

# Effects of Cognitive Arousal and Physiological Arousal on Sleep Perception

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**Study Objectives:** Two experiments were conducted to investigate the effect of presleep arousal on sleep perception. Experiment 1 examined the link between presleep cognitive arousal and distorted perception of sleep and compared the relative effect of anxious and neutral cognitive arousal on sleep perception. Experiment 2 compared the relative effect of anxious cognitive arousal and physiological arousal on sleep perception.

**Design:** Participants completed a nap session. Just prior to the nap, the participants were randomly assigned to 1 of 3 groups to receive different arousal manipulations. They were then allowed to go to sleep and were asked to report their sleep perception upon waking.

**Setting:** Sleep laboratory.

**Participants:** Fifty-four healthy good sleepers in each experiment.

**Interventions:** N/A.

**Measurements and Results:** Self-reported sleep, actigraphy-defined sleep, and the discrepancy between them were indexed. In Experiment 1, participants who were experimentally manipulated to experience anxious cognitive arousal during the presleep period reported longer sleep-onset

latency. Both the Anxious Cognitive Arousal Group and the Neutral Cognitive Arousal Group exhibited a greater discrepancy between self-reported and actigraphy-defined sleep, relative to participants who received no manipulation. In Experiment 2, participants who were experimentally manipulated to experience anxious cognitive arousal or physiological arousal during the presleep period reported longer sleep-onset latency and shorter total sleep time, and both groups exhibited a greater discrepancy between the self-reported and actigraphy-defined sleep, relative to participants who received no manipulation.

**Conclusions:** Results suggest that *both* presleep cognitive arousal and presleep physiological arousal contribute to distorted perception of sleep.

**Key Words:** cognitive arousal, worry, physiological arousal, caffeine, sleep, distorted perception, insomnia

**Abbreviations:** SOL = Sleep-onset latency, TST = Total sleep time

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## INTRODUCTION

THE TRANSITION FROM WAKEFULNESS TO SLEEP IS MARKED BY A PROGRESSIVE LOSS OF CONSCIOUSNESS, REDUCTION OF STIMULUS RECEPTION, CESSATION OF BEHAVIORAL RESPONSE, AND AN ABSENCE OF MEMORY.<sup>1</sup> Accordingly, for the sleeper, the exact point of sleep onset is elusive. One interesting finding to emerge from research on human sleep is that many people with insomnia overestimate their sleep-onset latency (SOL) and underestimate their total sleep time (TST), relative to polysomnography.<sup>2-9</sup> Further, when woken up from polysomnographically defined Stage 2 sleep, people with insomnia are more likely than good sleepers to report having been awake the moment just before they were woken,<sup>10-15</sup> a finding recently replicated by Mercer and colleagues.<sup>16</sup>

*The International Classification of Sleep Disorders, Revised*, defines sleep state misperception (also known as pseudoinsomnia or subjective insomnia) as a disorder in which a complaint of insomnia arises when polysomnography demonstrates a “normal sleep pattern” with sleep latencies of less than 15 to 20 minutes, sleep durations in excess of 6.5 hours, and a normal number and duration of awakenings.<sup>17p33</sup> However, the utility of this diagnosis has been questioned on the basis that distorted perception of sleep is ubiquitous among people with insomnia.<sup>18-19</sup> Indeed, there is evidence that distorted perception of sleep occurs across various diagnoses published in *The International Classification of Sleep Disorders, Revised*, including psychophysiologic insomnia, insomnia

associated with depression, and insomnia associated with inadequate sleep hygiene.<sup>6,20</sup> It seems likely then that individuals with sleep state misperception represent only 1 extreme of a continuum.<sup>18,21</sup>

The role of distorted perception of sleep in the maintenance of insomnia has been increasingly recognized, as reflected in its incorporation into 2 recent theoretical models of chronic insomnia. First, Perlis and colleagues,<sup>22-23</sup> based on the observation that people with insomnia exhibit more high-frequency electroencephalographic activity (in the beta to gamma range) at or around sleep onset, have proposed that cortical arousal may account for distorted perception. Cortical arousal is conceived of as 1 of 3 forms of arousal that may contribute to insomnia: cortical, cognitive, and somatic arousal. While the model recognizes that these 3 forms of arousal interact, it is suggested that cortical arousal (as a conditioned response) is the “primary feature of insomnia.”<sup>22p185</sup> Second, Harvey<sup>24</sup> has proposed that distorted perception of sleep is 1 of the core maintaining processes in chronic insomnia. According to this model, the perception of insufficient sleep increases worry about sleep and anxiety that, in turn, worsens sleep because worry and arousal are antithetical to optimal sleep onset.<sup>24</sup> Consistent with this proposal, it has been suggested that distorted perception of sleep, in its severe form, may be considered a “prodromic or transitional state” in the development of objective insomnia.<sup>19p908</sup> Despite its potential theoretical and clinical significance, little is known about the mechanisms underpinning distorted perception of sleep in insomnia.

One mechanism proposed to underpin distorted perception of sleep in insomnia is cognitive arousal. The majority of individuals with insomnia cite cognitive arousal as the primary determinant of their sleep disturbance.<sup>25-26</sup> The presence of cognitive arousal during the presleep period is highly correlated with subjectively estimated and objectively measured SOL.<sup>27</sup> Further, a number of laboratory studies have demonstrated that experimentally increasing presleep cognitive arousal, in good sleepers, increases objective SOL.<sup>28-30</sup> Taken together, these findings raise the possibility that cognitive arousal is a cause, rather than a by-product, of sleep disturbances. Further, based on the robust finding from the time-perception literature that time seems longer when the number of units of information processed per unit of time increases,<sup>31-33</sup> it has been proposed that cognitive arousal during the presleep period may be the cause

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of distorted perception of sleep.<sup>34,35</sup> Consistent with this proposal, a positive correlation has been noted between negative presleep cognitive arousal and distorted perception of sleep.<sup>27,36</sup>

Another mechanism proposed to underpin distorted perception of sleep in insomnia is physiological arousal. Individuals with insomnia have been reported to exhibit a generally higher level of physiological activation, as indicated by higher body temperature,<sup>2</sup> higher whole-body oxygen consumption,<sup>37</sup> higher phasic vasoconstriction,<sup>38</sup> greater frontal and mentalis electromyogram activity,<sup>39</sup> and greater basal skin resistance.<sup>38</sup> Further, laboratory studies have shown that sleep disturbances mimicking insomnia (eg, longer SOL and shorter TST) can be experimentally induced, in good sleepers, by the administration of caffeine, a stimulant drug that increases arousal.<sup>37,40-41</sup> Taken together, these findings raise the possibility that physiological arousal is causal, rather than epiphenomenal, to poor sleep. Further, in good sleepers, the distorted perception of SOL decreases following the administration of benzodiazepines and increases following the administration of caffeine. Based on these findings, it has been proposed that physiological arousal present at or around sleep onset may be the cause of distorted perception of sleep.<sup>42</sup>

The present paper describes 2 experiments that aimed to systematically investigate the adequacy of these 2 mechanisms proposed to underpin distorted perception of sleep.

## EXPERIMENT 1

### Overview

Despite the intuitive appeal of the proposal that cognitive arousal is the mechanism that underpins distorted perception of sleep, no previous study has directly tested the impact of presleep cognitive arousal on sleep perception. Accordingly, the primary aim of the present study was to examine the association between presleep cognitive arousal and distorted perception of sleep. The second aim was to establish whether it is anxious cognitive arousal (ie, worry), rather than neutral cognitive arousal, that contributes to distorted perception of sleep. This point has not been clear in previous theorizing.

In previous studies, anxious cognitive arousal has been experimentally manipulated by asking participants to give a speech at the end of a nap session,<sup>28,43</sup> whereas neutral cognitive arousal has been experimentally manipulated by asking participants to do 6 hours of study (2 hours reading own textbook and 4 hours of Ravens Progressive Matrices) before going to bed<sup>30</sup> and by asking participants to think about “a series of moderately difficult mental arithmetic problems” for 7.5 minutes.<sup>29p602</sup> In the present study, anxious presleep cognitive arousal was activated by a “speech threat”<sup>28,43</sup> and neutral presleep cognitive arousal was activated by a parallel “essay task.”

It was hypothesized that participants in the Anxious Cognitive Arousal Group and the Neutral Cognitive Arousal Group would (1) report worse sleep and (2) exhibit a greater discrepancy between self-reported sleep and actigraphy-defined sleep, relative to participants in the No Manipulation Group. The study was exploratory regarding the relative impact of anxious and neutral cognitive arousal on distorted perception of sleep. The participants for this study were good sleepers. This analogue sample was selected because they are relatively free from distorted perception of sleep. Hence, it will be possible to observe the effect of manipulating cognitive arousal on sleep perception with more clarity.<sup>28</sup> Inspired by previous studies,<sup>44-45</sup> we employed an afternoon nap as the testing format for this study. This testing format was selected because it (1) allowed tight experimental control over the procedure, (2) enabled testing to be conducted in a resource- and time-efficient manner, and (3) relevant only to Experiment 2, the selected testing format minimized potential ethical concerns about the impact of a moderate to strong dose of caffeine given at night on the sleep obtained on the following day.

## Method

### Participants

Participants were 97 individuals, aged 18 to 40 years, recruited from 2 local universities in Oxford, UK. Inclusion criteria were that participants had to (1) regard themselves as “good sleepers”; (2) have no difficulty falling asleep, as indicated by a typical SOL of less than 30 minutes; (3) have no problem with sleep maintenance, as indicated by a typical duration of wakefulness after sleep onset of less than 30 minutes; (4) have rated 5 or above for typical sleep quality (“How well do you sleep?” with a response scale of 1, *Not at all well*, to 10, *Very well*); and (5) have rated 5 or above for sleep satisfaction (“How satisfied are you with your sleep?” with a response scale of 1, *Not at all satisfied*, to 10, *Very satisfied*). The second to the fifth inclusion criteria were asked with reference to the sleep patterns over the last month. Participants were excluded if they (1) had taken sleep medications, psychotropic drugs, or recreational drugs in the past month; (2) had consumed alcohol or caffeine the morning prior to the experiment; (3) did not manage to fall asleep during the experiment (as it was not possible to calculate the discrepancy between subjective and objective sleep estimates, a key dependent variable, for these participants); and (4) did not comply with the experiment instructions. After the initial contact, a total of 29 respondents did not respond to the request to schedule an appointment. Of the 68 participants who attended the screening interview, 7 were excluded for reporting a SOL of more than 30 minutes ( $n = 2$ ), low sleep satisfaction ( $n = 4$ ), or the use of psychotropic medications ( $n = 1$ ). Of the 61 participants tested, a further 7 were not included in the final analysis for failing to fall asleep during the session (Anxious Cognitive Arousal Group:  $n = 2$ ; Neutral Cognitive Arousal Group:  $n = 1$ ; No Manipulation Group:  $n = 0$ ) or due to noncompliance with the experiment instructions ( $n = 4$ ). The final sample comprised 54 good sleepers.

### Design

Prior to the nap, participants were randomly assigned to 1 of 3 groups. The first group was the Anxious Cognitive Arousal Group, who received a manipulation designed to raise the level of presleep cognitive activity and anxiety (speech threat;  $n = 18$ ). The second group was the Neutral Cognitive Arousal Group, who received a manipulation designed to raise the level of presleep cognitive activity *without* increasing anxiety (essay task;  $n = 18$ ). The third group was the No Manipulation Group, who received no manipulation and served as an index of baseline response ( $n = 18$ ). Five minutes after waking, the participants were asked to report their perception of the amount of sleep they had just obtained. The research protocol was approved by the Ethics Committee of the Department of Experimental Psychology, University of Oxford, UK.

### Procedure

#### Before the Nap

Prior to the study, the participants were informed that the investigation aimed to study “human sleep and postsleep activity” and that they might be asked to perform a “linguistic task” following the nap. Also, the participants were asked to keep their normal sleep-wake patterns the week before the experiment and to refrain from consuming any alcoholic or caffeinated beverages the morning preceding the experiment. The nap session was scheduled to capitalize on the “postlunch dip” of core body temperature, which is associated with an increased likelihood of sleep initiation.<sup>46,47</sup> For “morning”-type participants (ie, those who usually go to bed before midnight and wake up before 9 AM), the nap session started in the early afternoon (1:00-2:30 PM). For “evening”-type participants (ie, those who usually go to bed after midnight and wake up after 9 AM), the nap session started later in the afternoon (2:30-4:00 PM).<sup>46</sup> The participants reported to the sleep laboratory approximately 30 minutes before the nap session began. On arriving at the laboratory, informed consent was obtained and a screening interview was adminis-

tered to index the selection criteria. The participants then completed a battery of questionnaires, which included the Pittsburgh Sleep Quality Index (PSQI),<sup>48</sup> the Penn State Worry Questionnaire (PSWQ),<sup>49</sup> the Beck Depression Inventory (BDI),<sup>50</sup> the Beck Anxiety Inventory (BAI),<sup>51</sup> and the State-Trait Anxiety Inventory (STAI),<sup>52</sup> to index their sleep quality, tendency to worry, level of depression, and level of anxiety, respectively. Next, the participants entered into a sound-attenuated sleep laboratory, where they were fitted with an actigraph (a sleep-monitoring device) before settling in bed. The participants were then instructed to lie in a supine position, with the lights on and their eyes open, for a 5-minute baseline period. When 5 minutes had elapsed, the participants were asked to rate their level of cognitive arousal (“In the last five minutes, how active or preoccupied by thoughts has your mind been?” with a response scale of 1, *not at all preoccupied*, to 10 *Very much preoccupied*) and anxiety (“In the last five minutes, how anxious have you felt?” with a response scale of 1 *Not at all anxious* to 10 *Very anxious*) with reference to their experience during the baseline period.

Immediately prior to the nap, the experimental manipulation was administered. The Anxious Cognitive Arousal Group was given a speech threat. Participants in this group were told that they would be asked to give a speech to 3 psychologists and to a video camera on waking. The Neutral Cognitive Arousal Group was given an essay task. Participants

in this group were told that they would be asked to write an essay on waking. The topic was identical for the speech task and the essay task; both were asked to comment on foot and mouth disease in Britain (at the time of the study this was an impending national crisis). Two manipulation checks were administered immediately after they were told about their tasks; participants were asked to rate, on a 10-point scale, (1) the perceived task difficulty (“How hard or easy do you think it will be to give a speech [or to write an essay]?” with a response scale of 1, *Not at all hard*, to 10, *Very hard*) and (2) their task-induced anxiety (“How anxious do you feel about giving the speech [or writing the essay]?” with a response scale of 1, *Not at all anxious*, to 10, *Very anxious*). The participants in the No Manipulation Group were given no manipulation and were simply told to “take a nap.” Following that, participants were given 1 hour for the nap. As the crucial dependent variable was time perception, the participants were not informed of the duration of the nap.

### After the Nap

When the nap session was over, the participant was given 5 minutes to overcome sleep inertia and get back to the “daytime mode” (eg, switching on the light, tightening loosened clothing, collecting their thoughts). A postnap questionnaire was then administered, in which the participants estimated the time it took them to fall asleep (ie, SOL) and the amount of time they had slept during the nap session (ie, TST). In addition, the participants completed the Presleep Cognitive Activity Questionnaire that indexed the presence of presleep cognitive activity (Cronbach’s  $\alpha = .83$ ; item-total correlation ranges from .58 to .87). This questionnaire was adapted from the cognitive subscale of the Pre-Sleep Arousal Scale by Nicassio and colleagues<sup>53</sup> to measure *neutral* presleep cognitive activity. Thus, the items in the Pre-Sleep Arousal Scale that concern anxious or depressive thoughts were dropped, and the initial instructions were slightly reworded so that the questions referred specifically to the presleep period during the nap session. The Presleep Cognitive Activity Questionnaire comprised 7 items (eg, “While you were trying to get to sleep, to what extent were you actively organizing your thoughts and trying to make sense out of them?” with a response scale of 1, *Not at all*, to 10 *Extremely*). The total score will be referred to as the *Presleep Cognitive Activity Score*. The participants were also asked to complete the STAI state scale.<sup>52</sup> Similarly, the initial instructions were slightly reworded so that the questions referred to the prenap period as an index of their level of anxiety while trying to get to sleep. This score will be referred to as the *Presleep Anxiety Score*. Just before ending the session, the participants were probed as to whether or not they believed that they would be asked to give a speech or to write an essay (Response scale: *Yes, No*). Those who answered *No* to this question were considered to have failed the manipulation check (Anxious Cognitive Arousal Group:  $n = 4$ ; Neutral Cognitive Arousal Group:  $n = 0$ ; No Manipulation Group:  $n = 0$ ) and were thus not included in the final analysis. The participants were then informed that neither a speech nor an essay would be required, were fully debriefed, and then paid an honorarium. Before leaving the session, the participants were asked to not disclose the details of the experiment to fellow students or colleagues who may volunteer.

**Table 1**—Participant Characteristics, Baseline Variables, Manipulation Checks and Dependent Variables for Experiment 1\*

	Groups			Statistical Analysis		
	Anxious Cognitive Arousal	Neutral Cognitive Arousal	No Manipulation	Test	Results	
<b>Participant Characteristics</b>						
Sex, no.						
Female	11	12	7	$\chi^2$	3.15	
Male	7	6	11			
Age, y	21.5 ± 3.1	20.9 ± 2.3	21.9 ± 2.9	F <sub>2,51</sub>	0.53	
Sleep quality†	8.2 ± 1.0	8.2 ± 0.9	8.1 ± 0.8		0.06	
Sleep satisfaction‡	7.9 ± 1.4	7.9 ± 1.3	7.8 ± 1.2		0.08	
SOL, min	15.6 ± 13.3	9.9 ± 6.0	11.8 ± 5.6		1.82	
TST, h	8.0 ± 1.3	7.7 ± 0.9	7.9 ± 0.7		0.31	
Sleep efficiency, %	94.6 ± 7.7	93.6 ± 7.0	94.5 ± 4.5		0.13	
PSQI	3.4 ± 1.5	3.1 ± 1.4	3.3 ± 1.7		0.15	
PSWQ	45.3 ± 12.3	44.7 ± 13.0	44.8 ± 12.3		0.01	
BDI	6.2 ± 5.0	4.9 ± 5.2	4.6 ± 4.2		0.53	
BAI	8.3 ± 5.5	7.3 ± 4.2	8.3 ± 6.5		0.21	
STAI-Trait	32.9 ± 9.9	32.4 ± 10.8	31.3 ± 6.8		0.37	
STAI-State	35.9 ± 10.2	34.4 ± 11.0	37.6 ± 11.3		0.15	
<b>Baseline Variables</b>						
Cognitive activity	5.5 ± 1.6	5.0 ± 1.5	4.8 ± 1.9			0.88
Anxiety	2.6 ± 1.1	2.6 ± 1.5	2.8 ± 1.4		0.21	
<b>Manipulation Checks</b>						
Task Difficulty§	6.4 ± 2.3	5.7 ± 2.1	N/A	t <sub>34</sub>	0.46	
Task-induced Anxiety¶	5.8 ± 2.6	3.4 ± 1.8	N/A		3.23**	
Presleep Cognitive Activity	32.1 ± 9.7 <sup>a</sup>	32.2 ± 8.9 <sup>a</sup>	23.3 ± 5.5 <sup>b</sup>	F <sub>2,51</sub>	6.85**	
Presleep Anxiety	39.2 ± 8.0 <sup>a</sup>	32.6 ± 8.6 <sup>b</sup>	31.0 ± 6.0 <sup>b</sup>		5.86**	
<b>Dependent Variables</b>						
Self-reported SOL	24.4 ± 17.1 <sup>a</sup>	18.6 ± 7.4	14.3 ± 4.3 <sup>b</sup>		3.80#	
Self-reported TST	19.2 ± 14.1	20.1 ± 13.1	30.3 ± 13.0		3.79#	
Actigraphy-defined SOL¶	8.2 ± 4.4 <sup>a</sup>	9.1 ± 2.6	11.8 ± 2.6 <sup>b</sup>		6.00**	
Actigraphy-defined TST¶	50.3 ± 6.7	48.3 ± 7.9	47.7 ± 3.0		0.91	

\* Data are presented as mean ± SD, except for sex, where frequency is reported. Means in the same row that do not share superscripts represent significant differences.

†1 = Not at all well, 10 = Very well

‡1 = Not at all satisfied, 10 = Very satisfied

§1 = Not at all hard, 10 = Very hard

¶1 = Not at all anxious, 10 = Very anxious

¶ The sum of the actigraphy-defined SOL and TST was less than 60 minutes (the duration of the nap) because the actigraphic data indicated that there were awakenings after sleep onset in some participants and other participants woke prior to the ending of the nap.

#P < .05

\*\* P < .01

SOL refers to sleep-onset latency; TST, total sleep time; PSQI, Pittsburgh Sleep Quality Index; PSWQ, Penn State Worry Questionnaire; BDI, Beck Depression Inventory; BAI, Beck Anxiety Inventory; STAI, State-Trait Anxiety Inventory; N/A, not applicable.

## Materials

### Actigraphy

A Mini-Motionlogger Actigraph Basic (supplied by Ambulatory Monitoring Inc., Ardsley, NY) was used to provide objective estimates of SOL and TST. The Mini-Motionlogger is a wristwatch-like device containing a miniaturized piezoelectric acceleration sensor that detects and stores motor information along with an actual clock time. Data were collected in zero-crossing mode at 60-second intervals and were used to generate an estimation of the sleep-wake cycle using the Cole-Kripke algorithm with Webster's rescoring rules.<sup>54,55</sup> To facilitate more-accurate scoring of sleep, the participants were instructed to depress the event marker on the Mini-Motionlogger once when the nap began and once when the nap was over. Actigraphy is a useful and noninvasive instrument to measure the sleep-wake schedule.<sup>56</sup> Relative to polysomnographic sleep estimates, "the gold standard," the epoch-by-epoch agreement rate for sleep and wakefulness detection in adult good sleepers ranges from 74% to 98%.<sup>57-59</sup> The correlation between actigraphic and polysomnographic estimates ranges from 0.77 to 0.98 for SOL and from 0.82 to 0.90 for TST in adult good sleepers.<sup>50,54</sup> Actigraphy is highly sensitive (87%-99%) in detecting sleep epochs identified by polysomnography<sup>57,60-61</sup>; it may however be less reliable at detecting wake (specificity: 28%-90%).<sup>57,60-61</sup> As will be discussed further in the limitations, actigraphy may not be accurate in some circumstances, such as in participants who lie immobile for an extended period of time.

## Results

Unless otherwise specified, 2-group comparisons were analyzed with independent sample *t*-tests. Three-group comparisons were analyzed with 1-way analysis of variance (ANOVA). To explore significant main effects, Scheffé tests were conducted. Wherever the equal variance assumption was not upheld, Dunnett's T3 tests were conducted. Table 1 displays the mean values of all variables.

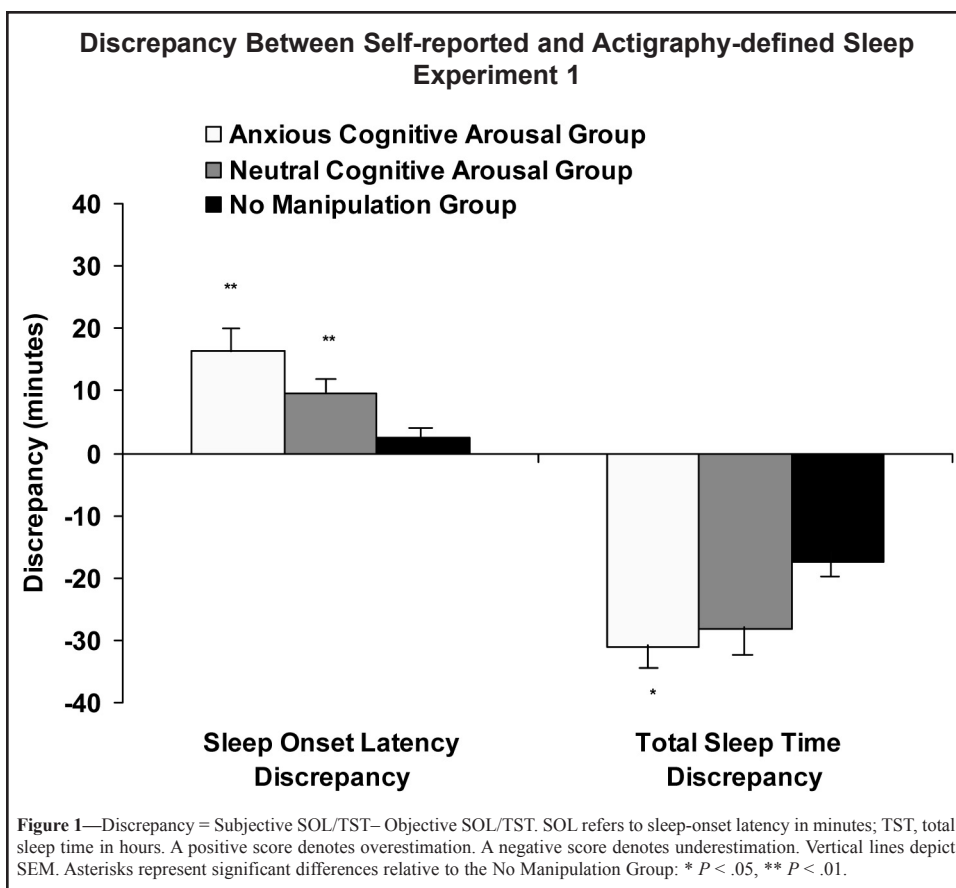


Figure 1—Discrepancy = Subjective SOL/TST– Objective SOL/TST. SOL refers to sleep-onset latency in minutes; TST, total sleep time in hours. A positive score denotes overestimation. A negative score denotes underestimation. Vertical lines depict SEM. Asterisks represent significant differences relative to the No Manipulation Group: \*  $P < .05$ , \*\*  $P < .01$ .

## Participant Characteristics

There were no differences between the groups' composition for the Anxious Cognitive Arousal Group, the Neutral Cognitive Arousal Group, and the No Manipulation Group on sex (analyzed with  $\chi^2$ ), age, sleep quality, sleep satisfaction, typical SOL, typical TST, sleep efficiency, and scores on the PSQI, PSWQ, BDI, BAI, STAI-trait, and STAI-state.

### Baseline Variables

There were no differences in the level of cognitive activity or anxiety following the 5-minute baseline period among the 3 groups.

### Manipulation Checks

The Anxious Cognitive Arousal Group and the Neutral Cognitive Arousal Group did not differ in their rating of task difficulty. However, as expected, the Anxious Cognitive Arousal Group rated their task-induced anxiety higher compared to the Neutral Cognitive Arousal Group. Confirming our assumptions about the 2 manipulations, there was a significant difference for the Presleep Cognitive Activity Score, such that both the Anxious Cognitive Arousal ( $P < .01$ ) and the Neutral Cognitive Arousal ( $P < .01$ ) Groups experienced more cognitive activity during the presleep period compared to the No Manipulation Group. There was also a significant difference for the presleep anxiety score, such that the Anxious Cognitive Arousal Group experienced more anxiety during the presleep period compared with the Neutral Cognitive Arousal Group ( $P < .05$ ) and the No Manipulation Group ( $P < .01$ ).

### Self-reported Sleep

There was a significant difference for self-reported SOL, such that the Anxious Cognitive Arousal Group reported a longer SOL compared to the No Manipulation Group ( $P < .05$ ). There was also a trend ( $P < .054$ ) for self-reported TST, such that the Anxious Cognitive Arousal Group reported a shorter TST compared to the No Manipulation Group.

### Actigraphy-defined Sleep

There was a significant difference for actigraphy-defined SOL, such that the Anxious Cognitive Arousal Group ( $P < .01$ ) had a shorter actigraphy-defined SOL compared to the No Manipulation Group. There was a trend ( $P < .051$ ) for the Neutral Cognitive Arousal Group, such that they had a shorter actigraphy-defined SOL compared to the No Manipulation Group. There were no significant differences for actigraphy-defined TST.

### Discrepancy Between Self-reported and Actigraphy-defined Sleep

Figure 1 depicts the amount of discrepancy between the self-reported and actigraphy-defined SOL and TST. The discrepancy score was calculated by subtracting the actigraphy-defined SOL and TST from the self-reported SOL and TST. A positive value denotes an overestimation, a negative value denotes an underestimation. There were significant differences in the discrepancy score for SOL [ $F_{2,51} = 8.47$ ,  $P < .001$ ] and the discrepancy score for TST [ $F_{2,51} = 5.10$ ,  $P < .01$ ]. The Anxious Cognitive Arousal Group ( $P < .01$ ) and the Neutral Cognitive Arousal Group ( $P < .01$ ) both over-

estimated the time it took them to get to sleep relative to the No Manipulation Group. Furthermore, the Anxious Cognitive Arousal Group underestimated the length of their nap compared to the No Manipulation Group ( $P < .05$ ).

## Discussion

The speech threat and the essay task, matched on task difficulty, were employed to induce anxious cognitive arousal and neutral cognitive arousal, respectively. Confirming the assumptions made about the 2 experimental manipulations, checks administered immediately following the nap indicated that both the Anxious Cognitive Arousal Group and the Neutral Cognitive Arousal Group experienced more cognitive arousal compared to the No Manipulation Group. Yet, only the Anxious Cognitive Arousal Group experienced a significant increase in anxiety during the presleep period.

The first and the second predictions tested were that participants who experienced heightened cognitive arousal would report worse sleep and exhibit a greater discrepancy between self-reported and actigraphy-defined sleep, relative to participants who received no manipulation. In support of the first hypothesis, the Anxious Cognitive Arousal Group reported their SOL to be longer and TST to be shorter compared to the No Manipulation Group (although the latter finding was only marginally significant). In support of the second hypothesis, participants in the Anxious Cognitive Arousal Group and the Neutral Cognitive Arousal Group overestimated their SOL significantly more (+16.3 minutes and +9.6 minutes, respectively) compared to the No Manipulation Group (+3 minutes). In addition, the Anxious Cognitive Arousal Group (-31.2 minutes) underestimated their TST significantly more compared to the No Manipulation Group (-18 minutes). Consistent with the correlational findings that presleep cognitive arousal is positively associated with subjective sleep estimates and the discrepancy between subjective and objective sleep estimates,<sup>27,36</sup> these findings add weight to the suggestion that cognitive arousal may be a mechanism that underpins distorted perception of sleep.<sup>24,34-35</sup>

Interestingly, the heightened cognitive arousal induced by the manipulations did not lead to an objective sleep-onset delay. Instead, the actigraphy-defined SOL of the Anxious Cognitive Arousal Group was significantly shorter, compared to that of the No Manipulation Group, although the absolute difference was small (3.6 minutes). This finding suggests that the participants who experienced heightened cognitive activity and anxiety during the presleep period fell asleep more quickly and is in contrast to a previous experiment where delayed sleep-onset was noted for good sleepers who were assigned a speech task.<sup>28</sup> Two possible explanations can be offered. First, the objective sleep estimates were derived from actigraphy, whereas Gross and Borkovec<sup>28</sup> employed polysomnography. Perhaps some of the variation in results is referable to differences in technology. Because actigraphy estimates sleep by physical movement, we cannot rule out the possibility that the participants given the speech threat or the essay task laid in bed motionless as they planned their upcoming presentation and that this resulted in shorter actigraphy-defined SOL, compared to those who were given no experimental manipulation. A second possibility is that we recruited male and female good sleepers, whereas Gross and Borkovec<sup>28</sup> employed only female college students. Perhaps the 2 samples reacted differentially to the presleep cognitive arousal.

The present experiment provided an opportunity to establish whether cognitive arousal that is anxious or neutral differed in its relative impact on sleep perception. The findings indicated that the Anxious Cognitive Arousal Group reported a longer SOL and a shorter TST compared to the Neutral Cognitive Arousal Group, although the differences were not significant. Also, an inspection of the mean values for the discrepancy score (see Figure 1) indicated that, for both SOL and TST estimates, the Anxious Cognitive Arousal Group had a larger discrepancy score compared to the Neutral Cognitive Arousal Group, although the differences were not significant. These findings are suggestive that anxious cognitive arousal maybe more potent than neutral cognitive arousal in eliciting

distorted perception of sleep. As such, the anxious cognitive arousal manipulation was taken forward and used in Experiment 2.

## EXPERIMENT 2

### Overview

Experiment 2 adapted the methods established in Experiment 1 to investigate the relative impact of anxious cognitive arousal versus physiological arousal on sleep perception. Several hypotheses were tested. First, it was predicted that the Anxious Cognitive Arousal Group would report worse sleep and exhibit more distorted perception of sleep compared to controls, replicating the findings of Experiment 1. Second, based on Bonnet and Arand's findings,<sup>42</sup> it was hypothesized that participants in the Physiologic Arousal Group would also report worse sleep and exhibit more distorted perception of sleep relative to controls. Finally, as no previous research has directly compared the 2 competing proposals of mechanisms that underpin distorted perception of sleep, the study was exploratory with regard to the relative impact of anxious cognitive arousal and physiological arousal on sleep perception.

Based on previous research,<sup>37</sup> caffeine was used as the agent to induce an acute increase in bodily arousal. Caffeine (methylxanthines) is a central nervous system stimulant commonly used to induce vigilance.<sup>62</sup> It is highly soluble and can be quickly absorbed in small to moderate doses.<sup>63</sup> Previous research has shown that the administration of a moderate to strong dose of caffeine results in arousal in the form of increased blood pressure,<sup>64,65</sup> respiratory rate,<sup>66</sup> skin conductance,<sup>65,67</sup> and body metabolism.<sup>37</sup>

### Methods

#### Participants

An independent sample of 93 individuals, aged 18 to 40 years, were recruited from 2 local universities in Oxford, UK. In addition to the inclusion and exclusion criteria outlined for Experiment 1, the participants were also excluded if they had a current medical condition that contraindicated the use of caffeine. After the initial contact, a total of 16 respondents did not respond to the request to schedule an appointment. Of the 77 participants who attended the screening interview, 4 were not included for health reasons (hypertension = 1, mitral valve prolapse = 1, allergy to caffeine = 1; mild current depression = 1), 1 for regular usage of recreational drugs, and 1 for not meeting the full good-sleeper criteria. Of the 71 participants tested, a further 17 were excluded from the analysis due to experiment discontinuation ( $n = 2$ ), actigraphic data-file corruption ( $n = 1$ ), inability to fall asleep during the experiment (Anxious Cognitive Arousal Group:  $n = 2$ ; Physiologic Arousal Group:  $n = 1$ ; Placebo Group:  $n = 3$ ), or manipulation failure ( $n = 8$ ). The final sample comprised 54 healthy good sleepers, none of whom participated in Experiment 1.

#### Design

Prior to the nap, participants were randomly assigned to 1 of 3 groups. The first group was the Anxious Cognitive Arousal Group, who received a manipulation that was designed to raise the level of presleep cognitive activity and anxiety (speech threat;  $n = 18$ ). The second group was the Physiologic Arousal Group who swallowed a caffeine capsule that was intended to raise the level of presleep physiological arousal without also increasing cognitive activity ( $n = 18$ ). The third group was the Placebo Group who swallowed a placebo capsule and served as a control for possible placebo effect due to the capsule administration ( $n = 18$ ). Five minutes after waking, the participants were asked to report their perception of the amount and the quality of sleep they had just obtained. The research protocol was approved by the Oxfordshire Psychiatric Research Ethics Committee, UK.

**Procedure**

**Before the Experiment**

A medical-condition screener was sent to all potential participants asking them to indicate their medical history and current health condition. Moreover, a letter was sent to each participant's physician giving full details regarding their involvement in the experiment. A nap session was only scheduled for those who were medically fit for the study. As for Experiment 1, participants were asked to keep their normal sleep-wake pattern the week before the experiment and to refrain from con-

suming any alcoholic or caffeinated beverages the morning preceding the experiment.

**Before the Nap**

The method of scheduling the nap session and the rationale given to participants were identical to those for Experiment 1. Participants reported to the sleep laboratory approximately 60 minutes before the nap began. Informed consent was obtained from all participants. Following the administration of the screening interview and the battery of ques-

tionnaires used in Experiment 1, the participants were introduced to the sleep laboratory for a 5-minute baseline period. Then the participants were asked to complete the Presleep Cognitive Activity Questionnaire (described in Experiment 1), the STAI-state scale<sup>52</sup> and a Presleep Physiological Arousal Questionnaire (comprised 5 questions to measure caffeine-related physiological arousal, eg, "While you were trying to get to sleep just prior to your nap, to what extent did you experience perspiration in the palms of your hands or other parts of your body?" with a response scale of 1, *Not at all*, to 10, *Extremely*; Cronbach's  $\alpha = .71$ , item-total correlation ranges from .36 to .81) with reference to their experience during the baseline period.

The participants were then asked to engage in 20 minutes of leisure-reading. This involved browsing a selection of neutral but engaging travel guides. Previous pharmacological research has indicated that the effect of caffeine is not immediate, taking typically around 30 minutes to take effect after ingestion.<sup>66,68</sup> That is, with the 20 minutes of leisure-reading and several minutes for further instructions and ratings, the effect of the caffeine should have been 'kicking in' at the beginning of the nap.

In a double-blind fashion, participants in the Physiologic Arousal Group and the Placebo Group were given either a caffeine capsule (caffeine citrate; manufacturer: BCM Specials, UK) or a placebo capsule (lactose; manufacturer: BCM Specials, UK) to swallow approximately 25 minutes prior to the beginning of the nap. The dosage of the capsule varied from 250 mg to 450 mg depending on the body weight of the participants (5 mg/kg). To check if the participants were truly blind to the allocation of the capsules, they were asked to rate their guess as to the content of the capsule on a 7-point scale (Response scale: 1, *Certainly not a caffeine capsule*, to 4, *Don't know*, to 7, *Certainly a caffeine capsule*). When the leisure-reading task was over, participants in the Cognitive Arousal Group were given the speech threat. The topic of the speech was to present a talk based on what they had read from the tour guides. The participants were then asked to rate, on a 10-point scale, how anxious they felt about having to give a speech (Response scale: 1, *Not at all anxious*, to 10, *Very anxious*). All participants were then given an hour for the nap. As for Experiment 1, they were not informed as to the duration of the nap session.

**Table 2—Participant Characteristics, Baseline Variables, Manipulation Checks, and Dependent Variables for Experiment 2\***

	Groups			Statistical Analysis	
	Anxious Cognitive Arousal	Physiologic Arousal	Placebo	Test	Results
<b>Participant Characteristics</b>					
Sex, no.					
Female	11	12	8	$\chi^2$	1.97
Male	7	6	10		
Age, y	23.2 ± 5.4	23.8 ± 5.2	23.8 ± 3.8	$F_{2,51}$	0.80
Body mass index†	22.4 ± 3.2	23.8 ± 4.3	24.0 ± 3.3		1.07
Sleep quality‡	8.4 ± 0.9	8.6 ± 0.9	8.4 ± 0.8		0.43
Sleep satisfaction§	8.2 ± 1.2	8.3 ± 1.1	8.2 ± 1.1		0.10
SOL, min	11.4 ± 8.4	14.7 ± 13.1	12.5 ± 5.9		0.54
TST, h	7.9 ± 1.0	8.3 ± 1.0	7.9 ± 0.7		0.78
Sleep efficiency, %	93.4 ± 5.9	95.7 ± 4.0	93.6 ± 6.2		0.95
PSQI	2.8 ± 1.4	2.9 ± 1.2	3.0 ± 1.1		0.15
PSWQ	42.1 ± 13.4	39.4 ± 11.9	38.2 ± 13.4		0.41
BDI	4.2 ± 3.8	4.3 ± 4.1	3.5 ± 3.6		0.23
BAI	6.0 ± 6.0	3.3 ± 2.6	3.2 ± 2.6		2.79
STAI-Trait	35.9 ± 9.0	35.8 ± 9.6	33.6 ± 8.3		0.40
STAI-State	31.4 ± 9.0	33.1 ± 9.9	29.1 ± 7.8		0.91
Habitual caffeine intake‡	2.1 ± 1.5	1.7 ± 2.0	2.5 ± 2.0		0.86
<b>Baseline Variables</b>					
Cognitive activity	25.8 ± 8.9	23.6 ± 9.6	21.6 ± 7.0		1.09
Anxiety	28.2 ± 5.9	28.9 ± 6.4	25.4 ± 4.8		1.82
Physiologic arousal	6.9 ± 2.8	6.9 ± 2.6	5.8 ± 1.2		1.23
<b>Manipulation Checks</b>					
Task-induced anxiety¶	5.8 ± 1.7	N/A	N/A	$F_{2,51}$	N/A
Presleep Cognitive Activity	33.7 ± 10.3 <sup>a</sup>	23.6 ± 10.0 <sup>b</sup>	20.1 ± 8.0 <sup>b</sup>		10.06 <sup>***</sup>
Presleep Anxiety	37.8 ± 8.2 <sup>a</sup>	32.2 ± 8.0	26.8 ± 4.8 <sup>b</sup>		10.42 <sup>***</sup>
Presleep Physiologic Arousal	8.9 ± 3.9	10.5 ± 4.2 <sup>a</sup>	7.0 ± 2.2 <sup>b</sup>		3.90 <sup>¶¶</sup>
Knowledge of the Capsule#	N/A	3.8 ± 1.5	3.1 ± 1.3	$t_{34}$	1.60
<b>Dependent Variables</b>					
Self-reported SOL	24.2 ± 18.1 <sup>a</sup>	25.8 ± 19.7 <sup>a</sup>	10.7 ± 4.9 <sup>b</sup>	$F_{2,51}$	5.06 <sup>##</sup>
Self-reported TST	17.9 ± 13.4 <sup>a</sup>	21.4 ± 20.7 <sup>a</sup>	47.8 ± 21.3 <sup>b</sup>		13.62 <sup>***</sup>
Actigraphy-defined SOL <sup>**</sup>	7.5 ± 3.1	9.5 ± 5.8	7.5 ± 1.6		1.62
Actigraphy-defined TST <sup>**</sup>	50.5 ± 5.1	47.2 ± 13.2	51.2 ± 4.8		1.10
Sleep Depth††	4.1 ± 1.7 <sup>a</sup>	4.4 ± 2.2 <sup>a</sup>	6.4 ± 2.2 <sup>b</sup>		6.33 <sup>###</sup>
Sleep Serenity‡‡	5.3 ± 1.6	4.7 ± 2.1 <sup>a</sup>	6.4 ± 2.0 <sup>b</sup>		3.47 <sup>¶¶</sup>
Sleep Quality§§	4.7 ± 1.7 <sup>a</sup>	5.3 ± 1.9	6.7 ± 2.0 <sup>b</sup>		5.15 <sup>**</sup>
Refreshed Feeling on Waking †††	5.1 ± 2.0 <sup>a</sup>	6.1 ± 2.0	7.1 ± 2.2 <sup>b</sup>		4.04 <sup>¶¶</sup>

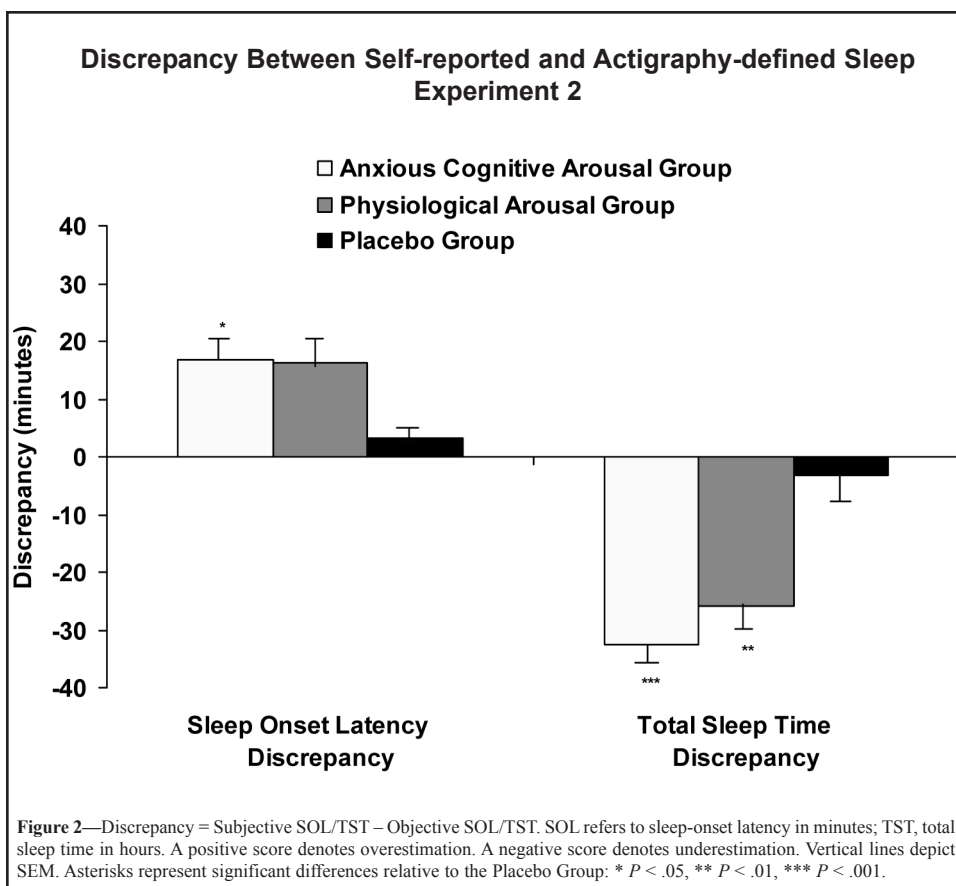
\*Data are presented as mean ± SD, except for sex, where frequency is reported. Means in the same row that do not share superscripts represent significant differences.  
 †Body mass index = weight (kg)/ height (m)<sup>2</sup>  
 ‡1 = Not at all well, 10 = Very well  
 § = Not at all satisfied, 10 = Very satisfied  
 ‡Habitual caffeine intake is reported in cups of coffee.  
 ¶1 = Not at all anxious, 10 = Very anxious  
 #1 = Certainly not a caffeine capsule, 4 = Don't know, 7 = Certainly a caffeine capsule  
 \*\*The sum of the actigraphy-defined SOL and TST was less than 60 minutes (the duration of the nap) because the actigraphic data indicated that there were awakenings after sleep onset in some participants and other participants woke prior to the ending of the nap.  
 ††1 = Very light, 10 = Very deep  
 ‡‡1 = Not at all serene, 10 = Very serene  
 §§1 = Very poor, 10 = Very good  
 †††1 = Not at all refreshed, 10 = Very refreshed  
 ¶¶ $P < .05$   
 ### $P < .01$   
 \*\*\* $P < .001$   
 SOL refers to sleep-onset latency; TST, total sleep time; PSQI, Pittsburgh Sleep Quality Index; PSWQ, Penn State Worry Questionnaire; BDI, Beck Depression Inventory; BAI, Beck Anxiety Inventory; STAI, State-Trait Anxiety Inventory; N/A, not applicable.

## After the Nap

Five minutes after waking, a postnap questionnaire was administered, in which the participants estimated their SOL and TST. To explore in detail the effect of the experimental manipulations on the qualitative aspects of sleep perception, the participants were also asked to rate the following 4 parameters: (1) sleep depth (“How would you rate the depth of the sleep you obtained during your nap?” with a response scale of 1, *Very light*, to 10, *Very deep*); (2) sleep serenity (“How would you rate the serenity of the sleep you obtained during your nap?” with a response scale of 1, *Not at all serene*, to 10, *Very serene*); (3) sleep quality (“How would you rate the quality of the sleep you obtained during your nap?” with a response scale of 1, *Very poor*, to 10, *Very good*); and (4) refreshed feeling on waking (“How refreshed did you feel on waking?” with a response scale of 1, *Not at all refreshed*, to 10, *Very refreshed*). Furthermore, the participants completed the Presleep Cognitive Activity Questionnaire (described in Experiment 1), the STAI-state scale<sup>48</sup> and the Presleep Physiologic Arousal Questionnaire with reference to their experience while they were trying to get to sleep. Just before ending the session, the participants were probed as to whether or not they believed that they would need to give a speech (Response scale: *Yes, No*). Those who answered *No* to this question were considered to have failed the manipulation check (Anxious Cognitive Arousal Group:  $n = 8$ ) and were thus not included in the final analysis. Participants were then informed that a speech would not be required, were fully debriefed, and then paid an honorarium. Before leaving the session, the participants were asked to not disclose the details of the experiment to fellow students or colleagues who may volunteer.

## Results

The data-analysis method was identical to that described for Experiment 1. Table 2 displays the mean values for all variables.



## Participant Characteristics

The Anxious Cognitive Arousal Group, the Physiologic Arousal Group, and the Placebo Group were matched on sex composition (analyzed with  $\chi^2$ ); age; body mass index; sleep quality; sleep satisfaction; typical SOL; typical TST; sleep efficiency; scores on the PSQI, PSWQ, BDI, BAI, STAI-trait and STAI-state; and habitual caffeine intake.

## Baseline Variables

There were no differences in the level of cognitive activity, anxiety, or physiological arousal following the 5-minute baseline period across the 3 groups.

## Manipulation Checks

The participants in the Anxious Cognitive Arousal Group felt anxious when they received the speech threat. Confirming our assumptions about the speech threat, there was a significant difference for Presleep Cognitive Activity Score, such that the Anxious Cognitive Arousal Group experienced more cognitive activity during the presleep period, compared to the Physiologic Arousal Group ( $P < .01$ ) and the Placebo Group ( $P < .001$ ). There was also a significant difference for presleep anxiety score, such that the Anxious Cognitive Arousal Group experienced more anxiety during the presleep period, compared to the Placebo Group ( $P < .001$ ). The participants in the 2 capsule-taking groups were equally blind to the allocation of the capsules. Confirming our expectation of the administration of the caffeine and placebo capsules, there was a significant difference for Presleep Physiologic Arousal Score, such that the participants in the Physiologic Arousal Group experienced more bodily arousal compared to the Placebo Group ( $P < .05$ ).

## Self-reported Sleep

There was a significant difference for self-reported SOL, such that both the Anxious Cognitive Arousal Group ( $P < .05$ ) and the Physiologic Arousal Group ( $P < .05$ ) reported a longer SOL compared to the Placebo Group. Likewise, there was a significant difference for TST, such that both the Anxious Cognitive Arousal Group ( $P < .001$ ) and the Physiologic Arousal Group ( $P < .001$ ) reported a shorter TST compared to the Placebo Group. With respect to the qualitative aspects of the sleep perception (sleep depth, serenity, quality, and refreshed feeling on waking), both the Anxious Cognitive Arousal Group and Physiologic Arousal Group rated the sleep they obtained from the nap more negatively relative to the Placebo Group.

## Actigraphy-defined Sleep

There were no significant differences for actigraphy-defined SOL and TST.

## Discrepancy Between Self-reported and Actigraphy-defined Sleep

Figure 2 depicts the amount of discrepancy between the self-reported and actigraphy-defined SOL and TST. The calculation of the discrepancy score was identical to Experiment 1 in that a positive value denotes an overestimation, a negative value denotes an underestimation. There were significant differences in the discrepancy scores for SOL ( $F_{2,51} = 4.07$ ,  $P < .05$ ) and for TST ( $F_{2,51} = 10.2$ ,  $P < .001$ ). The Anxious Cognitive Arousal Group significantly

overestimated the time it took them to get to sleep relative to the Placebo Group ( $P < .05$ ). There was also a trend ( $P < .052$ ) such that the Physiologic Arousal Group also overestimated the time it took them to get to sleep compared to the Placebo Group. Relative to the Placebo Group, both the Anxious Cognitive Arousal Group ( $P < .001$ ) and the Physiologic Arousal Group ( $P < .01$ ) significantly underestimated the length of the sleep they obtained from the nap.

## Discussion

The first and second hypotheses of the present experiment were that the Anxious Cognitive Arousal Group and the Physiologic Arousal Group would report worse sleep and exhibit a more distorted perception of sleep, relative to the Placebo Group. In support of the first hypothesis, the Anxious Cognitive Arousal Group reported a longer SOL and a shorter TST and had larger discrepancy scores for both SOL and TST estimates, relative to the Placebo Group. The manipulation check administered following the nap confirmed the assumption that the Anxious Cognitive Arousal Group experienced more presleep cognitive activity and anxiety during the presleep period, relative to the other 2 groups. These findings replicate the results of Experiment 1 and are consistent with the proposal that worry fuels distorted perception of sleep.<sup>24,34,35</sup> Consistent with the second hypothesis, the Physiologic Arousal Group reported a longer SOL and a shorter TST and had larger discrepancy scores for SOL and TST estimates compared to the Placebo Group, although the difference for SOL only approached significance ( $P < .052$ ). Given that the capsules were administered in a double-blind fashion, the significant difference in sleep perception between the Physiologic Arousal Group and the Placebo Group is unlikely to be explained by a placebo effect or an expectation effect. A manipulation check confirmed that the participants were unable to guess the nature of the capsule they swallowed (mean = 3.4). Taken together, these findings lend support to the proposal that physiological arousal fuels distorted perception of sleep.<sup>42,69</sup>

Intriguingly, there were no significant differences for objective SOL and TST estimates in the Physiologic Arousal Group. While these findings resemble those of Landolt et al.,<sup>70</sup> showing little effect of caffeine on polysomnography-defined SOL and sleep architecture, as defined by the Rechtschaffen and Kales sleep-scoring system,<sup>71</sup> they are contrary to a previous study that recorded a dose-related effect of caffeine on objective estimates of sleep.<sup>40</sup> Three possible explanations can be offered. First, large standard deviations were observed for objective SOL and TST estimates in the Physiologic Arousal Group. This suggests that there was substantial variation in the individual response to caffeine. Several measures were taken to control for possible individual differences in the response to caffeine, including giving capsules that had a dose proportional to the participants' weight (5 mg/kg), asking the participants to not ingest caffeine the morning prior to the experiment, and scheduling the nap according to the participants' circadian rhythm. However, future research should control for the possibility that frequent and heavy drinkers of caffeinated beverages are less susceptible to the effect of a moderate dose of caffeine.<sup>72</sup> Second, it is possible that the caffeine capsule needed longer than 25 minutes to be fully ingested. Perhaps the impact of the caffeine on objective sleep estimates would have been more prominent had the leisure-reading time been lengthened. Third, the current experiment took the form of an afternoon nap session. The participants were woken up after an hour had elapsed. It is possible that the short nap duration and the forced awakening may have obscured the full-scale impact of caffeine on sleep.

Finally, it is noted that twice as many people failed the manipulation check (ie, did not believe that they would have to give a speech) in Experiment 2 ( $n = 8$ ), compared to Experiment 1 ( $n = 4$ ). It is possible that this difference is a function of the change in speech topic across the 2 experiments. Unlike Experiment 1, where the participants were given foot and mouth disease as the speech topic (a then-impending national crisis), the participants in Experiment 2 were asked to come up with their own speech topic, based on their reading during the leisure-reading peri-

od. The lack of a specific serious speech topic might have led the participants in Experiment 2 to take the speech threat less seriously.

## GENERAL DISCUSSION

Experiment 1 tested the causal link between presleep cognitive arousal and distorted perception of sleep and compared the relative impact of anxious and neutral cognitive arousal on sleep perception. Consistent with our predictions, the presence of both anxious cognitive arousal and neutral cognitive arousal during the presleep period led to more distorted perception of sleep. Experiment 2 compared the relative effect of anxious cognitive arousal and physiological arousal on sleep perception. The presence of both cognitive and physiological arousal during the presleep period not only distorted the participants' quantitative estimation of SOL and TST, but also led the participants to evaluate the qualitative aspects of the sleep they had obtained more negatively. Together, the results from both experiments provide support for the suggestion that worry and arousal "trick the person into believing that they obtain significantly less sleep than they actually obtained."<sup>24p872</sup>

These results must be interpreted within the confines of several methodological limitations. First, while the use of good sleepers provided abundant and relatively 'clean' subjects for experimentation, future research is needed to replicate these findings with patients with chronic insomnia. However, it should be noted that care would be needed in using actigraphy as the objective measure because patients with insomnia tend to lie awake but motionless for long periods.<sup>61</sup> Second, the advantage of using an afternoon nap as the testing format is that it provides a resource-efficient way of exploring the mechanisms driving distorted perception of sleep, in that large samples can be tested in a relatively short time frame. While a number of studies in the insomnia literature have shown that an afternoon nap is a viable testing format,<sup>45</sup> it will be important in future research to confirm that the findings of nap studies are a true analog of nighttime sleep. Third, we employed actigraphy in both experiments to provide nonintrusive estimates of SOL and TST. Because actigraphy does not provide a direct measure of sleep, there are concerns as to its merit in distinguishing sleep from long periods of quiet wakefulness, especially in people who can lie immobile for an extended period of time.<sup>73,74</sup> Accordingly, the discrepancy between self-reported sleep and actigraphy-defined sleep is used in this paper as an index of the discrepancy between subjective and objective sleep, and yet we emphasize that it needs to be interpreted within the confines of the methods used. A further limitation is that the validation of actigraphy is based on full-night sleep studies rather than naps. In addition, we recognize that it is possible that there was a correlation between movement and the experimental conditions. That is, the shorter SOL observed for the participants who were given the speech threat or the essay task might have been caused by a lack of movement when they were lying quietly in bed planning their upcoming presentation. The use of polysomnography in future research would clarify this issue. Fourth, we have excluded participants who did not manage to fall asleep during the testing session from the final analysis because it was not possible to calculate the discrepancy between subjective and objective sleep estimates, a key dependent variable, for these participants. We recognize that this may have eliminated the participants who were the strongest responders to the experimental manipulations. Having said that, an inspection of the data suggests that the participants who were excluded due to an inability to fall asleep during the testing session were fairly equally distributed across the groups.

One interesting issue that deserves further attention is the multidimensionality of arousal. Even though arousal is usually taken as a term that describes a single integrated state, it is likely that different types of arousal can be distinguished because the production of an aroused state involves different response systems. The most common partition is the one adopted in the current paper; cognitive arousal versus physiological arousal, with the former representing an elevated activation in the mind and the latter representing an elevated activation in the body.<sup>53,75,76</sup> The interrelationship between these 2 types of arousal is controversial. While



there is a high correlation between the 2 types of arousal,<sup>53,76</sup> they do not concord with each other perfectly.<sup>77</sup> In fact, some previous research has shown either no or a selective concordance between cognitive arousal and physiological arousal in response to stressful stimuli.<sup>78,79</sup> Also, the degree of covariance between the cognitive and the physiological arousal systems varies according to the contextual and individual differences. A synchrony is more likely to be observed in high-stress situations and in high-anxiety individuals, whereas a desynchrony is likely to be observed in low-stress situations and in low-anxiety individuals.<sup>80</sup> Findings of the present experiment seem to side with the view that there is a lack of obvious concordance between the cognitive arousal system and the physiological arousal system. Manipulation checks administered immediately following the nap indicate that participants who received a speech threat just prior to the nap reported an increase in cognitive activity and anxiety without a concomitant increase in physiological arousal. Likewise, participants who swallowed a caffeine capsule 25 minutes prior to the nap reported an increase in physiological arousal without a concomitant increase in cognitive arousal. Nevertheless, this null finding should be interpreted with caution because a lack of concordance between the 2 arousal systems may be due to limitations in measurement.<sup>81</sup> Further investigation, employing multiple measurements for each of the arousal systems, is needed to serve as an independent check of the cognitive and physiological arousal manipulations and to delineate the interrelationship between the cognitive arousal system and the physiological arousal system.

## CONCLUSION

This paper describes 2 interlinked experiments that investigated the association between distorted perception of sleep and 2 types of presleep arousal: cognitive arousal and physiological arousal. The evidence indicates that *both* presleep cognitive arousal and presleep physiological arousal appear to contribute to distorted perception of sleep. It would be interesting for future research to delineate the interrelationship between cognitive, physiological, and cortical arousal and to examine their relative contributions to distorted perception of sleep in chronic insomnia.<sup>22,23</sup>

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