST) was defined as the amount of time spent asleep in hours. Sleep onset latency (SOL) consisted of the time it took one to fall asleep in minutes (Benca et al., 1992). Wake after sleep onset (WASO) was defined as the total amount of time spent awake throughout the night in minutes following sleep onset. Lastly, sleep efficiency (SE) was derived using the ratio of time spent asleep and time spent in bed, which was then multiplied by 100 to get a percentage (Shrivastava et al., 2014).

Positive Affect Negative Affect Schedule

To assess treatment efficacy on affect in participants with insomnia, the Positive Affect Negative Affect Schedule (PANAS) was used. The PANAS assesses one's emotions or feelings within the last two weeks. It is composed of 10 descriptive words for positive feelings or emotions (e.g., "excited") and 10 other words for negative feelings or emotions (e.g., "distressed"). Individuals were asked to rate how much each word reflects the emotions or feelings that they experienced over the past two weeks on a 5-point Likert scale (1 = not at all to 5 = very much). The overall scores ranged from 10 to 50. Lower scores on the PANAS reflected low positive or negative affect, whereas higher scores reflected high positive or negative affect (Watson et al., 1988).

MRI Data Acquisition

A 3-Tesla GE Discovery MR750 MRI scanner with a 32-channel head coil was used to acquire the MRI data at 6:00 PM. A T2*-weighted sequence was used during the emotion reactivity task (312 volumes, 41 axial slices, TR 2500ms, TE 26ms, flip angle 77°, field of view 25.6cm, resolution 4x4x4mm, matrix size 64x64). T1-weighted structural images were also obtained using the following BRAVO sequence parameters: 196 axial slices, TR 7908ms, TE 3.06ms, flip angle 12°, field of view 25.6cm, resolution 1x1x1mm, matrix size 256x256.

MRI Processing

FSLeyes version 0.34.2 and Mango version 4.1 softwares were used to visually check the quality of the MRI data. Then, the data were pre-processed using Statistical Parametric Mapping 12 (SPM12) within MATLAB. Thus, the T1-weighted structural images in native space were first normalized into standard space using the MNI 152 template (ICBM 152; Fonov et al., 2009). In terms of the functional scans, the first two volumes out of 312 volumes were discarded because it

provides enough time for the longitudinal magnetization to reach a stable state. Slice timing correction was performed to correct for the delays in slice acquisition. Thus, each slice was temporally aligned to the reference slice using a least squares approach as well as spatial transformation using six parameters: rotations along the x, y, and z-axis and left-right, up-down, and forward-backward translations. Thus, the images were realigned to correct for any motion. This was followed by coregistration in which the functional images were overlayed with the T1-weighted structural image with the goal of maximizing mutual information. The next step was normalization. The transformation matrix that was defined for the T1 normalization was applied on the functional images. Lastly, the normalized images underwent spatial smoothing to improve the signal-to-noise ratio and to minimize spatial interindividual variability.

First-Level Analyses: Individual Analyses

Statistical analyses involving fMRI data were conducted in SPM12. For each participant, a general linear model was conducted to examine brain activation within each voxel of the brain that was related to the onset and duration of the following conditions: neutral, negative, and sleep-related. To account for delays in the hemodynamic response, the timing of each condition was convolved with the timing of the canonical hemodynamic response function. The six motion parameter estimates were included as covariates. The following first-level contrasts were defined: each condition relative to the unmodelled baseline (i.e., fixation crosses during the emotion reactivity task). Contrasts between conditions (i.e., negative vs. neutral or sleep-related vs. neutral) identified voxels that were more activated in response to one condition over another.

Second-Level Analyses

Emotion Reactivity in Insomnia

In terms of the second-level analyses, contrasts for each condition were analyzed within each group. Then, to examine if participants with insomnia had heightened emotion reactivity to negatively valenced stimuli at baseline, contrasts for the negative and sleep-related conditions were compared between participants with insomnia and good sleepers. Age and sex were included as covariates. We specifically assessed group differences in brain activity in response to negative vs. neutral images as well as sleep vs. neutral images. For region of interest (ROI) analyses, the anatomical masks were created using the Wake Forest University PickAtlas 3 (WFU PickAtlas 3) toolbox (Maldjian et al., 2003; Maldjian et al., 2004) on MATLAB. Although we did not correct for multiple comparisons across the whole brain, we set the uncorrected *p* value to .001 at the voxel level. The extent threshold was set to zero voxels.

Therapists

In total, there were five different therapists who administered the CBTi to participants with insomnia. To make sure there were no differences between the two insomnia groups that could be accounted for by therapists, we used the Pearson chi-squared test. It indicated that there was no statistically significant association between insomnia group and therapist allegiance, $\chi^2(5) = 3.81$, $p = .58$. As we were already controlling for age and sex, we did not account for therapist in our statistical analyses.

Duration of Insomnia Symptoms

We also investigated if duration of insomnia symptoms (in months) should be included as an additional covariate in the statistical analyses. To ensure that there were no differences between the two insomnia groups that could be accounted for by duration of insomnia symptoms, we

performed an independent-samples *t*-test. It indicated that the TX (M = 215.11, SD = 203.77) and WL (M = 143.56, SD = 111.33) groups did not differ in duration of insomnia symptoms, $t(16)$ = 0.92, p = .37, Hedges' g = 0.41. As there were no differences, we also did not account for duration of insomnia symptoms in our statistical analyses.

Effect of CBTi

A mixed model with two factors (group and visit) was applied to the contrasts obtained from the first-level analyses. In order to determine if there were differences in brain activation in response to a given condition between groups or across time, group comparisons were made between the immediate CBTi group and the wait-list control group. Visit consisted of the first and second neuroimaging assessments. Age and sex were included as covariates. Once again, the contrasts that we examined were the following: negative vs. neutral images and sleep vs. neutral images.

Prediction of Treatment Response

Multiple linear regression was also conducted to investigate if amygdala activity while viewing sleep-related images during the first exposure to the emotion task predicts response to CBTi in participants with insomnia. Age and sex were included as the main covariates of interest. The primary outcome variable was total ISI score, whereas subjective sleep parameters obtained from the sleep diaries (i.e., total sleep time, sleep onset latency, sleep efficiency, wake after sleep onset) were secondary outcome variables.

Valence Ratings

A two-way repeated measures analysis of variance (ANOVA) was conducted to investigate if there were differences in valence ratings for each image category on the emotion reactivity task between the participants with insomnia and the good sleepers at baseline. A mixed model ANOVA

was also conducted to examine if there were differences in valence ratings for each image category on the task in the immediate treatment group after receiving CBTi relative to the wait-list control group.

PANAS

Independent-samples *t*-tests were performed to examine if there were any differences in subjective positive and negative affect between the insomnia group and good sleepers at baseline. A mixed model ANOVA was also conducted to determine if there were changes in subjective affect in the immediate treatment group, following CBTi, compared to the wait-list control group.

Results

Data Integrity

A few issues were addressed before running the analyses. One good sleeper was excluded from the study because a software update on the laptop interrupted the MRI scans. Therefore, we did not acquire the subjective ratings and the MRI data during the emotion reactivity task for this participant. Overall, the final sample size was 26 instead of 27.

Statistical assumptions were also assessed. For the mixed ANOVAs, outliers were identified using the boxplot method. If a given value was greater than 'Q3 + 3*IQR' or lower than 'Q1 – 3*IQR,' then it was considered an extreme outlier. Nevertheless, none of the participants were excluded from the statistical analyses. The Levene's test was performed to test the assumption of homogeneity of variances. To test the assumption of sphericity, the Mauchly's test was used. If this assumption was violated, the Greenhouse-Geisser correction was implemented. The Shapiro-Wilk test was conducted to determine if the outcome variable of interest was normally distributed for each cell. If normality was violated, the aligned rank transform (ART) method was used (Wobbrock et al., 2011; Kay et al., 2021). Once the data were transformed, the ANOVA was performed. To conduct post-hoc pairwise comparisons with this method, contrast tests with ART were performed (Elkin et al., 2021). The Bonferroni method was used to correct for multiple comparisons. For independent-samples *t*-tests or dependent-samples *t*-tests, outliers were detected using the method that was previous described. The assumption of homogeneity of variances was either assessed using the Levene's test or Bartlett's test. The assumption of normality was assessed using the previously described test. If the assumption of normality was violated, the Wilcoxon rank sum test was used instead of the independent-samples *t*-test.

The Cronbach's alpha coefficients were calculated for the scores on the ISI and PANAS questionnaires in our own sample. When including all participants in the analysis, the Cronbach's alpha coefficients for the PANAS scores at V0 and ISI scores at V1 ranged from .79 to .93. When including all participants who completed the same questionnaires during V2, the Cronbach's alpha coefficients for the PANAS and ISI scores were between .91 and .95. More detailed results are presented in Table B1.

Participant Characteristics

Demographic data are presented in Table 2. There were four males in each group. In addition, there were five females in the two insomnia groups and four female good sleepers. The one-way ANOVA, to examine if the three groups differed in age, showed no statistically significant difference between groups ($F(2,23) = 2.20$, $p = .13$). The Kruskal-Wallis test, to determine if there were differences in education level between the three group, showed no difference in education level ($H(2) = 0.17$, $p = .92$). In terms of handedness, one participant in the TX group and two participants in the WL group were left-handed. However, none of the good sleepers were left-handed.

Subjective Sleep Measures

ISI

We conducted an independent-samples *t*-test to evaluate if participants with insomnia and good sleepers differed in severity of insomnia symptoms at baseline. As shown in Figure C1, the total ISI score was higher in the insomnia group ($M = 15.61$, $SD = 3.84$) compared to the good sleepers at V1 ($M = 1.50$, $SD = 1.42$) at V1, $t(23.62) = 13.66$, $p < .001$, Hedges' $g = 4.12$.

Furthermore, we examined the effects of group and visit on total ISI score in participants with insomnia. The analysis of variance of align rank transformed data test indicated that there

Table 2

Demographic data of each group

Characteristic	TX (n = 9)	WL (n = 9)	GS (n = 8)
Sex			
Male/Female	4/5	4/5	4/4
Age (year)	46.1 ± 13.52	42.6 ± 13.55	33.7 ± 9.48
range	26–65	28–65	25–49
Education			
High school	1	0	0
CEGEP	0	1	0
Bachelor	3	4	4
Master	5	3	3
PhD	0	1	1

Note. Sex and education are presented as frequencies, whereas age is presented as means ± standard deviations. TX: immediate treatment group; WL: wait-list control group; GS: good sleeper group

were group, visit, and group by visit interaction effects on total ISI score (Table C1). As shown in Figure C2, the interaction contrast indicated that the difference in total ISI score between the TX group and WL group at V1 differed from that at V2, $\chi^2(1) = 20.13$, $p < .001$. The total ISI score was lower in the TX group than the WL group at V2. Overall, seven out of nine participants in the TX group had a change in total ISI score by seven or more points, suggesting that they were responders to CBTi. No participants in the WL group had a decrease in total ISI score by seven or more points. Furthermore, eight out of nine participants in the TX group had a total ISI score lower than eight following CBTi, indicating that they were in remission. Only one out of nine participants in the WL group had a total score lower than eight at V2.

Sleep Diaries

In comparison with the good sleepers, participants with insomnia self-reported on sleep diaries having longer sleep onset latency ($W = 33$, $p = .03$), more awakenings ($W = 11$, $p < .001$), longer awakenings ($t(20.77) = 4.33$, $p < .001$, Hedges' $g = 1.23$), shorter sleep duration ($W = 117$, $p = .01$), and lower sleep efficiency following V0 ($t(24) = –3.05$, $p = .01$, Hedges' $g = –1.26$). The average subjective sleep measures, which were acquired from sleep diaries, for participants with insomnia and good sleepers at baseline are presented in Table C2. On average, the number of days that all participants filled out sleep diaries following V0 was 13.54 days.

PANAS

We performed independent-samples *t*-tests to test whether participants with insomnia and good sleepers differed with respect to self-reported ratings of positive and negative affect at baseline. As presented in Figures D1 and D2, we did not find a statistically significant difference in total positive affect scores between participants with insomnia ($M = 33.28$, $SD = 7.47$) and good sleepers ($M = 33.63$, $SD = 6.25$) at V0, $t(24) = 0.11$, $p = .91$, Hedges' $g = 0.05$. However,

participants with insomnia self-reported having greater negative affect (M = 19.28, SD = 5.96) than good sleepers (M = 15.25, SD = 2.38) at V0, $t(23.91) = 2.46$, $p = .02$, Hedges' $g = 0.75$.

Additionally, we conducted a mixed model ANOVA to determine if there were changes in subjective affect in the TX group, following CBTi, compared to the WL group. As shown in Figure D3 or Table D1, there was a group effect on positive affect. On average, participants in the WL group self-reported having less positive affect than those in the TX group, $F(1,16) = 9.92$, $p = .01$. But, there was no visit nor group by visit interaction effect on positive affect. Additionally, there were no group, visit, nor group by visit interaction effects on negative affect (Figure D4; Table D2).

Valence Ratings

We conducted a two-way repeated measures ANOVA to investigate whether participants with insomnia and good sleepers differed with respect to valence ratings of neutral, negative, and sleep-related images during the emotion reactivity task. As shown in Figure E1 or Table E1, there was no statistically significant group by condition interaction effect on rating, $F(1.62, 38.84) = 2.66$, $p = .09$. However, there was a statistically significant main effect of condition on rating, $F(1.62, 38.84) = 98.93$, $p < .001$. Negative images (M = 3.58, SE = 0.12) were rated as more negative than neutral images (M = 1.74, SE = 0.10), $M_{difference} = 1.84$, $SE = 0.10$, $p < .001$. Sleep images (M = 2.66, SE = 0.15) were also rated as more negative than neutral images, $M_{difference} = 0.92$, $SE = 0.16$, $p < .001$. Additionally, negative images were perceived as more negative than sleep images, $M_{difference} = 0.91$, $SE = 0.13$, $p < .001$. There was also a statistically significant effect of group on rating, $F(1, 24) = 5.06$, $p = .03$. On average, participants with insomnia ($M = 2.89$, $SE = 0.11$) rated the images as more negative than good sleepers ($M = 2.43$, $SE = 0.17$) at V1, $M_{difference} = 0.46$, $SE = 0.21$, $p = .03$. The average valence ratings for both groups at V1 are presented in

Table E2. Independent-samples *t*-tests showed no statistically significant difference in valence rating of neutral images between participants with insomnia and good sleepers at V1 ($t(24) = 1.88$, $p = .07$, Hedges' $g = 0.77$). There was also no statistically significant difference in rating of negative images between the insomnia group and good sleepers at V1, $t(24) = 0.83$, $p = .42$, Hedges' $g = 0.34$. But, participants with insomnia did rate sleep-related images as more negative than good sleepers at V1, $t(24) = 2.63$, $p = .01$, Hedges' $g = 1.08$.

Furthermore, we ran a mixed model ANOVA to investigate if there were differences in valence ratings for each image category on the task in the TX group after receiving CBTi relative to the WL group. As shown in Figure E2 or Table E3, there were no group nor group by visit interaction effects on rating of neutral images. However, there was a statistically significant visit effect. On average, participants with insomnia rated the neutral images as more negative at V1 than at V2, $t(16) = 2.85$, $p = .01$. Similarly, although there were no group nor group by visit interaction effects on rating of negative images, there was a statistically significant main effect of visit (Figure E3; Table E4). Participants with insomnia, on average, reported that the negative images were more negative at V1 than V2, $t(17) = 3.03$, $p = .01$. However, there were no group, visit, nor group by visit interaction effects on rating of sleep-related images (Figure E4; Table E5).

Objective 1: Insomnia vs. Good Sleepers at Baseline

Activity in Response to Emotional Pictures Within the Amygdala

Irrespective of controlling for the family-wise error rate (FWER) at the level of .05, amygdala activity in response to sleep-related vs. neutral images did not differ between participants with insomnia and healthy sleepers at V1. There were also no group differences in amygdala activity in response to negative vs. neutral images at V1. Thus, we investigated if there

were group differences in activity within the whole brain in response to the negatively valenced images at baseline.

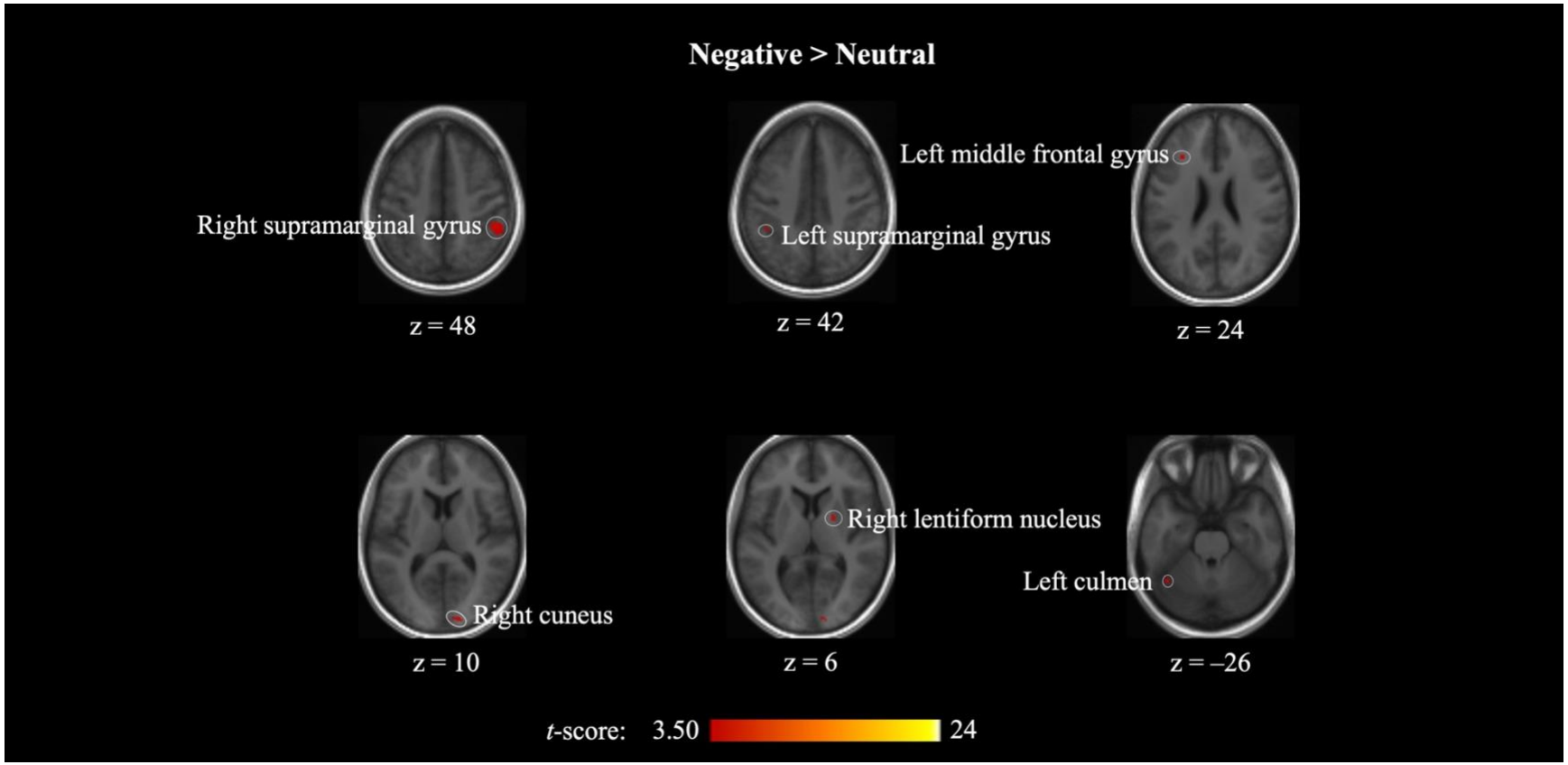
Activity in Response to Emotional Pictures Within the Whole Brain

We first examined brain regions that were activated in response to neutral, negative, and sleep images relative to baseline in each group at V1. The brain maps for the good sleepers and participants with insomnia are presented in Figures F1–F3. More details about the clusters are presented in Tables F1–F6.

When we examined if participants with insomnia had greater brain activity than good sleepers at V1 in response to sleep in contrast to neutral images, we did not find any statistically significant differences. We also assessed if good sleepers had greater brain activity than participants with insomnia at V1 in response to negative relative to neutral images. In this case, good sleepers had greater activity in the right cuneus, right lentiform nucleus, left and right supramarginal gyri, left culmen, left middle frontal gyrus, left and right middle occipital gyri, right inferior frontal gyrus, left superior frontal gyrus, and left superior temporal gyrus (Figure 3; Table 3).

Figure 3

Brain regions that are more activated in good sleepers compared to participants with insomnia in response to negative relative to neutral images at V1



Note. Height threshold T: 3.50 ($p < .001$ uncorrected at the voxel level).

Table 3

Clusters statistically significantly more activated in good sleepers compared to participants with insomnia in response to negative vs. neutral images at V1

Brain region (BA)	Coordinates (x, y, z)	Cluster-level		Peak-level		
		Cluster size (voxels)	$p_{uncorrected}$	t	z_E	$p_{uncorrected}$
Right cuneus	14, –96, 10	21	.41	4.70	3.87	< .001
Right lentiform nucleus	20, 0, 6	8	.62	4.49	3.75	< .001
Right supramarginal gyrus (40)	48, –40, 48	104	.07	4.36	3.66	< .001
Right supramarginal gyrus (40)	54, –50, 50			4.03	3.45	< .001
Left culmen	–42, –58, –26	13	.52	3.93	3.38	< .001
Left middle frontal gyrus (9)	–32, 26, 24	8	.62	3.84	3.32	< .001
Left supramarginal gyrus (40)	–42, –44, 42	18	.44	3.82	3.31	< .001
Left supramarginal gyrus (40)	–40, –36, 38			3.73	3.25	.001
Right inferior frontal gyrus (9)	58, 16, 26	5	.71	3.73	3.25	.001
Left superior frontal gyrus (6)	–8, 12, 60	8	.62	3.68	3.21	.001

Note. Coordinates represent peak voxel coordinates in millimeters; For clarity, only clusters with at least five voxels were listed in the table; BA = Brodmann area; Height threshold T: 3.50 ($p < .001$ uncorrected at the voxel level).

Objective 2: Immediate Treatment vs. Wait-List Control Groups (Baseline vs. V2)

Activity in Response to Sleep-Related Pictures Within the Amygdala

We were not able to investigate the interaction effects within the amygdala (using ROI analyses) as this region was not activated enough by the task. Hence, we did not assess the effects of CBTi on activity within the left and right amygdala in response to sleep relative to neutral images. But, we did investigate if there were group by visit interaction effects on activity within the whole brain in response to this particular contrast in participants with chronic primary insomnia.

Activity in Response to Sleep-Related Pictures Within the Whole Brain

Using voxel-based whole brain analyses, we examined the effects of CBTi on brain activity in response to sleep relative to neutral images (Table 4). While controlling for age and sex, we found a statistically significant interaction specifically within the left insula and left superior temporal gyrus (Figure 4). The TX group had greater activity within the left insula and left superior temporal gyrus than the WL group at V1. As shown in Figures 5 and 6, activity within those regions decreased over time in the TX group, whereas it increased over time in the WL group. We also found a statistically significant main effect of group, such that participants in the WL group, on average, had greater activity in the left insula, right insula, and left cingulate gyrus than those in the TX group. However, participants in the TX group, on average, had greater activity in the left superior temporal gyrus and left precentral gyrus than those in the WL group. While accounting for age and sex, there was also a statistically significant main effect of visit. On average, activity in the left precentral gyrus and left caudate was higher at V1 than at V2, whereas activity in the left cingulate gyrus and left middle frontal gyrus was higher at V2 than at V1.

Table 4

Activated brain regions in response to sleep relative to neutral images

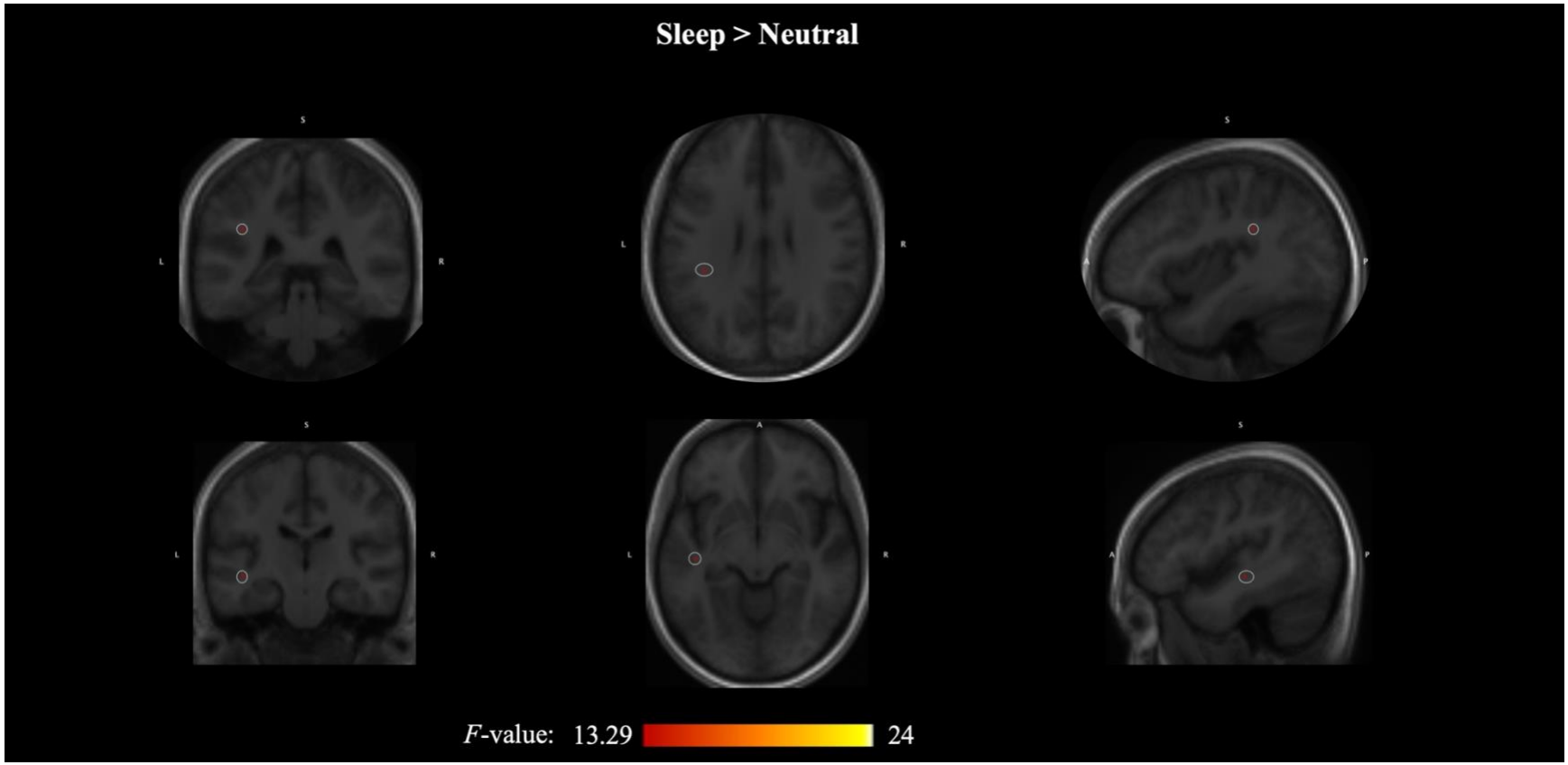
Effect	Brain region (BA)	Coordinates (x, y, z)	Cluster-level		Peak-level		
			Cluster size (voxels)	$p_{uncorrected}$	F	z_E	$p_{uncorrected}$
Group							
	Right insula (13)	42, –8, 22	9	.523	19.32	3.66	.000
	Right insula (13)	48, –10, 12	8	.549	15.89	3.36	.000
	Left superior temporal gyrus (22)	–68, –38, 10	6	.610	15.61	3.33	.000
	Left insula (13)	–42, –6, 18	7	.578	15.00	3.27	.001
	Left cingulate gyrus (24)	–16, 8, 30	1	.861	14.22	3.19	.001
	Left cingulate gyrus (24)	–16, 4, 30	1	.861	14.02	3.17	.001

	Left precentral gyrus (6)	–46, 0, 42	1	.861	13.49	3.11	.001
Visit							
	Left precentral gyrus (6)	–36, –10, 26	47	.142	29.61	4.35	.000
	Left cingulate gyrus (23)	–2, –10, 28	1	.861	13.61	3.12	.001
	Left middle frontal gyrus (10)	–32, 38, 12	1	.861	13.46	3.11	.001
	Left caudate	–20, 6, 22	1	.861	13.45	3.11	.001
Interaction							
	Left insula (13)	–38, –36, 28	1	.861	13.46	3.11	.001
	Left superior temporal gyrus (22)	–44, –22, –8	1	.861	13.37	3.10	.001

Note. Coordinates represent peak voxel coordinates in millimeters; BA: Brodmann area; Height threshold F: 13.29 ($p < .001$ uncorrected at the voxel level).

Figure 4

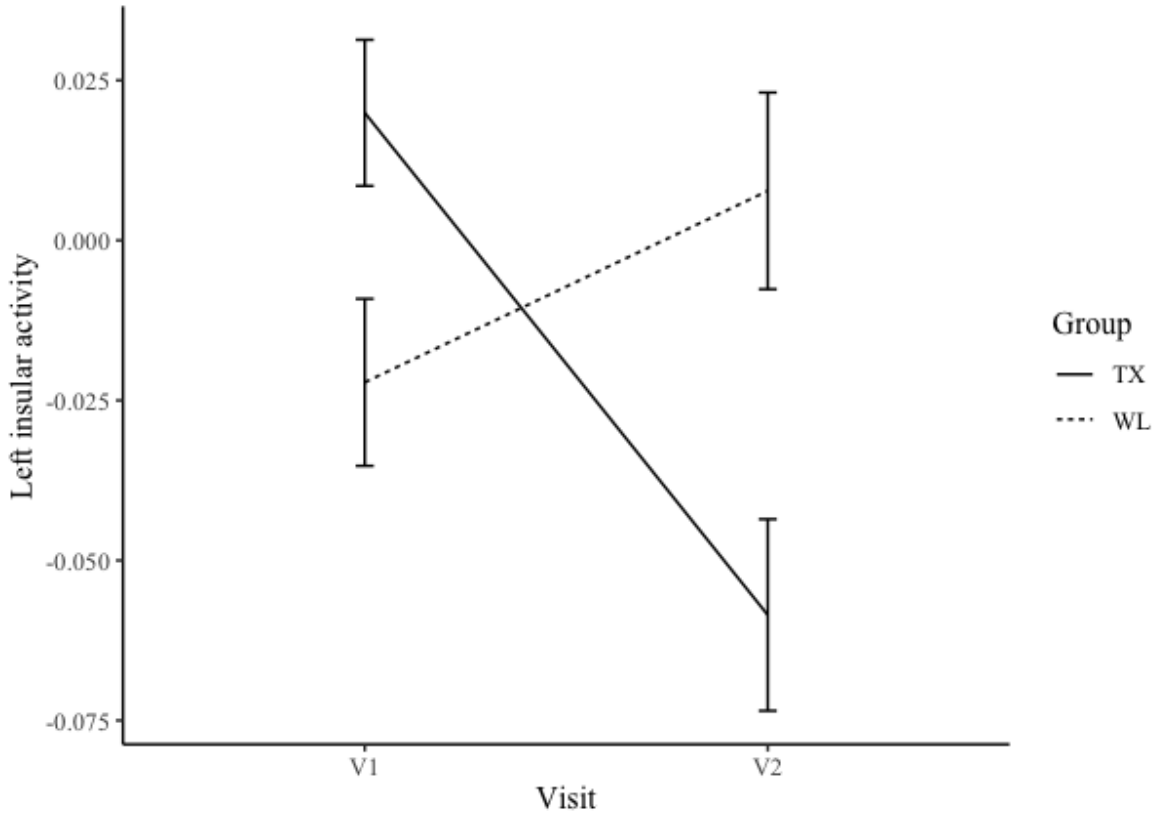
Statistically significant group by visit interaction on activity within the left insula and superior temporal gyrus



*Note***.** Group (TX vs. WL) by visit (V1 vs. V2) interaction on activity within the left insula (top panel) and left superior temporal gyrus (bottom panel) in response to sleep relative to neutral images

Figure 5

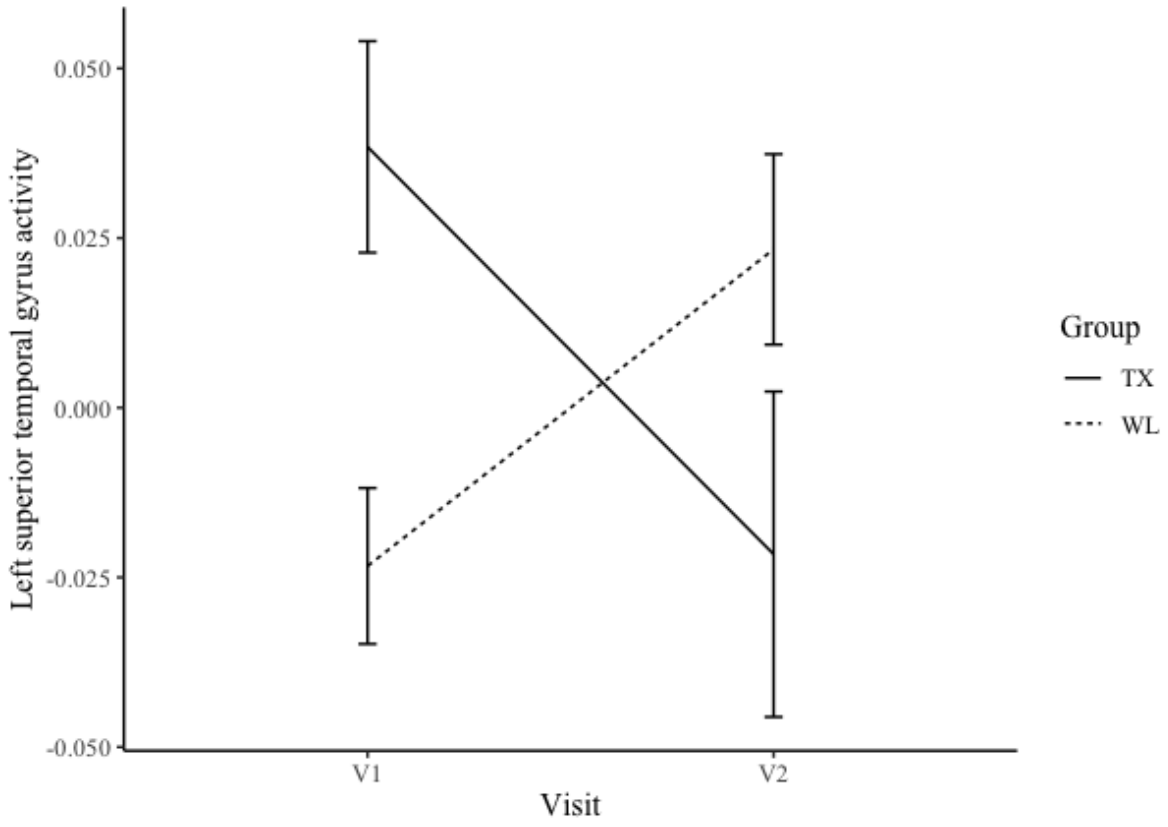
Left insular activity between the insomnia groups at V1 and V2



Note. Error bars represent standard error of the mean; TX: immediate treatment group; WL: wait-list control group

Figure 6

Left superior temporal gyrus activity between the insomnia groups at V1 and V2



Note. Error bars represent standard error of the mean; TX: immediate treatment group; WL: wait-list control group

Objective 3: Neural Predictors of Treatment Response

We originally wanted to investigate if amygdala activity in response to sleep relative to neutral images would predict treatment response in those who received immediate CBTi. However, we could not test this hypothesis, given that there was no amygdala activity in response to this contrast in individuals with insomnia at baseline. Thus, we identified brain regions that were more activated while all participants with insomnia were exposed to sleep images relative to neutral images at V1: the left angular gyrus (Brodmann area 39) and the left middle frontal gyrus (Brodmann area 6). We then explored if activity in each of these brain regions individually predicted CBTi treatment response in the TX group, but also compared it to the WL group.

Left Angular Gyrus and Treatment Response

When we investigated if activity within the left angular gyrus in response to sleep in contrast to neutral images predicted treatment response as assessed by the ISI, we did not find a brain activity by group interaction. Thus, the slopes of the regression between activity within the left angular gyrus at V1 and change in total ISI scores were similar for the TX and WL groups (Tables 5–6; Figure 7). However, there was a statistically significant difference in intercepts between the regression lines of the TX and WL groups, whereby it was higher in the WL group. We also found a statistically significant effect of group on change in total ISI scores. On average, participants in the TX group had greater reductions in total ISI scores over time than those in the WL group. But, we did not find a statistically significant effect of activity in the left angular gyrus on change in total ISI scores.

In terms of number of awakenings, we did not find a brain activity by group interaction. Hence, the slopes of the regression between activity within the left angular gyrus at V1 and change in the number of awakenings were similar for both insomnia groups (Tables G1–G2). But, there

Table 5

Results for BA39 Activity Predicting Change in Total ISI Scores With an Interaction

Source	*df*	*SS*	*MS*	*F*	*p*	η^2
BA39 Activity	1	109.70	109.70	3.67	.08	0.09
Group	1	636.30	636.30	21.32	< .001	0.55
BA39 Activity*Group	1	0.60	0.60	0.02	.89	0.00
Residuals	14	417.90	29.80			

Note. BA39: Brodmann area 39

Table 6

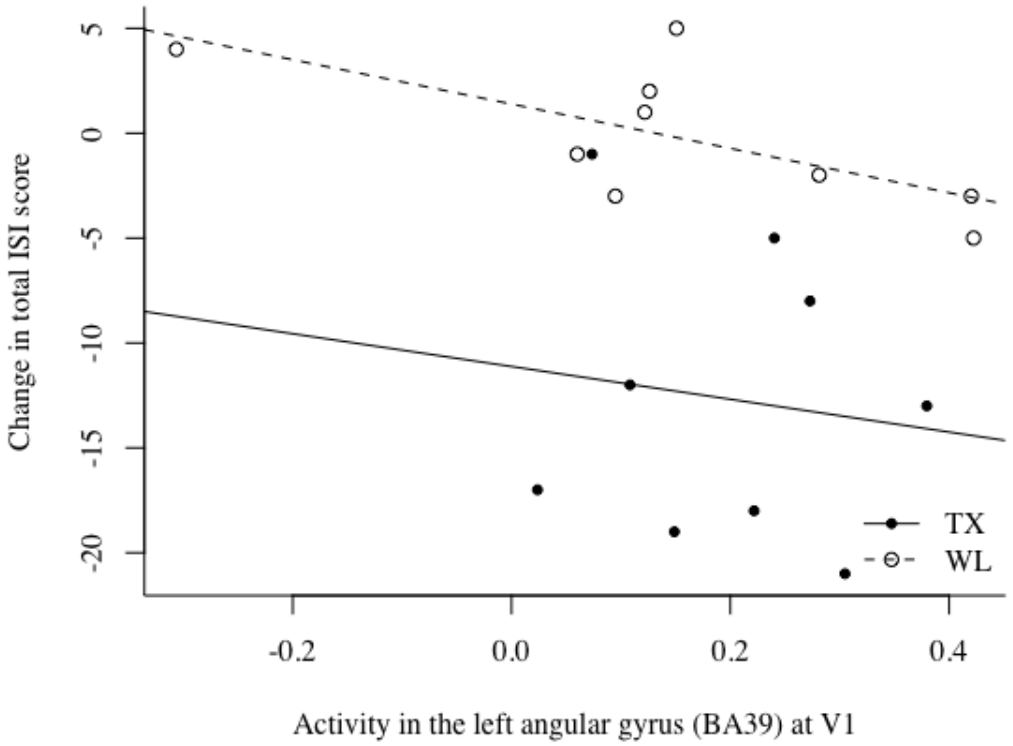
Results for BA39 Activity Predicting Change in Total ISI Scores Without an Interaction

Source	*df*	*SS*	*MS*	*F*	*p*	η^2
BA39 Activity	1	109.70	109.70	3.93	.07	0.09
Group	1	636.30	636.30	22.81	< .001	0.55
Residuals	15	418.50	27.90			

Note. BA39: Brodmann area 39

Figure 7

Brodmann area 39 (BA39) activity predicting change in total ISI scores



Note. TX: immediate treatment group; WL: wait-list control group

was a statistically significant difference in intercepts between the regression lines of the TX and WL groups. More precisely, those in the WL group who had no activity in the left angular gyrus during V1 had more awakenings at V2 than at V1. But, those in the TX group who had lower activity in this region had fewer awakenings at V2 than at V1. Furthermore, there was a statistically significant effect of group on change in number of awakenings throughout the night. Participants in the TX group, on average, had fewer awakenings over time than those in the WL group. However, we did not find a statistically significant effect of activity in the left angular gyrus on change in number of awakenings.

Regarding total sleep time, we did not find a brain activity by group interaction. Thus, the slopes of the regression between activity within the left angular gyrus at V1 and change in total sleep time were similar for both groups (Tables G3–G4). But, there was a statistically significant difference in intercepts between the regression lines of the TX and WL groups, such that the intercept was higher in the TX group. There was also a statistically significant effect of group on change in total sleep time. Participants in the TX group, on average, slept longer across time than those in the WL group. But, we did not find a statistically significant effect of activity in the left angular gyrus on change in total sleep time.

We did not observe a brain activity by group interaction for the following outcome variables: sleep onset latency, duration in awakenings throughout the night, sleep efficiency, or sleep quality. Thus, the slopes of the regression between activity within the left angular gyrus at V1 and change in those outcome variables did not differ for both groups (Figures G1–G4). There was no statistically significant difference in intercepts between the regression lines of the TX and WL groups. Moreover, we did not find a statistically significant effect of group nor effect of activity in the left angular gyrus on change in the previously mentioned outcome variables.

Left Middle Frontal Gyrus and Treatment Response

When we investigated if activity within the left middle frontal gyrus in response to sleep relative to neutral images predicted treatment response as assessed by the ISI, we did not find a brain activity by group interaction. Therefore, the slopes of the regression between activity within the left middle frontal gyrus at V1 and change in total ISI scores did not differ for both insomnia groups (Tables 7–8; Figure 8). However, there was a statistically significant difference in intercepts between the regression lines of the TX and WL groups, such that the intercept was higher in the WL group. Furthermore, we found a statistically significant effect of group on change in total ISI scores. On average, participants in the TX group had a greater decline in total ISI scores across time than those in the WL group. But, we did not find a statistically significant effect of activity in the left middle frontal gyrus on change in total ISI scores.

In terms of number of awakenings, we did not find a brain activity by group interaction. Thus, the slopes of the regression between activity within the left middle frontal gyrus at V1 and change in the number of awakenings did not differ for the TX and WL groups (Tables G5–G6). Nonetheless, there was a statistically significant difference in intercepts between the regression lines of the TX and WL groups, such that the intercept was higher in the WL group. Moreover, we found a statistically significant effect of group on change in number of awakenings. On average, participants in the TX group had less awakenings throughout the night over time than those in the WL group. But, we did not find a statistically significant effect of activity in the left middle frontal gyrus on change in the number of awakenings.

Regarding total sleep time, no brain activity by group interaction was found. This means that the slopes of the regression between activity within the left middle frontal gyrus at V1 and change in total sleep time did not differ for the two groups (Tables G7–G8). But, there was a

Table 7

Results for BA6 Activity Predicting Change in Total ISI Scores With an Interaction

Source	*df*	*SS*	*MS*	*F*	*p*	η^2
BA6 Activity	1	4.60	4.60	0.14	.71	0.00
Group	1	707.60	707.60	21.98	< .001	0.61
BA6 Activity*Group	1	1.60	1.60	0.05	.83	0.00
Residuals	14	450.60	32.20			

Note. BA6: Brodmann area 6

Table 8

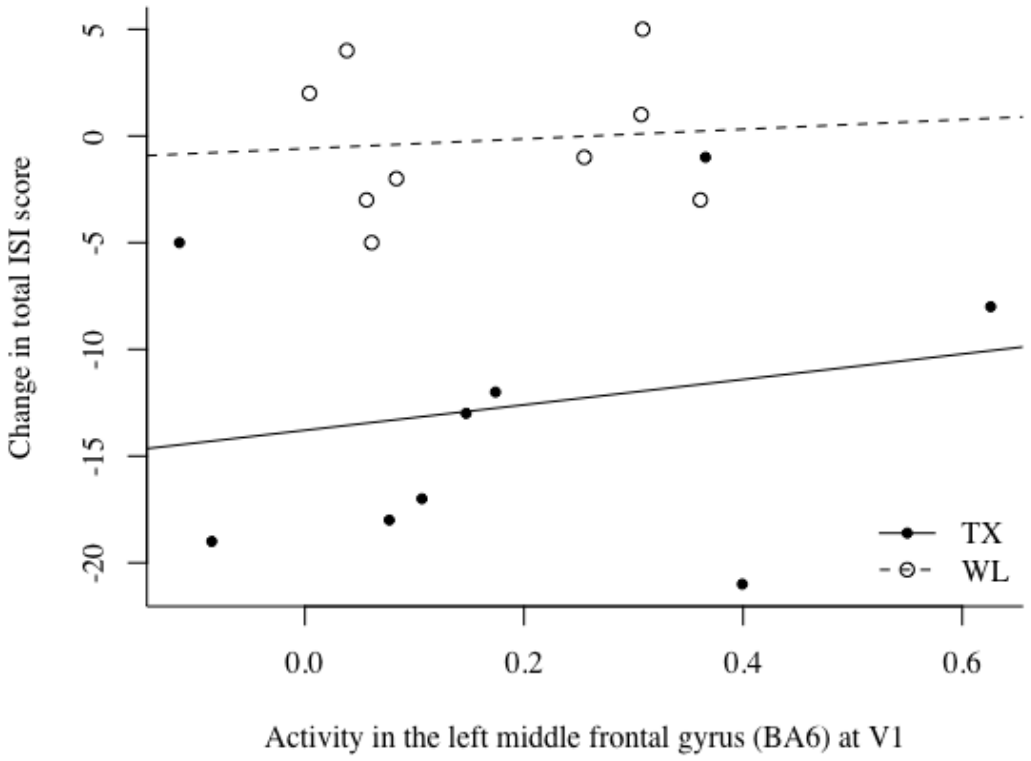
Results for BA6 Activity Predicting Change in Total ISI Scores Without an Interaction

Source	*df*	*SS*	*MS*	*F*	*p*	η^2
BA6 Activity	1	4.60	4.60	0.15	.70	0.00
Group	1	707.60	707.60	23.48	< .001	0.61
Residuals	15	452.2	30.10			

Note. BA6: Brodmann area 6

Figure 8

Brodmann area 6 (BA6) activity predicting change in total ISI scores



Note. TX: immediate treatment group; WL: wait-list control group

statistically significant difference in intercepts between the regression lines of the TX and WL groups. In this case, the intercept was higher in the TX group. Moreover, there was a statistically significant effect of group on change in total sleep time. Participants in the TX group, on average, slept more over time than those in the WL group. But, we did not find a statistically significant effect of activity in the left middle frontal gyrus on change in total sleep time.

In terms of sleep efficiency, no brain activity by group interaction was observed. This indicates that the slopes of the regression between activity within the left middle frontal gyrus at V1 and change in sleep efficiency did not differ for the insomnia groups (Tables G9–G10). But, there was a statistically significant difference in intercepts between the regression lines of the TX and WL groups. More specifically, the intercept was higher in the TX group. There was also a statistically significant effect of group on change in sleep efficiency. Participants in the TX group, on average, had greater sleep efficiency over time than those in the WL group. However, we did not find a statistically significant effect of activity in the left middle frontal gyrus on change in sleep efficiency.

Regarding sleep onset latency, duration of awakenings, and sleep quality, we did not find a brain activity by group interaction. Thus, there were no statistically significant differences in slopes of the regression between activity within the left middle frontal gyrus at V1 and change in sleep efficiency for the two insomnia groups (Figures G5–G7). Additionally, no statistically significant differences in intercepts between the regression lines of the TX and WL groups were observed. There was no statistically significant effect of group on change in sleep onset latency, duration in awakenings throughout the night, or sleep quality. Furthermore, we did not observe a statistically significant effect of activity in the left middle frontal gyrus on change in these outcome variables.

Discussion

In the current study, we examined if there were any differences in self-reported measures of sleep and affect as well as subjective and brain responses during an fMRI-based emotion reactivity task between adults with chronic primary insomnia and good sleepers at baseline. Furthermore, we assessed the effects of CBTi on self-reported measures of sleep and affect as well as subjective and brain responses during this emotion reactivity task in adults with insomnia. We also investigated if amygdala activity in response to sleep vs. neutral images at baseline would predict CBTi treatment response in those in the TX group.

Sleep and CBTi

Consistent with findings from previous studies (Harvey et al., 2008; Li et al., 2017; Sanz-Arigita et al., 2021; Talbot et al., 2012; Wu et al., 2013), our participants with insomnia did report having greater insomnia severity, longer sleep onset latency, more as well as longer awakenings, shorter sleep duration, and poorer sleep efficiency compared to the good sleepers at baseline. These results indicate that our participants with insomnia did report experiencing sleep disturbances that are normally uncommon among healthy sleepers. Following CBTi, participants in the immediate treatment group self-reported having longer sleep duration, increased sleep efficiency, and fewer awakenings throughout the night, which is consistent with previous studies (Cervena et al., 2004; Kim et al., 2017; Kim et al., 2019; Van Houdenhove et al., 2011; Wu et al., 2006). In addition, these individuals had a reduction in total ISI scores after receiving this treatment. A drop in total ISI scores following this specific intervention has been noted in similar studies as well (Cervena et al., 2004; Kim et al., 2017; Kim et al., 2019). Thus, these results show that participants with insomnia reported having improvements in their sleep after completing the CBTi sessions and

provide further evidence for the effectiveness of individualized CBTi for sleep in adults with chronic primary insomnia.

Affect

As mood disorders can co-occur or emerge following chronic insomnia and proposed models suggest that emotions can play a role in the onset and maintenance of insomnia, we assessed if there were differences in affect between participants with insomnia and good sleepers (Harvey, 2002; Johnson et al., 2006; Kales et al., 1976; Ohayon & Roth, 2003). Although our participants with insomnia did not differ from good sleepers in terms of positive affect at baseline, they did report having greater negative affect. Similarly, Buysse and colleagues (2007) found that individuals with primary insomnia reported having more negative mood, particularly in the evening, in comparison to good sleepers. Furthermore, Talbot et al. (2012) reported that individuals with insomnia had more negative mood in the morning and evening than healthy sleepers. In line with our findings, they also did not find any differences in positive mood during the morning and evening between these two groups. Additionally, Ong et al. (2011) found similar results among individuals who were classified as poor sleepers. These poor sleepers reported having more negative affect during the daytime and at night than those classified as good sleepers. On the contrary, Howlett and colleagues (2020) failed to find any differences in positive and negative affect between individuals diagnosed with insomnia and good sleepers. A positive association between negative affect and sleep disturbances has been reported in previous studies (Latif et al., 2019; Ong et al., 2011). Thus, perhaps Howlett and colleagues (2020) did not find any differences in affect because there were also no observed differences in subjective sleep measures, aside from reduced total sleep time. Nevertheless, these findings highlight the heterogeneity of

insomnia whereby some individuals have daytime complaints that consist of mood disturbances (Levenson et al., 2015).

Effects of CBTi on Affect

We hypothesized that participants who received immediate treatment would have less negative affect after receiving CBTi. However, in this study, we only found a difference in positive affect between the two insomnia groups. Positive affect was, on average, lower in the WL group. In Van Houdenhove and colleagues' (2011) study, they reported that individuals with chronic primary insomnia who initially had less positive affect had reduced insomnia severity after receiving CBTi than those with more positive affect. However, Ong et al. (2009) showed that there were no statistically significant impacts of CBTi combined with mindfulness meditation on both positive and negative affect in adults with insomnia. Saxon and colleagues (2017) found improvements, following cognitive-behavioural therapy, in positive affect and a drop in negative affect in individuals who experienced symptoms of depression, anxiety, or both. However, this treatment was not intended to specifically target sleep problems or insomnia symptoms. Although there appeared to be improvements in subjective sleep measures in the TX group, these results may indicate that there were no transfer effects. Thus, CBTi, a treatment primarily used to treat sleep problems, by itself may not be beneficial or effective enough for altering general positive or negative affect (Anderson, 2017).

Valence Ratings of Emotional Pictures

Based on Baglioni and colleagues' (2010) study, we did not expect to find group differences in valence ratings of neutral images. In accordance with this hypothesis, there were no discernable differences in rating for neutral pictures of faces or people between participants with insomnia and good sleepers in our study. Similarly, in Jansson-Fröjmark and colleagues' (2013)

study, participants with primary insomnia and good sleepers did not rate the neutral images differently in terms of valence and arousal. On the contrary, Akram (2020) noted that individuals who reported having clinically significant insomnia symptoms rated neutral faces as both more attractive and happier than good sleepers. However, those with clinically significant insomnia symptoms did not rate the neutral faces as more sad, trustworthy, approachable, healthy, or sociable than the healthy controls. Thus, in both groups, neutral faces may not be perceived as more negative. In a sleep restriction study, good sleepers rated neutral images as more negative when their sleep was restricted for five days than when they slept normally (Tempesta et al., 2019). One explanation for this inconsistent finding could be that they examined good sleepers who were deprived of sleep instead of individuals with chronic insomnia. However, short-term sleep loss may not have the same effects on emotion perception as chronic sleep disturbances.

Based on Baglioni and colleagues' (2010) study, we also did not expect to find group differences in valence ratings of negative images. Consistent with our hypothesis, our participants with insomnia and good sleepers rated negative images similarly. In a sleep restriction study with normal sleepers, males were shown negative pictures and were asked to rate them in terms of valence and arousal before and after a three-hour interval of early or late sleep (i.e., at 11:00 PM or at 3:00 AM) or wake. Before and after the three-hour interval, participants in the early sleep group and late sleep group did not differ in valence nor arousal rating of negative images from the wake group. Compared to the wake group, previously shown negative images were perceived as more positive than new negative images after early sleep, but they were rated as more negative after late sleep (Wagner et al., 2002). In another sleep restriction study with good sleepers, participants did not provide different valence ratings for unpleasant images when their sleep was restricted for five days than when they slept normally (Tempesta et al., 2019). Altogether, these

results suggest that individuals with sleep difficulties and good sleepers tend to accurately perceive negative pictures as negative and in a similar manner.

Based on Baglioni and colleagues' (2010) study, we expected that individuals with insomnia would find the sleep-related images to be more unpleasant than healthy sleepers. Unlike for the neutral or negative images, the participants with insomnia in our study specifically rated the sleep-related images as more negative than good sleepers. In Jansson-Fröjmark and colleagues' (2013) study, they found that individuals with primary insomnia provided higher valence and arousal ratings for threatening insomnia-related images. Thus, this indicates that individuals with insomnia may be particularly sensitive to sleep images, especially those that are negative.

Effects of CBTi on Valence Ratings

To our knowledge, this study is the first to evaluate the effects of CBTi on subjective ratings of images that were neutral, negative, or sleep-related in adults with chronic primary insomnia. Even though two sets of images were used in each version of the emotion reactivity task and the order was counterbalanced, participants with insomnia rated neutral and negative images as more negative at V1 than at V2. However, the valence ratings of sleep images did not differ between the two insomnia groups nor across time. Although these sleep images were categorized as negative in nature, the valence ratings for these images did not decrease over time just like the ratings for negative and neutral images. These results may demonstrate that there is a habituation effect only for non-sleep stimuli. But, individuals with insomnia may still perceive sleep stimuli as aversive, despite reporting improvements in sleep following the intervention. In a meta-analysis, Thakral et al. (2020) reported that CBTi reduced maladaptive beliefs about sleep in individuals with insomnia. Even though it is unclear if our participants with insomnia initially had dysfunctional beliefs about sleep, Okajima and colleagues (2014) previously stated that a decline

in dysfunctional beliefs and attitudes about sleep following CBTi did not mediate improvements in insomnia symptoms. This could mean that participants with insomnia may still perceive sleep stimuli as negative or may still be sensitive to sleep stimuli, regardless of benefitting from CBTi.

Brain Activity to Emotional Pictures

While the previously mentioned studies mainly focused on subjective responses to emotional images, we further explored subjective responses and brain responses to emotional images in adults with insomnia and good sleepers. Based on Baglioni and colleagues' (2014) study, we hypothesized that participants with insomnia would have enhanced amygdala activity when exposed to sleep images than healthy sleepers. Contrary to our hypothesis as well as the results from Baglioni and colleagues' (2014) study, both groups in our study did not have any activation in the left or right amygdala in response to negative or sleep images relative to baseline. Additionally, activity within the whole brain in response to sleep relative to neutral images did not differ between participants with insomnia and good sleepers at baseline. This may not be surprising because participants with insomnia and good sleepers rated that negative pictures were the most negative rather than sleep images. Although our study and Baglioni et al.'s (2014) study utilized sleep and neutral images with relatively moderate arousal levels, we may have only been able to detect elevated amygdala activity with images with higher arousal levels, especially with such a small sample size. In Kim and colleagues' (2017) study, participants with insomnia did have greater activation in the left and right precentral gyri, left prefrontal cortex, left fusiform gyrus, and left and right posterior cingulate gyri in response to sleep pictures than good sleepers. But, no differences in amygdala activity were observed between the groups. These inconsistent results can be attributed to the images that they incorporated into their task, which did not have any emotional or facial expressions.

In our study, we also did not detect any differences in amygdala activity in response to negative relative to neutral images between those with insomnia and good sleepers. However, participants with insomnia initially had decreased activation in the left superior temporal gyrus and right lentiform nucleus when they encountered negative images relative to neutral images compared to good sleepers. Huang and colleagues (2012) found that, in comparison to good sleepers, individuals with primary insomnia had lower functional connectivity during resting-state between the left amygdala and the left superior temporal gyrus and right lentiform nucleus. Additionally, participants with insomnia exhibited lower functional connectivity at rest between the right amygdala and the left superior temporal gyrus. Given that the coordination between the amygdala and associated brain areas is involved in emotional processing, these results may demonstrate that participants with insomnia have altered neural circuits that are specifically linked to emotions (Huang et al., 2012; Phillips et al., 2003). In Cerullo and colleagues' (2014) study, participants with bipolar I disorder had greater activation in the right lentiform nucleus while viewing emotional images compared to healthy controls. Zou et al. (2021) also found that, compared with good sleepers, patients with insomnia had reduced functional connectivity between the thalamus and the following subcortical structures: hippocampus, amygdala, parahippocampal gyrus, and lentiform nucleus. Altogether, these results may provide further support that activity within the limbic system is altered in individuals with insomnia.

Furthermore, our participants with insomnia had decreased activation in the right cuneus and bilateral middle occipital gyri when they viewed negative images in contrast to neutral images compared to good sleepers. The cuneus is a brain region that is a part of the visual network (Marques et al., 2017). The middle occipital gyrus is also thought to be involved in processing visual information (Teng et al., 2018). Huang et al. (2012) showed that participants with insomnia

had higher functional connectivity at rest between the right amygdala and right cuneus and left middle occipital gyrus than good sleepers. Additionally, in Cerullo et al.'s (2014) study, participants with bipolar I disorder had lower activity in the bilateral cuneus and bilateral middle occipital gyri in response to emotional images than healthy controls, indicating that visual processing of emotional stimuli may be disrupted in this clinical sample. Although participants with insomnia and good sleepers did not rate negative images differently from one another, these findings provide support for functional brain alterations pertaining to visual processing of emotional pictures among participants with insomnia.

Moreover, good sleepers had greater activity within the left middle frontal gyrus, left superior frontal gyrus, right inferior frontal gyrus, and bilateral supramarginal gyri when exposed to negative compared to neutral images than participants with insomnia. In a study conducted by Naor et al. (2020), healthy adults had greater activation in the left middle frontal gyrus, left superior frontal gyrus, left supramarginal gyrus, and bilateral inferior frontal gyri while viewing, in an empathic manner, pictures depicting painful situations relative to pictures depicting non-painful situations. On the contrary, they had greater activation in the bilateral supramarginal gyri and inferior frontal gyri while regulating their emotions toward these pictures using the reappraisal strategy. In particular, the inferior frontal gyrus is a region within the central executive network. This network is involved in maintaining and manipulating information in working memory as well as in decision-making or problem solving in a goal-driven manner (Menon & Uddin, 2010). Overall, these results can hint that these individuals with insomnia exhibited functional brain alterations linked with cognitive functioning or emotions in response to negative stimuli.

Effects of CBTi on Brain Activity

We hypothesized that, after CBTi, participants in the TX group would have lower amygdala activity while rating sleep-related images during the emotion reactivity task relative to the participants in the WL group. Although this hypothesis was not supported, we found that the TX group had reduced activity in the left superior temporal gyrus in response to sleep images following CBTi. The superior temporal gyrus is a brain region that plays a role in numerous functions, such as auditory processing, language, and social cognition (Bigler et al., 2007). Two components of social cognition are theory of mind and emotion understanding. The former refers to a person's ability to ascribe mental states, such as beliefs, desires, or intentions, to themselves or to others. The latter involves a person's ability to comprehend or predict their own emotions or the emotions of others (Bulgarelli & Molina, 2016). This heightened activity in the superior temporal gyrus at baseline could mean that the participants with insomnia initially were more capable of relating to the sleep-related images (e.g., images of individuals struggling to sleep or appearing restless in the morning) because they also had sleep difficulties at baseline. Indeed, participants in the TX group had greater number of awakenings throughout the night and slightly greater insomnia severity than those in the WL group. However, after receiving CBTi, activity in this region may have decreased only in the TX group because these participants were no longer able to relate as much to these sleep-related images, especially after reporting improvements in their sleep. Given that participants in the WL group still had sleep difficulties over time, activity in this region could have been amplified because these participants were still able to relate to the situations that were portrayed in the sleep pictures. In a study by Drummond et al. (2001), they found that better performance on a free recall memory task was associated with greater activity in the left superior temporal gyrus following sleep deprivation. They suggested that brain activity

was heightened to help individuals acquire more cognitive resources; thus, their performance was not as impaired during the cognitive task. With improvements in subjective sleep measures in the TX group following CBTi, this may also indicate that activity in the superior temporal gyrus was reduced in the TX group because these participants did not require as many cognitive resources during the emotion task as those who were still awaiting treatment. Taken together, these findings suggest that reduced activity within the left superior temporal gyrus following CBTi may be associated with improvements in cognitive functioning or reactivity to sleep-related stimuli.

Although activity in the left insula increased in response to sleep images over time for the WL group, activity in this region decreased among participants in the TX group after receiving CBTi. Guadagni et al. (2018) reported that young, healthy individuals had greater activity in the right insula, rather than the left insula, and the right amygdala while viewing sleep images in contrast to scrambled images. In addition, Kim et al. (2017) indicated that activity in the right insula declined following CBTi in response to sleep images in adults with insomnia. In a review, Fan et al. (2011) suggested that the right anterior insula (right Brodmann areas 13/47) played a role in the affective-perceptual form of empathy, which means that empathy can automatically be induced without individuals explicitly knowing the objective of a study (Blakemore et al., 2005, as cited in Fan et al., 2011). On the contrary, the left anterior insula (left Brodmann area 13) was implicated in the affective-perceptual and cognitive-evaluative forms of empathy (Fan et al., 2011). The cognitive-evaluative form of empathy means that empathy is affected by an individual using their imagination or by assessing his or her feelings in response to a target (Fan & Han, 2008, as cited in Fan et al., 2011). Overall, these findings could signify that the feeling of empathy was less prevalent in participants in the TX group after CBTi while they evaluated the valence of sleep images. Once again, perhaps the ability of these participants to understand or relate to feelings or

emotions that were expressed in the negative, sleep-related images may be reduced. On the other hand, participants in the WL group, who were waiting for treatment and still had sleep disturbances, may be more capable of experiencing feelings of empathy toward these sleep-related images, and this may be reflected by greater activation in the left insula.

Neural Predictors of Treatment Response

We postulated that lower amygdala activity, while rating sleep-related pictures during the emotion reactivity task at baseline, would predict better subjective sleep outcomes among individuals with insomnia. Given that there was no activation within the amygdala in response to these images, we could not assess if amygdala activity at baseline predicted changes in total ISI score or in any of the sleep diary measures. But, Rubin-Falcone et al. (2020) previously investigated if neural correlates of emotion reactivity and emotion regulation predicted treatment response to cognitive-behavioural therapy for depression in adults with major depressive disorder. In line with the findings from our study, activity within the amygdala did not predict treatment response in these adults with major depressive disorder. As no differences in amygdala activity in response to sleep relative to neutral images were identified between those with insomnia and healthy sleepers in our study, these results were as expected. Nevertheless, it is plausible that activity in other brain regions during the emotion reactivity task could be predictors of CBTi treatment response.

When comparing the two insomnia groups, activity in the left angular gyrus and left middle frontal gyrus (Brodmann areas 39 and 6, respectively) in response to sleep relative to neutral images at baseline did not predict differential changes in total ISI scores or in the sleep diary measures either. Evidently, the left angular gyrus was proposed to be involved in numerous functions, including semantic and number processing, reading and comprehension, spatial and

social cognition, and memory retrieval (Seghier, 2013). Given its role in social cognition, elevated activity in the left angular gyrus in response to sleep vs. neutral images at baseline could highlight that the participants with insomnia were able to empathize with the scenarios that were illustrated in the sleep-related images because they also had sleep disturbances at the start of the study. However, activity in this brain region did not seem to predict improvements in subjective sleep measures.

Brodmann area 6 consists of the premotor cortex and supplementary motor area. The former is involved in preparing for as well as engaging in a sequence of movements, whereas the latter deals with planning actions that are self-initiated mostly in the contralateral side of the body (Johns, 2014). Even though we did not examine reaction time during the emotion reactivity task, perhaps the participants with insomnia did not have to think for too long about how they would rate the sleep-related images compared with neutral images and were preparing to respond. To our knowledge, no study has focused on activity within this region or the angular gyrus to predict response to CBTi in adults with chronic primary insomnia. Nevertheless, Gallagher Thompson et al. (2015) were interested in examining if brain activity during an executive function task was predictive of response to CBT in older patients who were depressed. Although the left and right middle frontal gyri were two of their regions of interest, only greater activation within the right middle frontal gyrus at baseline was a predictor of improvements in depressive symptoms in these individuals. Based on a previous study, they stated that elevated activity in this region may be associated with better sustained attention and inhibitory control. Even though elevated activity in the right middle frontal gyrus at baseline during the emotion reactivity task did not predict improvements in sleep, this finding may indicate that sustained attention was enhanced at baseline

when participants with insomnia were exposed to sleep images. But, activation within this brain region did not appear to be indicative of better sleep following CBTi as well.

Limitations

This study has some potential limitations. For instance, the overall sample size as well as the number of participants in each group was quite small for an imaging study, even if we almost reached our target sample size of 10 participant per group. Thus, lack of power may explain why we did not observe significant differences, especially for analyses involving the fMRI data. Another potential limitation is that the good sleepers were only assessed once. Therefore, we could not make comparisons between the groups at V2. Another potential issue is that the arousal levels for the three types of images that were used in both versions of the emotion reactivity task, on average, were moderate. However, in Baglioni and colleagues' (2014) study, they selected images with high, moderate, and low arousal levels. If we compared neutral, negative, and sleep images with high arousal levels, we may have attained more promising results and may have seen activation within the amygdala. As previously mentioned, we also did not assess or account for dysfunctional beliefs about sleep. However, given our small sample size, we intentionally restricted the number of covariates that we included in our statistical analyses.

Strengths

Despite these limitations, this study also has some strengths. Participants were matched, based on age, sex, and education level. We were able to compare participants with insomnia and healthy sleepers at baseline. Although the good sleepers were only assessed once, we did have a wait-list control group who also served as an appropriate comparison at V2. Additionally, randomization of participants with insomnia was employed. In previous studies, researchers mostly assessed either subjective ratings or objective responses toward emotional stimuli instead

of both within a single study. As both were measured in our study, we had a more comprehensive understanding about how responses varied across groups or time. Another strength of this study is that individualized CBTi was employed rather than CBTi in a group setting. Yamadera and colleagues (2013) found that both individualized and group CBTi were beneficial for sleep. However, individualized CBTi yielded better subjective and objective sleep outcomes than group CBTi in individuals with chronic primary insomnia.

Future Directions

There are several future directions. Although some studies have incorporated negative, neutral, and positive stimuli, future studies should also include positive stimuli that are both related and unrelated to sleep in the emotion reactivity task to have a more global and better understanding of the influence of CBTi on emotion reactivity in individuals with chronic primary insomnia (Motomura et al., 2021; Tempesta et al., 2019). Similar to Baglioni and colleagues' (2014) study, it would be interesting to examine habituation effects for ratings and brain responses, considering that images were only rated and presented once throughout the study. As we did not find statistically significant differences in amygdala activity between groups during the emotion reactivity task, we will continue the study until we reach the sample size of the larger research project. We will then see if we obtain the same results within the whole brain, including the amygdala, as this current study. Future studies should also explore how individuals respond to emotional images physiologically (e.g., heart rate, blood pressure, or galvanic skin responses). In a previous study, researchers used videos rather than images (Sanz-Arigita et al., 2021). Therefore, it would be interesting to see how participants with insomnia and good sleepers respond to dynamic emotional stimuli, such as video clips, rather than static images because they may elicit more activation or be more salient. In Kim and colleagues' (2021) study, insomnia participants with

subjective-objective discrepancy of sleep had greater activity in the left fusiform, bilateral precuneus, right superior frontal gyrus, genu of the corpus callosum, and bilateral anterior corona radiata while viewing insomnia-related images relative to neutral images than those who did not have subjective-objective discrepancy of sleep. Thus, it would be interesting to assess if there is also a discrepancy between subjective ratings of images and objective brain activity in response to these emotional images prior to and following CBTi. These results would help us better understand the mechanism of CBTi for sleep and emotions from both a subjective and objective perspective. Given that CBTi did not seem to impact negative and positive affect in participants with chronic primary insomnia, future studies should evaluate if CBTi would be beneficial for improving sleep and emotions in individuals who suffer from both insomnia and mood disorders, such as major depressive disorder.

Conclusion

In conclusion, our results contribute to the body of knowledge about emotion reactivity, especially toward sleep stimuli, in chronic primary insomnia. Even though participants with insomnia rated sleep images as more negative than neutral images, there were no differences in amygdala activity between participants with insomnia and good sleepers in response to these sleep images. However, differences in activity within other brain regions at baseline were found between participants with insomnia and good sleepers specifically in response to negative images. Activity in the left superior temporal gyrus and left insula decreased following CBTi in the immediate treatment group, which may reflect improvements in cognitive functioning and emotions in response to sleep images. Lastly, activity within the left angular gyrus, and left middle frontal gyrus at baseline did not predict CBTi treatment response in individuals with insomnia.

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Appendix A: Valence, Arousal, and Sleep Ratings for Sleep-Related Images Obtained from the Amazon Mechanical Turk Survey

Table A1

Valence, arousal, and sleep ratings for sleep-related images in each version of the emotion reactivity task in participants with insomnia and good sleepers

Rating Type	Version 1				Version 2			
	INS		Good Sleepers		INS		Good Sleepers	
	M	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
Valence	2.60	0.74	3.10	0.76	2.80	0.61	3.40	0.57
Arousal	6.70	0.67	5.90	0.75	6.60	0.53	5.80	0.40
Sleep	6.90	0.52	6.50	0.59	6.80	0.45	6.20	0.53

Note. INS: participants with insomnia; Data are from good sleepers and participants with insomnia who completed the survey on the Amazon Mechanical Turk platform

Appendix B: Cronbach's Alpha Coefficients

Table B1

Cronbach's alpha coefficients for the PANAS and ISI scores across time

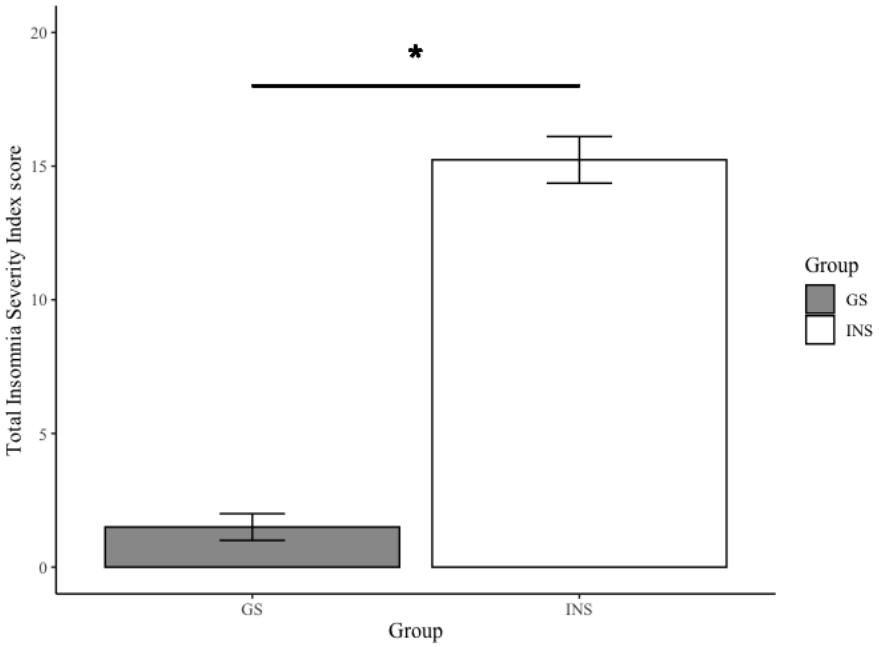
Visit	Measure	All participants ($N = 26$)	Insomnia ($N = 18$)	Good Sleepers ($N = 8$)	TX ($N = 9$)	WL ($N = 9$)
V0						
	PANAS – PA	.89	.91	.87	.84	.89
	PANAS – NA	.79	.82	.19	.59	.88
V1						
	ISI	.93	.66	.56	.66	.46
V2						
	PANAS – PA	—	.95	—	.92	.93
	PANAS – NA	—	.93	—	.60	.96
	ISI	—	.91	—	.77	.78

Note. The data are Cronbach's alpha coefficients. PA: positive affect; NA: negative affect

Appendix C: Subjective Sleep Data Between Groups

Figure C1

Differences in total ISI scores between good sleepers and participants with insomnia at V1



Note. *: $p < .001$. GS: good sleeper group; INS: insomnia group

Table C1

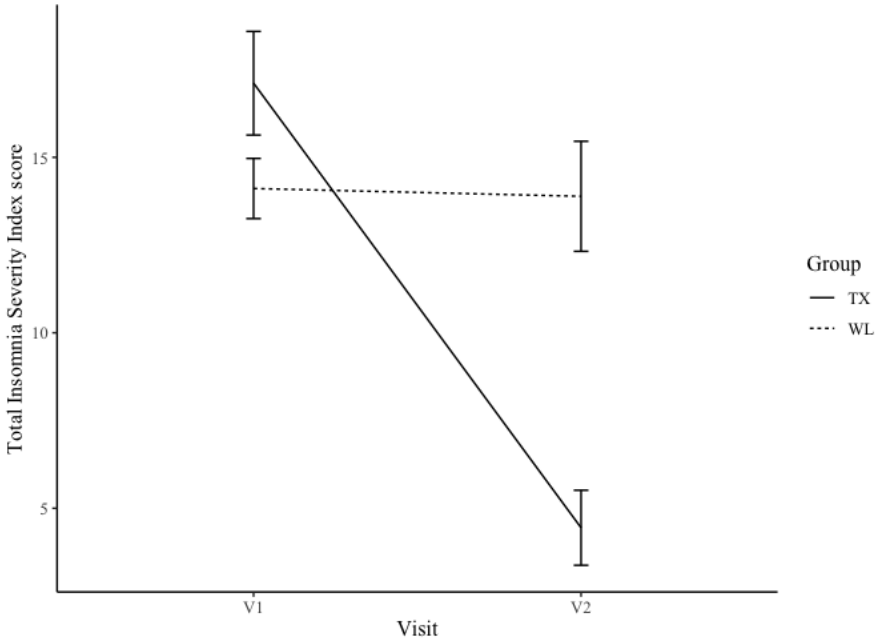
Analysis of Variance Results for the Total Insomnia Severity Index Score

Source	*df*	$df_{residual}$	*F*	*p*
Group	1	16	5.14	.04
Visit	1	16	30.30	< .001
Group*Visit	1	16	20.13	< .001

Note. ANOVA type III test

Figure C2

Total ISI scores between the insomnia groups at V1 and V2



Note. Error bars represent standard error of the mean; TX: immediate treatment group; WL: wait-list control group

Table C2

Average subjective sleep measures for participants with insomnia and good sleepers after V0

Sleep Measure	Insomnia ($N = 18$)	Good Sleepers ($N = 8$)	p
SOL (minutes)	32.12 (20.25)	16.10 (13.62)	.03
WASO (#)	2.13 (1.47)	0.59 (0.39)	< .001
WASO (minutes)	33.30 (23.29)	8.04 (5.53)	< .001
TST (hours)	6.52 (0.99)	7.43 (0.66)	.01
SE (%)	80.02 (7.85)	89.39 (5.45)	.01

Note. Data are mean (*SD*). SOL: sleep onset latency; WASO: wake after sleep onset; TST: total sleep time; SE: sleep efficiency

Table C3

Average subjective sleep measures for the immediate treatment and wait-list groups at V1 and V2

Sleep Measure	TX ($N = 9$)		WL ($N = 9$)	
	V1	V2	V1	V2
SOL (minutes)	35.26 (27.64)	19.83 (16.68)	23.84 (15.00)	21.82 (10.70)
WASO (#)	2.57 (1.17)	1.76 (0.96)	1.34 (1.19)	1.87 (1.55)
WASO (minutes)	55.46 (36.18)	22.96 (17.47)	38.05 (37.80)	28.99 (21.83)
TST (hours)	6.65 (1.16)	7.15 (0.99)	6.54 (0.53)	6.37 (0.38)
SE (%)	78.60 (9.44)	87.99 (6.29)	81.25 (10.85)	81.19 (5.68)

Note. Data are mean (*SD*). SOL: sleep onset latency; WASO: wake after sleep onset; TST: total sleep time; SE: sleep efficiency

Table C4

Analysis of Variance Results for Sleep Onset Latency

Source	*df*	$df_{residual}$	*F*	*p*
Group	1	16	0.01	.90
Visit	1	16	4.58	.05
Group*Visit	1	16	3.82	.07

Note. ANOVA type III test

Table C5

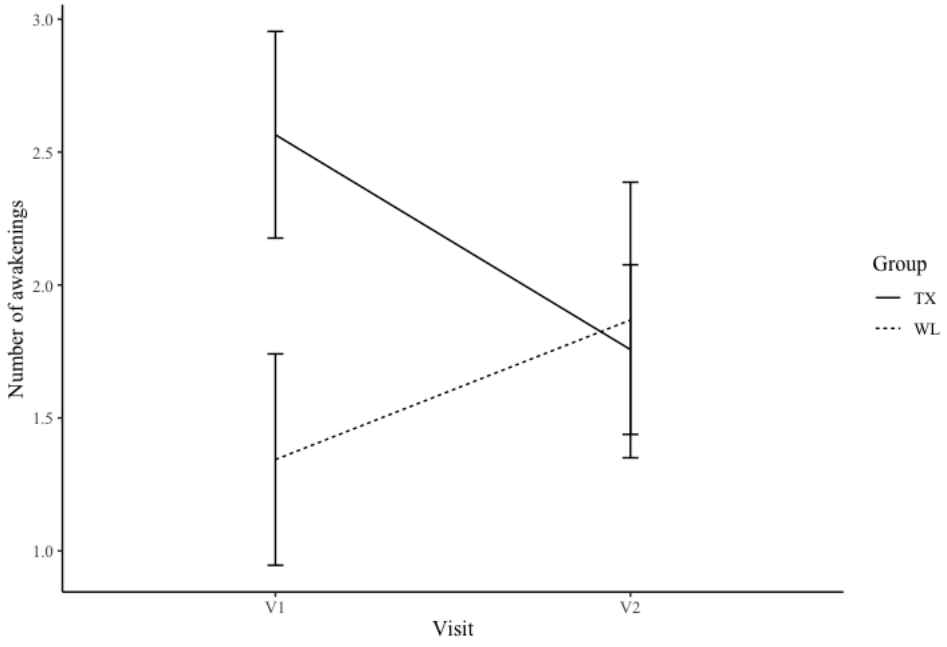
Analysis of Variance Results for Number of Awakenings After Sleep Onset

Source	*df*	$df_{residual}$	*F*	*p*
Group	1	16	2.04	.17
Visit	1	16	0.39	.54
Group*Visit	1	16	6.85	.02

Note. ANOVA type III test

Figure C3

Number of awakenings between the insomnia groups at V1 and V2



Note. Error bars represent standard error of the mean; TX: immediate treatment group; WL: wait-list control group

Table C6

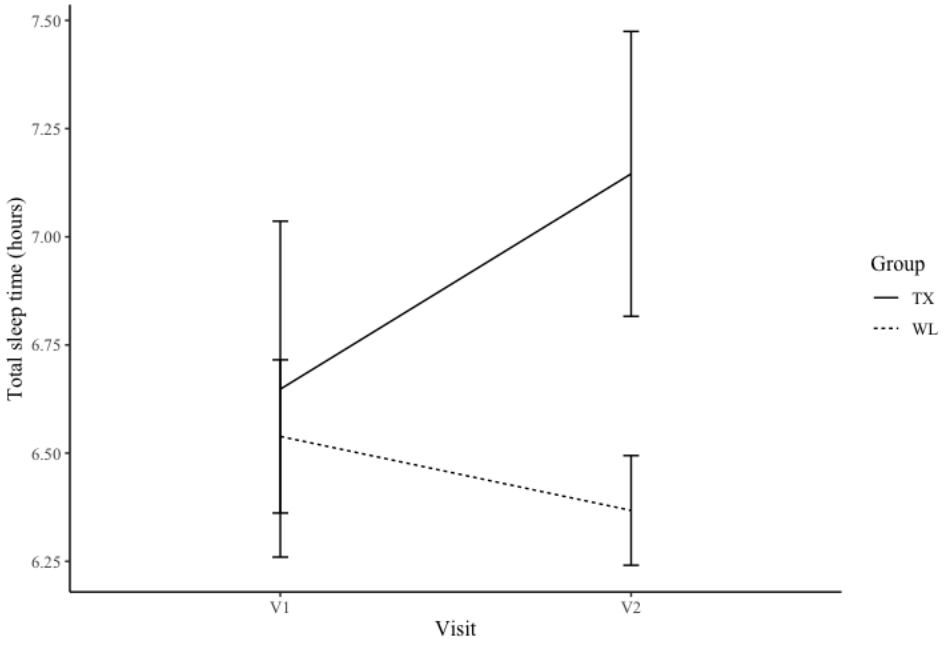
Analysis of Variance Results for Duration of Awakenings

Source	*df*	$df_{residual}$	*F*	*p*	η^2
Group	1	16	0.24	.63	0.01
Visit	1	16	7.20	.02	0.12
Group*Visit	1	16	2.29	.15	0.04

Note. ANOVA type III test

Figure C4

Total sleep time between the insomnia groups at V1 and V2



Note. Error bars represent standard error of the mean; TX: immediate treatment group; WL: wait-list control group; * $p < .05$

Table C7

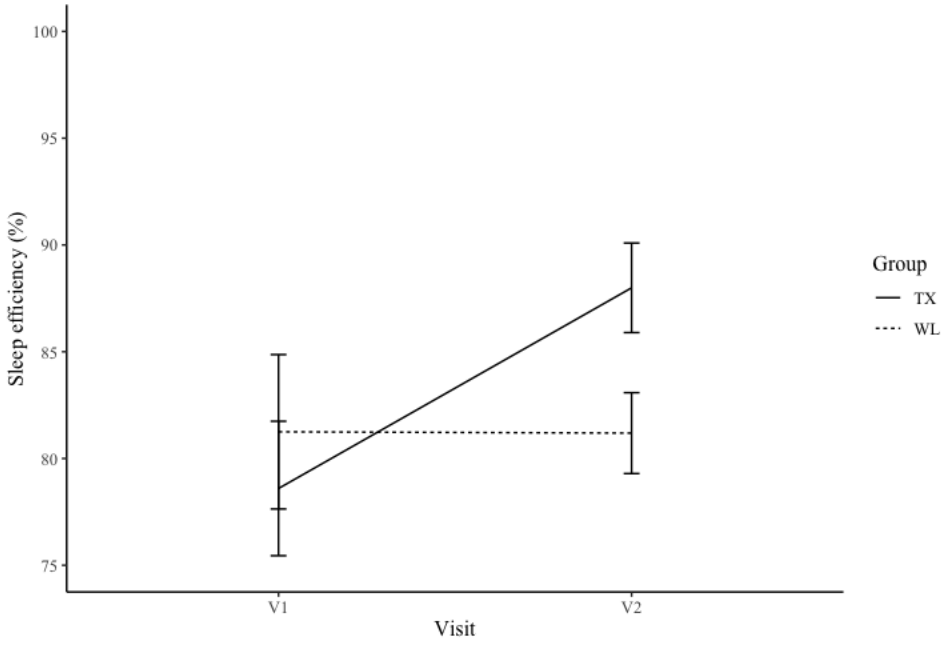
Analysis of Variance Results for Total Sleep Time

Source	*df*	$df_{residual}$	*F*	*p*	η^2
Group	1	16	1.41	.25	0.07
Visit	1	16	1.97	.18	0.01
Group*Visit	1	16	8.26	.01	0.04

Note. ANOVA type III test

Figure C5

Sleep efficiency between the insomnia groups at V1 and V2



Note. Error bars represent standard error of the mean; TX: immediate treatment group; WL: wait-list control group

Table C8

Analysis of Variance Results for Sleep Efficiency

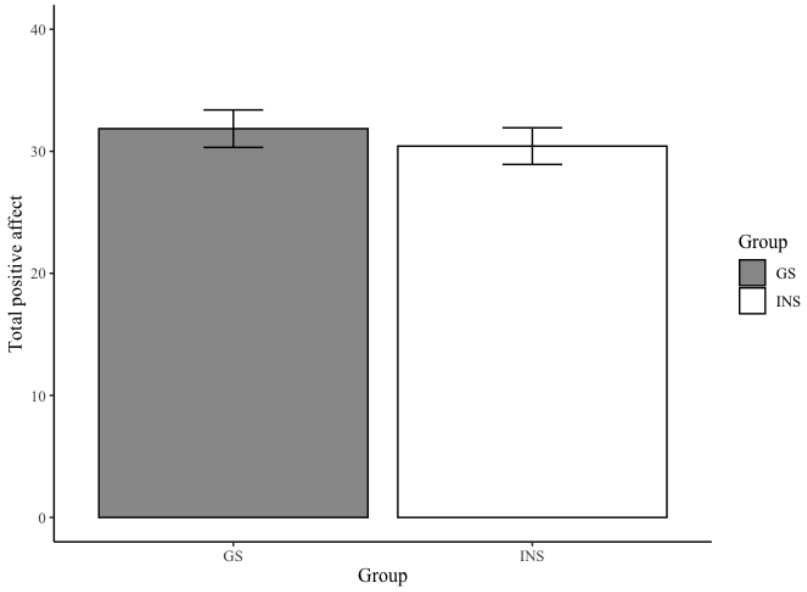
Source	*df*	$df_{residual}$	*F*	*p*	η^2
Group	1	16	0.39	.54	0.02
Visit	1	16	5.03	.04	0.08
Group*Visit	1	16	5.16	.04	0.08

Note. ANOVA type III test

Appendix D: Positive and Negative Affect Data

Figure D1

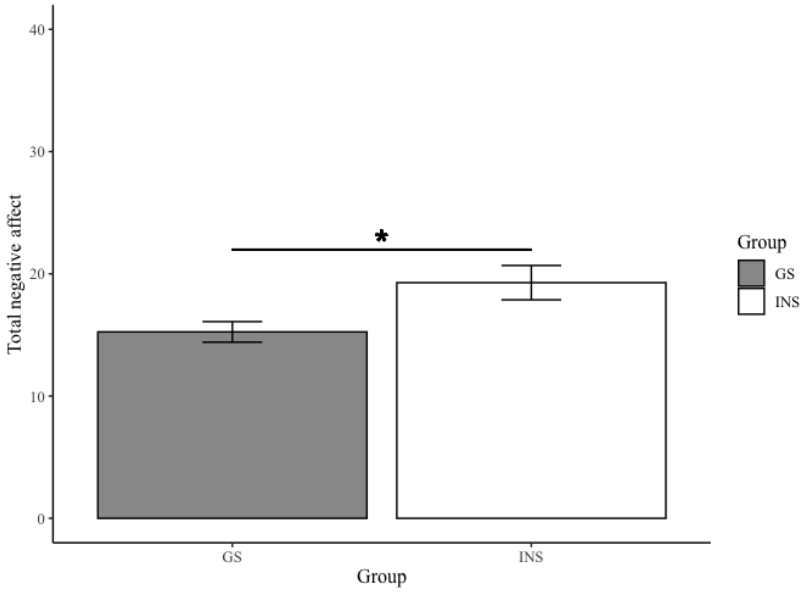
No differences in total positive affect between good sleepers and participants with insomnia at V0



Note. GS: good sleeper group; INS: insomnia group

Figure D2

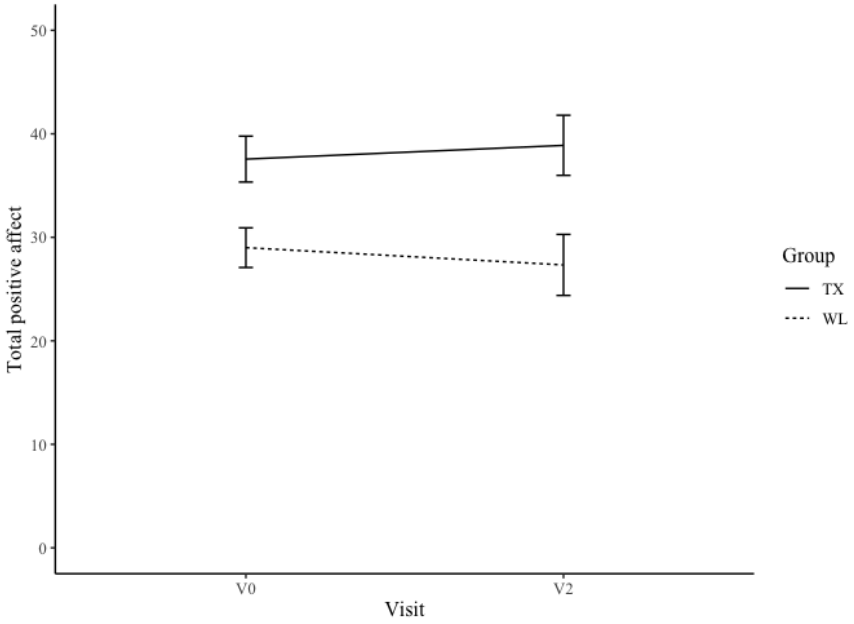
Differences in total negative affect between good sleepers and participants with insomnia at V0



Note. *: $p = .02$. GS: good sleeper group; INS: insomnia group

Figure D3

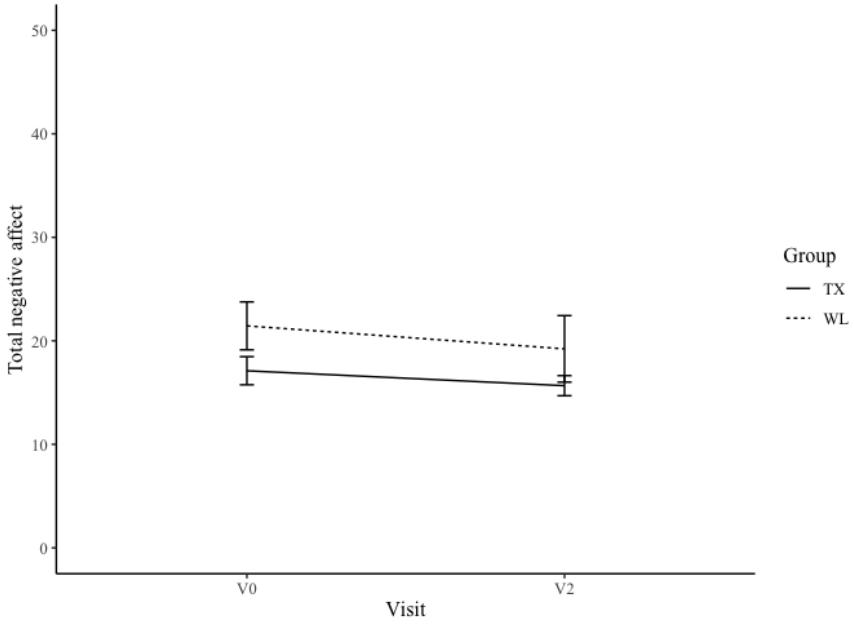
Total positive affect between insomnia groups at V1 and V2



Note. Error bars represent standard error of the mean; TX: immediate treatment group; WL: wait-list control group

Figure D4

Total negative affect between insomnia groups at V1 and V2



Note. Error bars represent standard error of the mean; TX: immediate treatment group; WL: wait-list control group

Table D1

Analysis of Variance Results for PANAS – Positive Affect

Source	*df*	$df_{residual}$	*F*	*p*	η^2
Group	1	16	9.92	.01	0.33
Visit	1	16	0.01	.92	0.00
Group*Visit	1	16	0.85	.37	0.01

Note. ANOVA type III test

Table D2

Analysis of Variance Results for PANAS – Negative Affect

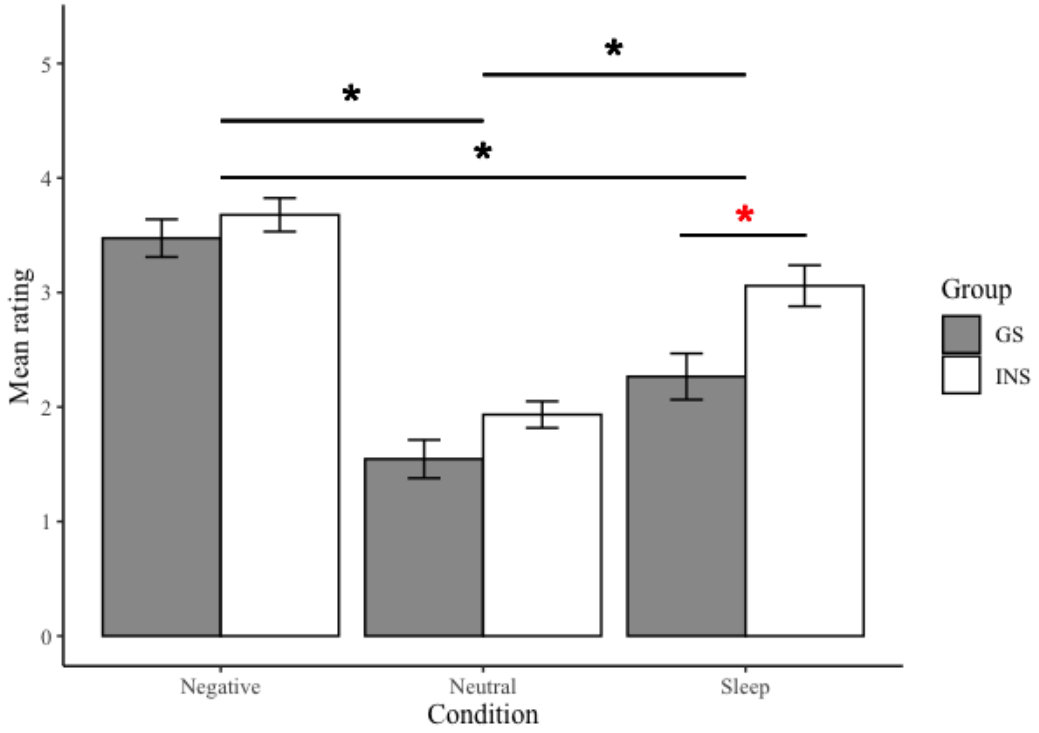
Source	*df*	$df_{residual}$	*F*	*p*
Group	1	16	0.89	.36
Visit	1	16	4.15	.06
Group*Visit	1	16	1.32	.27

Note. ANOVA type III test

Appendix E: Valence Ratings Data

Figure E1

Differences in valence ratings of images between good sleepers and participants with insomnia at V1



Note. * $p < .05$ for the effect of condition; * $p = .01$; GS: good sleeper group; INS: insomnia group

Table E1

Two-way repeated measures ANOVA results for valence ratings at V1

Source	*df*	$df_{residual}$	*F*	*p*	η^2
Group	1	24	5.06	.03	0.12
Condition	1.62	38.84	98.93	< .001	0.59
Group*Condition	1.62	38.84	2.66	.09	0.04

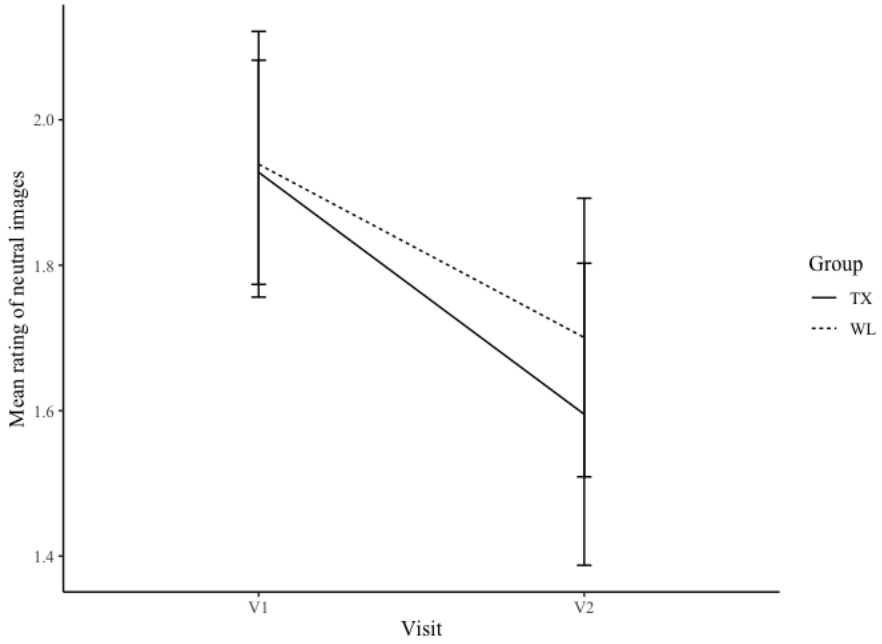
Table E2

Average valence ratings for participants with insomnia and good sleepers at V1

	Insomnia (n = 18)		Good sleepers (n = 8)	
Image Category	*M*	*SD*	*M*	*SD*
Neutral	1.93	0.49	1.55	0.47
Negative	3.68	0.62	3.47	0.47
Sleep	3.06	0.76	2.27	0.57

Figure E2

Mean rating of neutral images between the insomnia groups at V1 and V2



Note. Error bars represent standard error of the mean; TX: immediate treatment group; WL: wait-list control group

Table E3

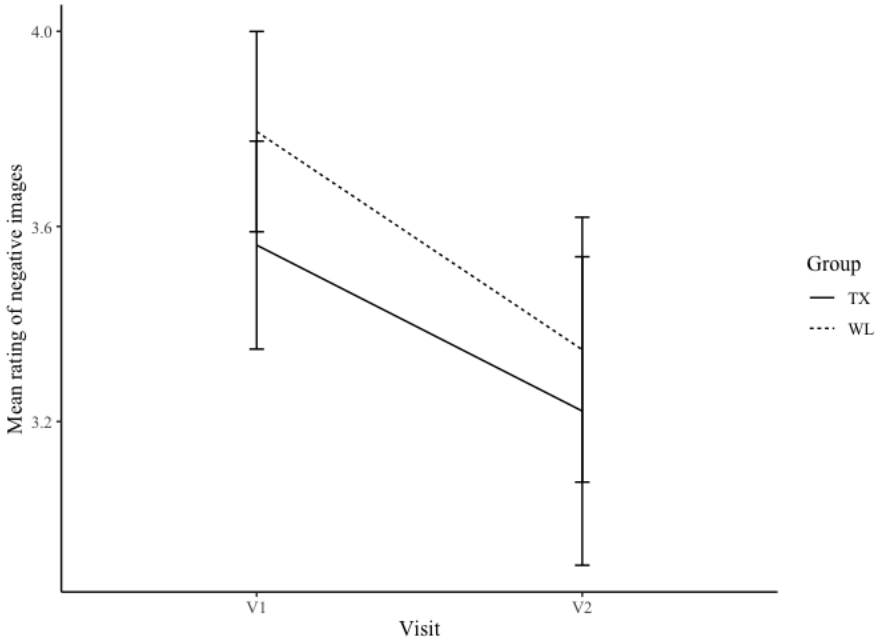
Analysis of Variance Results for Ratings of Neutral Images

Source	*df*	$df_{residual}$	*F*	*p*
Group	1	16	0.06	.81
Visit	1	16	8.12	.01
Group*Visit	1	16	0.28	.60

Note. ANOVA type III test

Figure E3

Mean rating of negative images between the insomnia groups at V1 and V2



Note. Error bars represent standard error of the mean; TX: immediate treatment group; WL: wait-list control group

Table E4

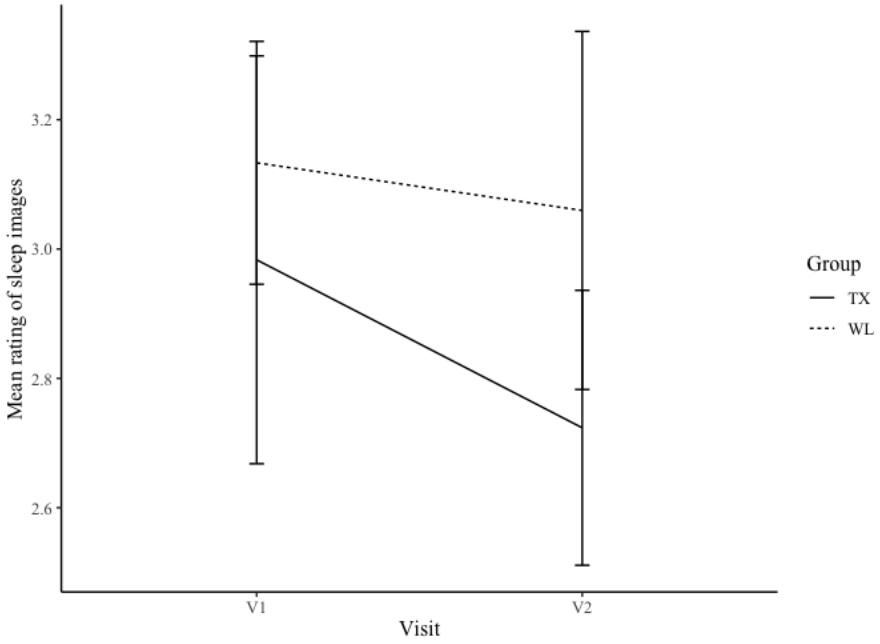
Analysis of Variance Results for Ratings of Negative Images

Source	*df*	$df_{residual}$	*F*	*p*	η^2
Group	1	16	0.29	.60	0.02
Visit	1	16	8.71	.01	0.07
Group*Visit	1	16	0.16	.69	0.00

Note. ANOVA type III test

Figure E4

Mean rating of sleep images between the insomnia groups at V1 and V2



Note. Error bars represent standard error of the mean; TX: immediate treatment group; WL: wait-list control group

Table E5

Analysis of Variance Results for Ratings of Sleep Images

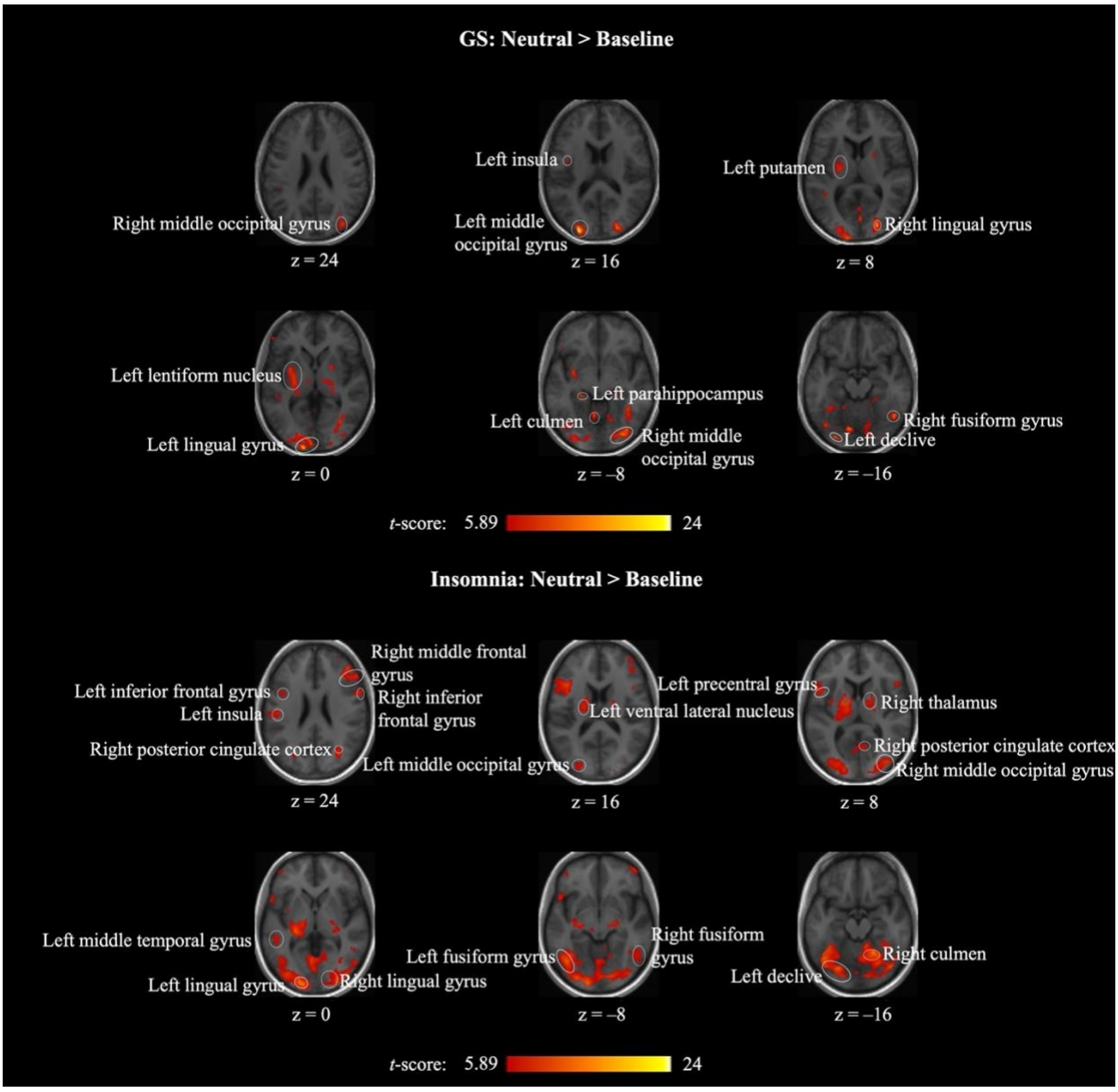
Source	*df*	$df_{residual}$	*F*	*p*	η^2
Group	1	16	0.53	.48	0.03
Visit	1	16	1.76	.20	0.01
Group*Visit	1	16	0.55	.47	0.00

Note. ANOVA type III test

Appendix F: Activated Brain Regions in Response to Images Relative to Baseline

Figure F1

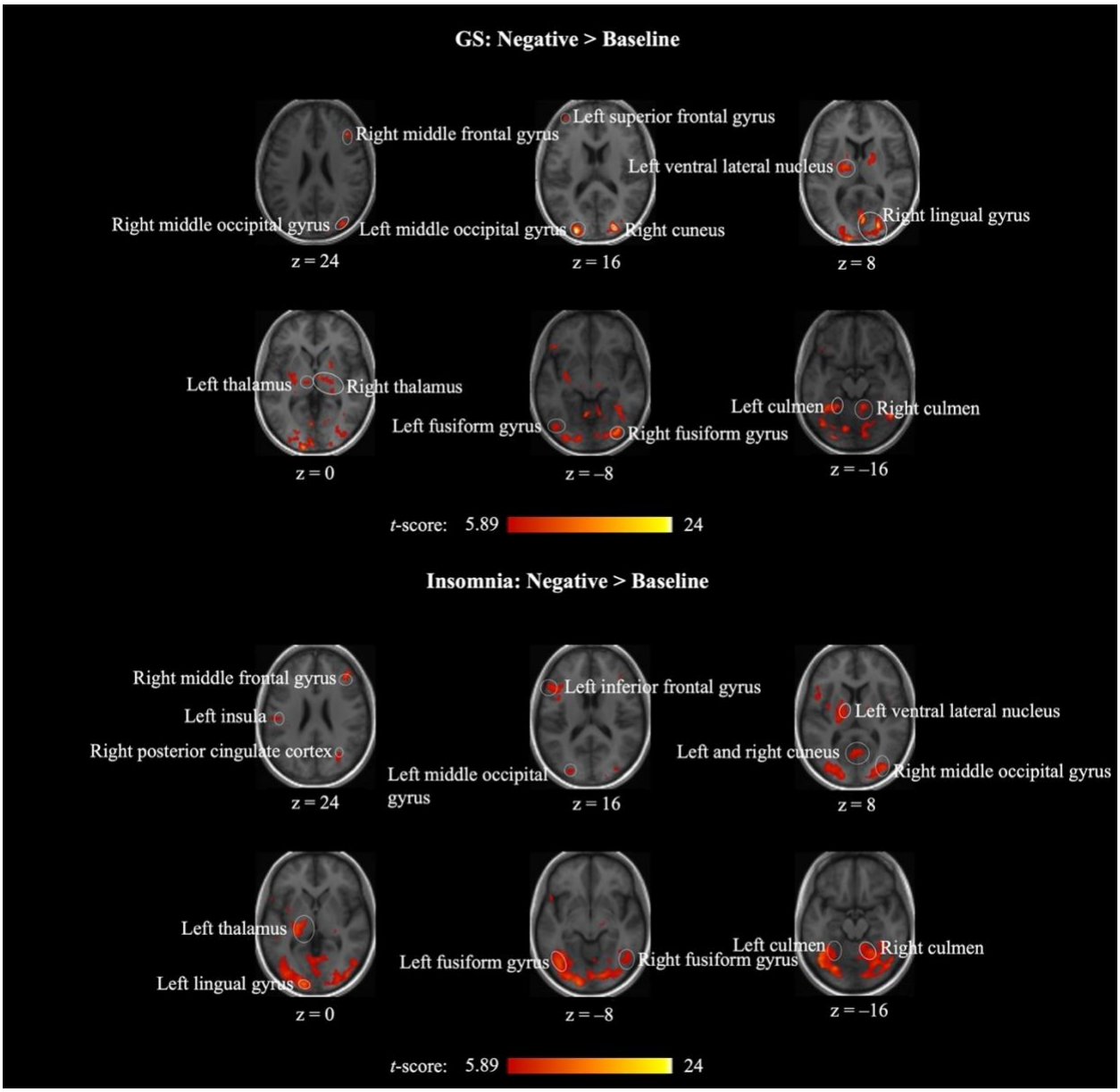
Activated brain regions in response to neutral images relative to baseline in good sleepers and participants with insomnia at V1



Note. GS: good sleepers; Top panel: Good sleepers; Bottom panel: Insomnia group (TX and WL)

Figure F2

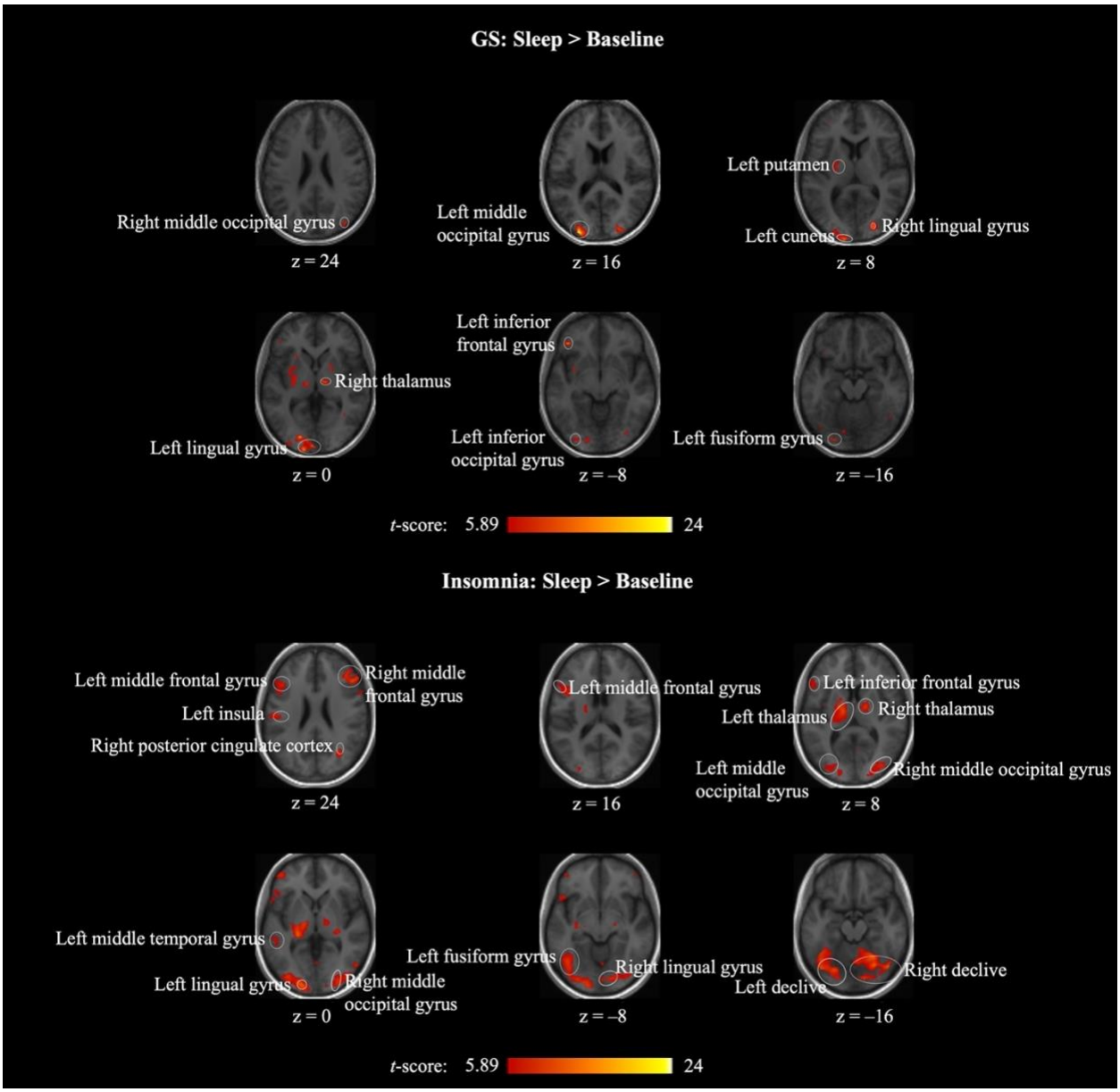
Activated brain regions in response to negative images relative to baseline in good sleepers and participants with insomnia at V1



Note. GS: good sleepers; Top panel: Good sleepers; Bottom panel: Insomnia group (TX and WL)

Figure F3

Activated brain regions in response to sleep images relative to baseline in good sleepers and participants with insomnia at V1



Note. GS: good sleepers; Top panel: Good sleepers; Bottom panel: Insomnia group (TX and WL)

Table F1

Clusters statistically significantly more activated in response to neutral images vs. baseline in good sleepers at V1

Brain region (BA)	Coordinates (x, y, z)	Cluster-level		Peak-level		
		Cluster size (voxels)	$p_{uncorrected}$	t	z_E	$p_{uncorrected}$
Left middle occipital gyrus (18)	–26, –94, 16	892	< .001	31.38	4.99	< .001
Left lingual gyrus (18)	–18, –102, 2			21.92	4.63	< .001
Left declive	–30, –88, –16			17.20	4.37	< .001
Right lingual gyrus (17)	24, –88, 10	1593	< .001	20.43	4.56	< .001
Right fusiform gyrus (37)	48, –60, –16			14.55	4.19	< .001
Right middle occipital gyrus (18)	30, –84, –8			13.72	4.13	< .001
Left middle frontal gyrus (9)	–50, 28, 32	30	.09	19.05	4.48	< .001
Left culmen	–30, –46, –22	276	< .001	16.78	4.35	< .001
Left culmen	–18, –46, –18			12.03	3.98	< .001
Left culmen	–26, –34, –22			9.09	3.64	< .001
Left parahippocampus (27)	–24, –34, –8	89	.01	14.40	4.18	< .001

Left lingual gyrus (18)	–2, –72, 4	83	.01	13.76	4.13	< .001
Right lingual gyrus (18)	2, –78, 8			8.93	3.62	< .001
Left inferior parietal lobule (40)	–48, –36, 50	176	< .001	13.45	4.10	< .001
Left inferior parietal lobule (40)	–46, –48, 56			9.75	3.73	< .001
Left supramarginal gyrus (40)	–48, –40, 36			7.59	3.42	< .001
Left superior parietal lobule (7)	–30, –58, 50	82	.01	12.68	4.04	< .001
Right cerebellar tonsil	24, –60, –38	236	< .001	12.40	4.01	< .001
Right inferior semi-lunar lobule	26, –68, –42			9.36	3.68	< .001
Right cerebellar tonsil	16, –58, –36			8.57	3.57	< .001
Left declive	–42, –72, –14	116	.003	11.68	3.94	< .001
Left declive	–30, –74, –14			6.45	3.21	.001
Left claustrum	–34, –4, –6	441	< .001	11.01	3.87	< .001
Left lentiform nucleus (putamen)	–26, –14, 4			9.82	3.74	< .001
Left culmen	–6, –60, –8	52	.03	10.81	3.85	< .001
Right lateral geniculum body	24, –24, –4	192	< .001	10.31	3.79	< .001
Right thalamus (pulvinar)	24, –32, 4			9.76	3.73	< .001

Right thalamus (ventral posteromedial nucleus)	18, –18, –2			8.34	3.54	< .001
Left middle frontal gyrus (9)	–24, 48, 34	7	.40	9.52	3.70	< .001
Right middle frontal gyrus (9)	58, 28, 28	5	.48	9.12	3.65	< .001
Right lentiform nucleus (putamen)	20, 2, 2	55	.03	9.01	3.63	< .001
Left lingual gyrus (18)	–18, –78, –2	11	.29	8.69	3.59	< .001
Brain stem	0, –36, –34	13	.25	8.55	3.57	< .001
Left inferior frontal gyrus (47)	–48, 30, –10	11	.29	8.45	3.55	< .001
Left inferior frontal gyrus (46)	–54, 44, –4	21	.15	8.29	3.53	< .001
Left inferior frontal gyrus (46)	–58, 38, 4			8.04	3.49	< .001
Right declive	40, –64, –24	19	.17	8.04	3.49	< .001
Left superior parietal lobule (7)	–32, –52, 64	28	.10	7.51	3.40	< .001
Left tuber	–40, –70, –28	23	.13	7.49	3.40	< .001
Left insula (13)	–40, –2, 16	6	.44	7.46	3.40	< .001
Left thalamus	–10, –14, 0	14	.24	7.28	3.37	< .001
Right lentiform nucleus (putamen)	32, 2, –4	12	.27	7.09	3.33	< .001

Left superior temporal gyrus (22)	–46, –38, 2	18	.18	6.98	3.31	< .001
Left superior temporal gyrus (39)	–42, –48, 8	5	.48	6.83	3.28	.001

Note. Coordinates represent peak voxel coordinates in millimeters; For clarity, only clusters with at least five voxels were listed in the table; BA = Brodmann area; Height threshold T: 5.89 ($p < .001$ uncorrected at the voxel level).

Table F2

Clusters statistically significantly more activated in response to neutral images vs. baseline in participants with insomnia at V1

Brain region (BA)	Coordinates (x, y, z)	Cluster-level		Peak-level		
		Cluster size (voxels)	$p_{uncorrected}$	t	z_E	$p_{uncorrected}$
Left culmen	–34, –60, –32	44881	< .001	15.55	6.44	< .001
Left inferior occipital gyrus (17)	–16, –96, –4			14.93	6.36	< .001
Left fusiform gyrus (37)	–42, –66, –12			14.92	6.35	< .001
Right middle frontal gyrus (9)	42, 40, 22	6913	< .001	11.21	5.72	< .001
Right inferior frontal gyrus (9)	58, 8, 26			9.83	5.41	< .001
Right middle frontal gyrus (9)	38, 34, 26			9.20	5.26	< .001
Left superior frontal gyrus (6)	–6, 6, 64	2885	< .001	8.63	5.10	< .001
Right superior frontal gyrus (8)	10, 24, 50			7.03	4.61	< .001
Left medial frontal gyrus (6)	–4, –4, 76			6.86	4.55	< .001
Left claustrum	–30, 22, –8	26	.24	4.23	3.38	< .001
Left superior parietal lobule (7)	–14, –44, 68	13	.40	4.20	3.36	< .001

Right inferior frontal gyrus (47)	46, 22, –10	5	.62	3.98	3.23	.001
Left culmen	–20, –36, –30	6	.58	3.95	3.22	.001

Note. Coordinates represent peak voxel coordinates in millimeters; For clarity, only clusters with at least five voxels were listed in the table; BA = Brodmann area; Height threshold T: 3.73 ($p < .001$ uncorrected at the voxel level).

Table F3

Clusters statistically significantly more activated in response to negative images vs. baseline in good sleepers at V1

Brain region (BA)	Coordinates (x, y, z)	Cluster-level		Peak-level		
		Cluster size (voxels)	$p_{uncorrected}$	t	z_E	$p_{uncorrected}$
Left middle occipital gyrus (18)	–26, –94, 16	2165	< .001	64.14	5.64	< .001
Right lingual gyrus (17)	24, –86, 8			35.35	5.10	< .001
Right cuneus (17)	24, –90, 16			27.68	4.86	< .001
Left inferior parietal lobule (40)	–46, –46, 56	383	< .001	50.06	5.42	< .001
Left inferior parietal lobule (40)	–50, –38, 48			30.96	4.97	< .001
Left inferior parietal lobule (39)	–30, –58, 46			12.45	4.02	< .001
Left lingual gyrus (18)	–4, –72, 2	190	< .001	22.41	4.65	< .001
Right lingual gyrus (18)	4, –82, 8			19.89	4.53	< .001
Left culmen	–6, –60, –8			13.18	4.08	< .001
Left culmen	–34, –50, 20	797	< .001	19.77	4.52	< .001
Left culmen	–26, –44, –24			15.64	4.27	< .001

Left declive	–42, –72, –14			15.51	4.26	< .001
Right middle frontal gyrus (9)	46, 26, 26	103	.002	17.65	4.40	< .001
Right middle frontal gyrus (9)	40, 32, 24			13.00	4.06	< .001
Right middle frontal gyrus (9)	48, 36, 30			10.62	3.83	< .001
Left inferior semi-lunar lobule	–4, –78, –40	64	.01	15.85	4.29	< .001
Right inferior semi-lunar lobule	6, –78, –42			12.99	4.06	< .001
Right uvula	8, –82, –32			10.76	3.85	< .001
Right thalamus (medial dorsal nucleus)	6, –10, 0	255	< .001	14.34	4.18	< .001
Right putamen	18, 6, 4			10.69	3.84	< .001
Right red nucleus	10, –22, –10			9.56	3.70	< .001
Right culmen	26, –40, –24	543	< .001	14.33	4.18	< .001
Right culmen	32, –46, –28			11.46	3.92	< .001
Right culmen	14, –46, –20			9.32	3.67	< .001
Left inferior frontal gyrus (47)	–46, 28, –12	43	.04	13.43	4.10	< .001
Right fusiform gyrus (37)	48, –60, –16	285	< .001	13.33	4.09	< .001

Right fusiform gyrus (37)	38, –48, –10			10.21	3.78	< .001
Right parahippocampus (19)	38, –60, –4			9.49	3.70	< .001
Left cerebellar tonsil	–26, –54, –42	24	.10	12.41	4.01	< .001
Left cerebellar tonsil	–18, –56, –44			6.76	3.27	.001
Left thalamus (ventral lateral nucleus)	–18, –8, 8	400	< .001	11.87	3.96	< .001
Left thalamus	–10, –14, 0			10.06	3.77	< .001
Left lentiform nucleus (lateral globus pallidus)	–26, –14, –2			8.31	3.53	< .001
Left superior frontal gyrus (10)	–40, 52, 14	23	.11	11.78	3.95	< .001
Left cerebellar tonsil	–18, –64, –40	47	.03	11.51	3.93	< .001
Left middle frontal gyrus (6)	–42, 4, 56	14	.20	10.21	3.78	< .001
Left precentral gyrus (6)	–40, 0, 40	20	.13	10.02	3.76	< .001
Right tuber	40, –64, –26	36	.05	9.54	3.70	< .001
Right lingual gyrus (18)	16, –68, 0	6	.41	8.65	3.58	< .001
Left precentral gyrus (9)	–46, 26, 36	22	.12	8.61	3.58	< .001

Left lentiform nucleus (putamen)	–16, 6, 6	10	.28	7.62	3.42	< .001
Right thalamus	22, –28, 0	32	.06	7.32	3.37	< .001
Left lingual gyrus (18)	–20, –78, –2	5	.45	7.13	3.34	< .001
Left parahippocampus (27)	–26, –30, –4	11	.26	7.12	3.34	< .001
Left fusiform gyrus (37)	–54, –68, –12	8	.34	6.98	3.31	< .001
Right thalamus	10, –30, –6	5	.45	6.98	3.31	< .001

Note. Coordinates represent peak voxel coordinates in millimeters; For clarity, only clusters with at least five voxels were listed in the table; BA = Brodmann area; Height threshold T: 5.89 ($p < .001$ uncorrected at the voxel level).

Table F4

Clusters statistically significantly more activated in response to negative images vs. baseline in participants with insomnia at V1

Brain region (BA)	Coordinates (x, y, z)	Cluster-level		Peak-level		
		Cluster size (voxels)	$p_{uncorrected}$	t	z_E	$p_{uncorrected}$
Left declive	–42, –68, –12	36637	< .001	15.12	6.38	< .001
Right culmen	36, –46, –22			15.08	6.38	< .001
Left fusiform gyrus (37)	–48, –62, –14			14.16	6.24	< .001
Right middle frontal gyrus (9)	40, 36, 22	4143	< .001	12.23	5.91	< .001
Right subthalamic nucleus	16, –16, –6			7.85	4.87	< .001
Right middle frontal gyrus (10)	40, 50, 18			6.44	4.39	< .001
Left superior frontal gyrus (6)	–6, 6, 66	1306	< .001	9.27	5.27	< .001
Left superior frontal gyrus (6)	–4, –2, 72			6.24	4.31	< .001
Right cingulate gyrus (32)	10, 24, 28			5.43	3.98	< .001
Left middle temporal gyrus (21)	–48, –32, –4	231	.003	7.09	4.63	< .001
Left middle temporal gyrus (22)	–52, –46, 2			6.01	4.22	< .001

Brain stem	–2, –26, –26	143	.01	6.59	4.45	< .001
Right middle frontal gyrus (10)	48, 52, –6	141	.01	5.32	3.93	< .001
Right middle frontal gyrus (10)	42, 54, 4			3.94	3.21	.001
Right inferior frontal gyrus (46)	54, 48, 4			3.91	3.20	.001
Left cingulate gyrus (24)	–12, 18, 30	37	.17	4.33	3.43	< .001
Left cingulate gyrus (32)	–8, 24, 24			4.13	3.32	< .001
Left medial frontal gyrus (6)	–8, –6, 50	33	.20	4.22	3.37	< .001
Left medial frontal gyrus (6)	–2, 10, 50	11	.46	3.92	3.20	.001
Left middle temporal gyrus (21)	–46, 2, –34	6	.59	3.91	3.20	.001

Note. Coordinates represent peak voxel coordinate in millimeters; For clarity, only clusters with at least five voxels were listed in the table; BA = Brodmann area; Height threshold T: 3.73 ($p < .001$ uncorrected at the voxel level).

Table F5

Clusters statistically significantly more activated in response to sleep images vs. baseline in good sleepers at V1

Brain region (BA)	Coordinates (x, y, z)	Cluster-level		Peak-level		
		Cluster size (voxels)	$p_{uncorrected}$	t	z_E	$p_{uncorrected}$
Left middle occipital gyrus (18)	–28, –96, 16	616	< .001	28.69	4.90	< .001
Left lingual gyrus (17)	–6, –96, –2			28.08	4.88	< .001
Left lingual gyrus (18)	–12, –90, –2			21.90	4.63	< .001
Left inferior frontal gyrus (47)	–42, 36, –8	46	.03	16.37	4.32	< .001
Right lingual gyrus (17)	24, –88, 10	157	< .001	15.54	4.26	< .001
Left cerebellar tonsil	–22, –54, –46	44	.04	15.37	4.25	< .001
Left cerebellar tonsil	–12, –60, –44			9.46	3.69	< .001
Left cerebellar tonsil	–22, –62, –40			6.86	3.29	.001
Right thalamus	12, –14, 2	34	.06	12.77	4.05	< .001
Left claustrum	–34, 4, –6	228	< .001	12.68	4.04	< .001

Left lentiform nucleus (putamen)	–24, –6, 2			8.54	3.57	< .001
Left lentiform nucleus (putamen)	–30, –18, –2			7.32	3.37	< .001
Left inferior parietal lobule (40)	–44, –46, 52	49	.03	10.65	3.83	< .001
Right inferior semi-lunar lobule	28, –68, –44	19	.15	10.02	3.76	< .001
Right cerebellar tonsil	18, –58, –42	65	.01	9.28	3.67	< .001
Right cerebellar tonsil	14, –64, –38			8.49	3.56	< .001
Left middle frontal gyrus (8)	–48, 30, 36	11	.26	8.80	3.60	< .001
Right middle occipital gyrus (19)	38, –84, 26	10	.29	8.71	3.59	< .001
Left angular gyrus (39)	–30, –56, 44	35	.06	8.55	3.57	< .001
Left thalamus (mammillary body)	–12, –16, 0	25	.10	8.37	3.54	< .001
Right lingual gyrus (18)	36, –78, –6	18	.16	8.27	3.53	< .001
Left declive	–40, –72, –14	18	.16	7.65	3.43	< .001

Left cerebellar tonsil	–14, –66, –36	15	.19	7.63	3.43	< .001
Right lentiform nucleus (putamen)	20, 4, 0	39	.05	7.60	3.42	< .001
Right thalamus (ventral anterior nucleus)	16, 0, 6			5.97	3.11	.001
Left claustrum	–26, 20, 0	9	.31	7.34	3.37	< .001
Right culmen	26, –42, –26	17	.17	6.82	3.28	.001
Right parahippocampus (19)	38, –60, –4	10	.29	6.68	3.25	.001
Left caudate	–8, 6, 6	5	.45	6.52	3.22	.001
Left culmen	–34, –52, –24	5	.45	6.19	3.16	.001

Note. Coordinates represent peak voxel coordinates in millimeters; For clarity, only clusters with at least five voxels were listed in the table; BA = Brodmann area; Height threshold T: 5.89 ($p < .001$ uncorrected at the voxel level).

Table F6

Clusters statistically significantly more activated in response to sleep images vs. baseline in participants with insomnia at V1

Brain region (BA)	Coordinates (x, y, z)	Cluster-level		Peak-level		
		Cluster size (voxels)	$p_{uncorrected}$	t	z_E	$p_{uncorrected}$
Left culmen	–34, –54, –26	26443	< .001	18.09	6.76	< .001
Left cerebellar tonsil	–32, –62, –34			17.16	6.65	< .001
Right declive	16, –70, –24			16.19	6.53	< .001
Right middle frontal gyrus (9)	54, 28, 26	3284	< .001	10.92	5.66	< .001
Right middle frontal gyrus (9)	28, 34, 26			10.11	5.48	< .001
Right precentral gyrus (9)	42, 28, 34			7.13	4.64	< .001
Right substantia nigra	18, –16, –6	1335	< .001	9.31	5.28	< .001
Right thalamus (ventral anterior nucleus)	16, –6, 8			9.02	5.21	< .001
Right lentiform nucleus (putamen)	30, –24, –2			8.50	5.07	< .001
Left superior frontal gyrus (6)	–10, 26, 52	2142	< .001	8.98	5.20	< .001

Left superior frontal gyrus (6)	–6, 6, 66			8.51	5.07	< .001
Right medial frontal gyrus (8)	4, 38, 38			7.67	4.82	< .001
Left supramarginal gyrus (40)	–58, –50, 28	96	.03	5.26	3.90	< .001
Left medial frontal gyrus (6)	–6, –8, 52	70	.05	4.61	3.58	< .001
Left cingulate gyrus (24)	–6, –2, 44			4.46	3.51	< .001
Left inferior frontal gyrus (47)	–36, 26, –18	6	.56	4.38	3.46	< .001
Left posterior cingulate gyrus (29)	–4, –40, 16	9	.47	4.37	3.46	< .001
Left middle temporal gyrus (21)	–46, 0, –36	15	.34	4.35	3.44	< .001
Left superior temporal gyrus (38)	–52, 8, –18	12	.40	4.25	3.39	< .001
Left middle frontal gyrus (10)	–34, 62, 14	7	.53	4.23	3.38	< .001
Left inferior frontal gyrus (47)	–34, 22, –10	16	.33	4.18	3.35	< .001
Right supramarginal gyrus (40)	64, –44, 28	7	.53	4.13	3.32	< .001
Left cingulate gyrus (32)	10, 22, 32	18	.30	4.08	3.29	< .001
Right lentiform nucleus (putamen)	34, 0, 4	25	.22	4.02	3.26	.001
Right lentiform nucleus (putamen)	32, 2, –4			3.84	3.16	.001
Left superior parietal lobule (7)	–14, –44, 68	8	.50	3.88	3.18	.001

Note. Coordinates represent peak voxel coordinates in millimeters; For clarity, only clusters with at least five voxels were listed in the table; BA = Brodmann area; Height threshold T: 3.73 ($p < .001$ uncorrected at the voxel level).

Appendix G: Data about Brain Activity Predicting Change in Subjective Sleep Measures

Table G1

Results for BA39 Activity Predicting Change in Number of Awakenings With an Interaction

Source	*df*	*SS*	*MS*	*F*	*p*	η^2
BA39 Activity	1	6.18	6.18	12.51	.00	0.32
Group	1	6.34	6.34	12.84	.00	0.33
BA39 Activity*Group	1	0.00	0.00	0.00	1.00	0.00
Residuals	14	6.92	0.49			

Note. BA39: Brodmann area 39

Table G2

Results for BA39 Activity Predicting Change in Number of Awakenings Without an Interaction

Source	*df*	*SS*	*MS*	*F*	*p*	η^2
BA39 Activity	1	6.18	6.18	13.41	.00	0.32
Group	1	6.34	6.34	13.75	.00	0.33
Residuals	15	6.92	0.46			

Note. BA39: Brodmann area 39

Table G3

Results for BA39 Activity Predicting Change in Total Sleep Time With an Interaction

Source	*df*	*SS*	*MS*	*F*	*p*	η^2
BA39 Activity	1	0.51	0.51	2.19	.16	0.09
Group	1	1.78	1.78	7.62	.02	0.30
BA39 Activity*Group	1	0.34	0.34	1.47	.25	0.06
Residuals	14	3.27	0.23			

Note. BA39: Brodmann area 39

Table G4

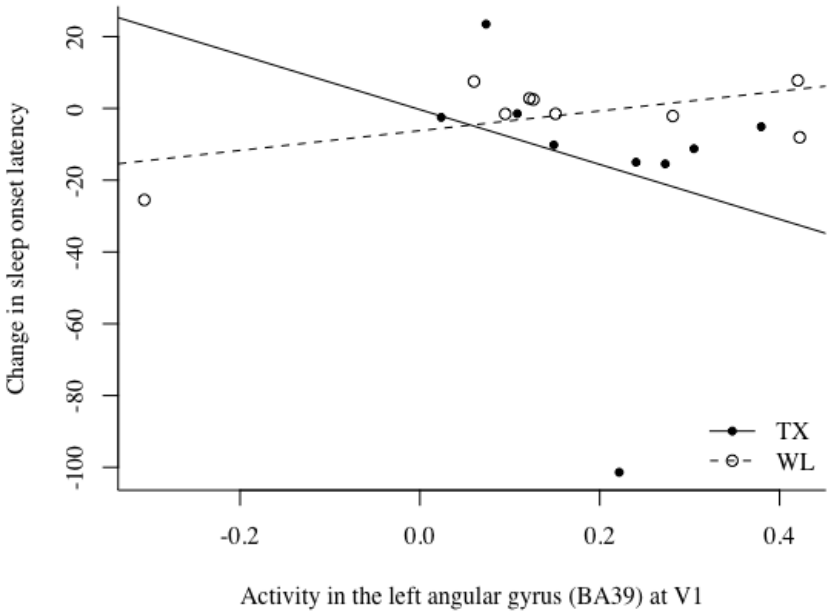
Results for BA39 Activity Predicting Change in Total Sleep Time Without an Interaction

Source	*df*	*SS*	*MS*	*F*	*p*	η^2
BA39 Activity	1	0.51	0.51	2.13	.17	0.09
Group	1	1.78	1.78	7.39	.02	0.30
Residuals	15	3.62	0.24			

Note. BA39: Brodmann area 39

Figure G1

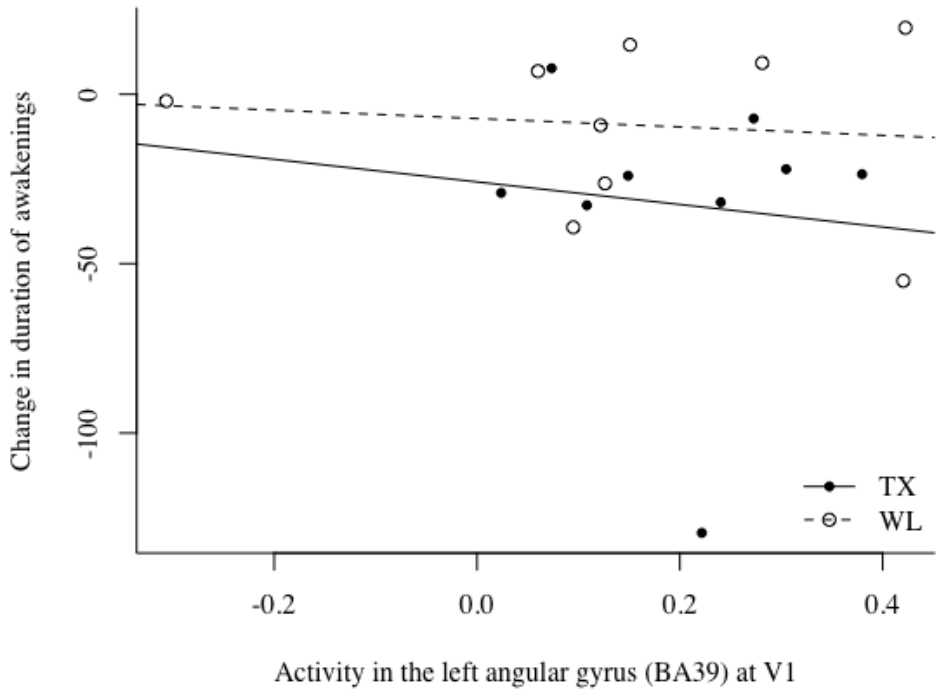
Brodmann area 39 (BA39) activity predicting change in sleep onset latency



Note. TX: immediate treatment group; WL: wait-list control group

Figure G2

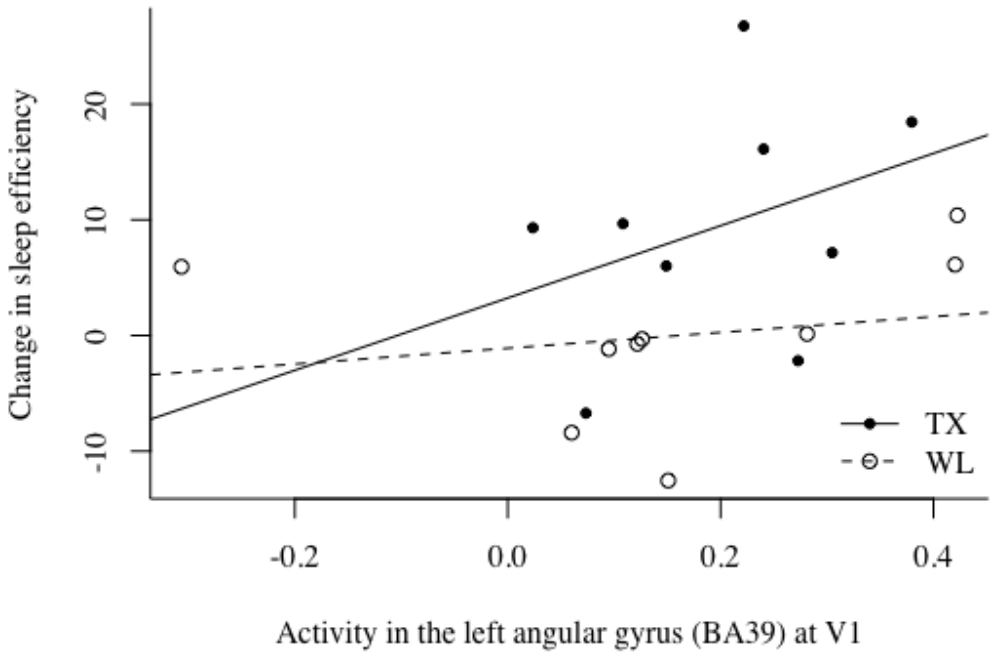
Brodmann area (BA39) activity predicting change in duration of awakenings



Note. TX: immediate treatment group; WL: wait-list control group

Figure G3

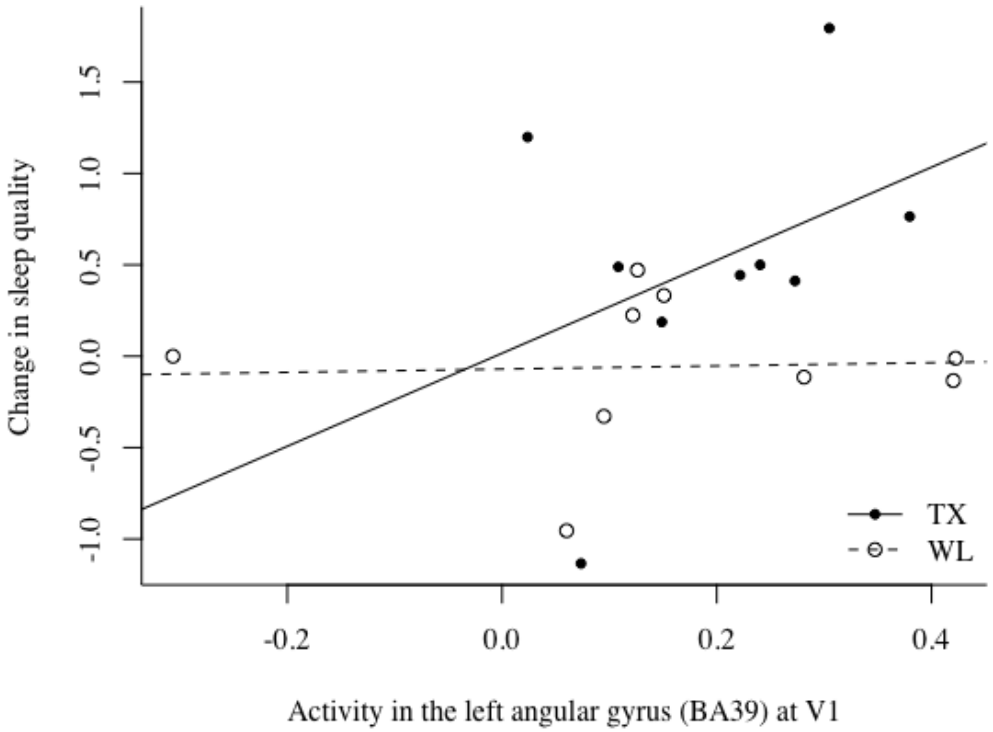
Brodmann area (BA39) activity predicting change in sleep efficiency



Note. TX: immediate treatment group; WL: wait-list control group

Figure G4

Brodmann area (BA39) activity predicting change in sleep quality



Note. TX: immediate treatment group; WL: wait-list control group

Table G5

Results for BA6 Activity Predicting Change in Number of Awakenings With an Interaction

Source	*df*	*SS*	*MS*	*F*	*p*	η^2
BA6 Activity	1	0.00	0.00	0.01	.95	0.00
Group	1	8.06	8.06	9.96	.01	0.41
BA6 Activity*Group	1	0.06	0.06	0.07	.79	0.00
Residuals	14	11.32	0.81			

Note. BA6: Brodmann area 6

Table G6

Results for BA6 Activity Predicting Change in Number of Awakenings Without an Interaction

Source	*df*	*SS*	*MS*	*F*	*p*	η^2
BA6 Activity	1	0.00	0.00	0.01	.95	0.00
Group	1	8.06	8.06	10.62	.01	0.41
Residuals	15	11.38	0.76			

Note. BA6: Brodmann area 6

Table G7

Results for BA6 Activity Predicting Change in Total Sleep Time With an Interaction

Source	*df*	*SS*	*MS*	*F*	*p*	η^2
BA6 Activity	1	0.48	0.48	2.41	.14	0.08
Group	1	2.15	2.15	10.92	.01	0.36
BA6 Activity*Group	1	0.52	0.52	2.63	.13	0.09
Residuals	14	2.76	0.20			

Note. BA6: Brodmann area 6

Table G8

Results for BA6 Activity Predicting Change in Total Sleep Time Without an Interaction

Source	*df*	*SS*	*MS*	*F*	*p*	η^2
BA6 Activity	1	0.48	0.48	2.18	.16	0.08
Group	1	2.15	2.15	9.85	.01	0.36
Residuals	15	3.28	0.22			

Note. BA6: Brodmann area 6

Table G9

Results for BA6 Activity Predicting Change in Sleep Efficiency With an Interaction

Source	*df*	*SS*	*MS*	*F*	*p*	η^2
BA6 Activity	1	312.70	312.70	4.97	.04	0.19
Group	1	453.40	453.40	7.21	.02	0.27
BA6 Activity*Group	1	4.60	4.60	0.07	.79	0.00
Residuals	14	880.40	62.90			

Note. BA6: Brodmann area 6

Table G10

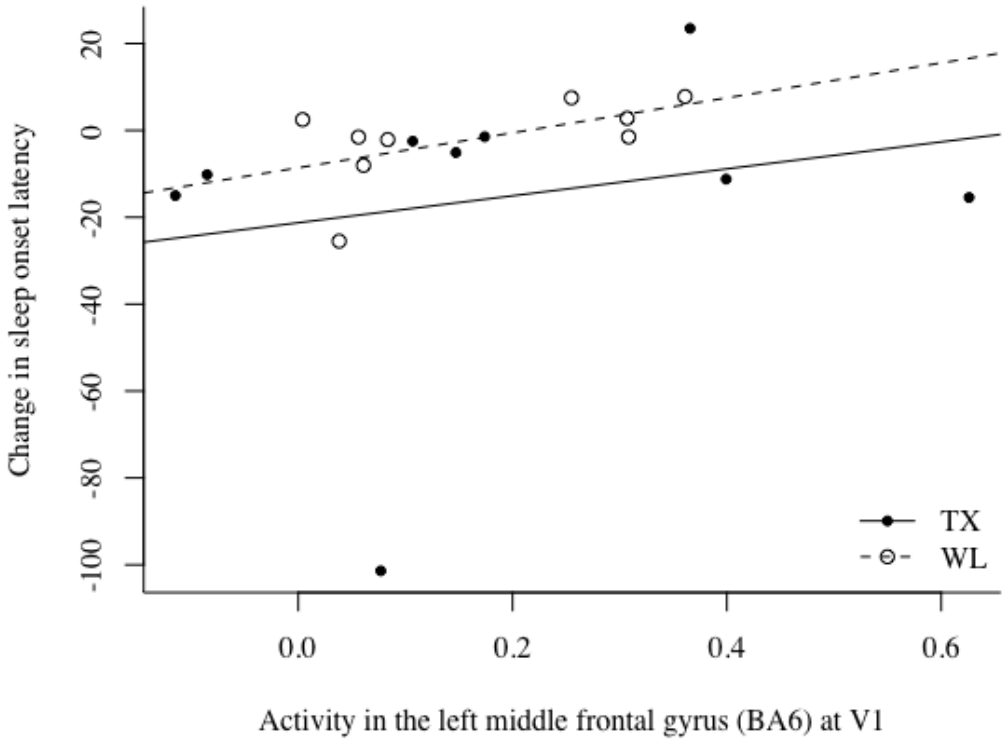
Results for BA6 Activity Predicting Change in Sleep Efficiency Without an Interaction

Source	*df*	*SS*	*MS*	*F*	*p*	η^2
BA6 Activity	1	312.70	312.70	5.30	.04	0.19
Group	1	453.40	453.40	7.69	.01	0.27
Residuals	15	885.00	59.00			

Note. BA6: Brodmann area 6

Figure G5

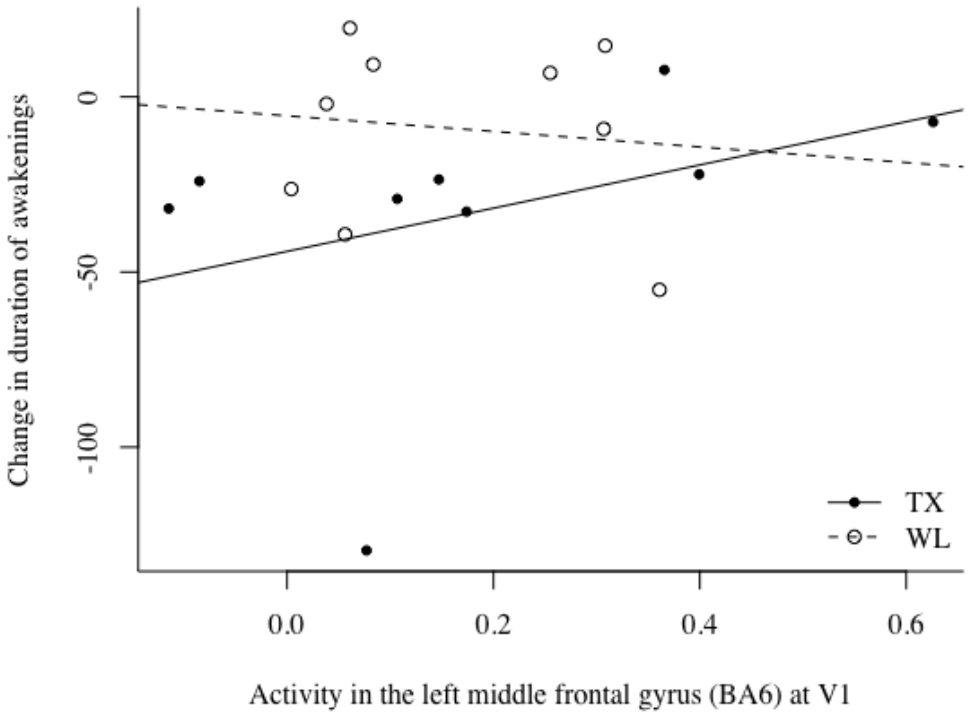
Brodmann area 6 (BA6) activity predicting change in sleep onset latency



Note. TX: immediate treatment group; WL: wait-list control group

Figure G6

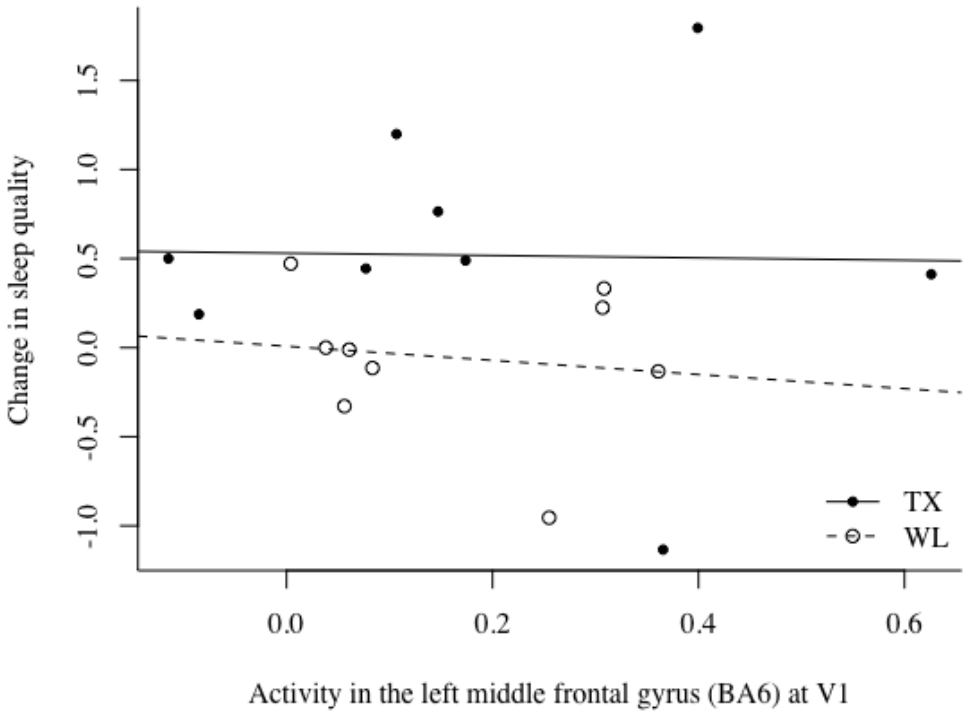
Brodmann area 6 (BA6) activity predicting change in duration of awakenings



Note. TX: immediate treatment group; WL: wait-list control group

Figure G7

Brodmann area 6 (BA6) activity predicting change in sleep quality



Note. TX: immediate treatment group; WL: wait-list control group