Sleep-Related Attentional Bias in Good, Moderate, and Poor (Primary Insomnia) Sleepers

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Evidence was sought of an attentional bias toward a highly representative object of the bedroom environment in good, moderate, and poor (primary insomnia) sleepers. Using a flicker paradigm for inducing change blindness, the authors briefly presented a single scene comprising a group of bedroom environment and neutral objects to participants and then briefly replaced this scene with an identical scene containing a change made to either a bedroom environment or a neutral object. In a 3 × 2 entirely between-participants design, change-detection latencies revealed a sleep-related attentional bias in poor sleepers but not in good sleepers. A possible bias in moderate sleepers was also revealed. It is suggested that attentional bias has a role in the perpetuation and possibly precipitation of primary insomnia.

Keywords: attentional bias, insomnia, charge-detection, sleep disruption, circadian

Sleep disruption is not unusual. Although good sleep usually returns, either through the disappearance of the precipitating agent or through the operation of mechanisms that appear to combat sleep disruption (the sleep homeostat or the circadian timer; Borbely, 1994), sleep disruption sometimes persists. When it persists for longer than a month (and is not a side effect of other medical, neurologic, psychiatric, or psychological disorders or the result of a circadian rhythm disorder), it is called primary insomnia (Diagnostic and Statistical Manual of Mental Disorders, 4th ed. [DSM–IV]; American Psychiatric Association, 1994). This report explores a possible role for attentional bias in the perpetuation and possibly the precipitation of primary insomnia. An attentional bias toward a particular class of stimulus (e.g., sleep-related) is said to have developed when disproportionate processing resources appear to be automatically allocated to exemplars, as compared with other otherwise equivalent stimuli—producing a disproportionate impact on current cognitions. This article is based, first, on a framework that has been helpful in addressing many other clinical disorders and, second, on an extension of this framework to encompass subclinical features of sleep disorder. This extension derives from research on attentional bias in relation to understanding alcohol consumption variability along the entire alcohol use–misuse–abuse–dependence continuum, not just at the clinical pole.

First, support has been found implicating attentional bias in the perpetuation of a wide range of anxiety-related psychological disorders and concerns (see Mogg & Bradley, 1998, for a review). Support has also been found implicating attentional bias in the perpetuation of a family of substance abuse and dependence disorders: for example, with alcohol (Sharma, Albery, & Cook, 2001), heroin (Franken, Kroon, Wiers, & Jansen, 2000) and nicotine (Waters & Feyerabend, 2000). Principally, through the use of the emotional Stroop paradigm (Williams, Mathews, & MacLeod, 2001), heroin (Franken, Kroon, Wiers, & Jansen, 2000) and abuses and dependences (e.g., Drummond, Tiffany, Glautier, & Remington, 1995) are frequently self-maintaining and why relapse so frequently occurs after initially successful treatment. In explanations such as these, attentional bias is conceived as an involuntary (i.e., unconscious, implicit) process but (important to note) gives rise to voluntary (i.e., conscious, explicit) processes. These cognitions feature as part of the disorder.

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and can contribute to subsequent behavior decisions. Franken, Kroon, and Hendriks (2000); Franken, Kroon, Wiers, and Jansen (2000); and Ingaldsson, Thayer, and Laberg (2003), in particular, related attentional bias and craving with risk of relapse in substance abuse and dependence.

Williams et al.’s (1996) model of emotional Stroop performance (extending Cohen, Dunbar, & McClelland’s, 1990, network model of the standard Stroop effect) accounts for attentional bias at the clinical pole of disorders, such as those mentioned above, through the increased responsivity of network input units representing disorder-related concepts—an increase caused by “a history of association with [anxiety-generating] threat or loss and thereby subject to neuromodulatory control affecting the responsivity of those units” (Williams et al., 1996, p. 17). As noted earlier, attentional biases, which reflect discrete changes in the direction of attentional focus in response to “threat,” have been revealed in the anxiety disorders (e.g., Keogh, Dillon, Georgiou, & Hunt, 2001). We would argue that attentional bias may also be a strong candidate model for the perpetuation of primary insomnia. Sleep is a fundamental life process much like eating or drinking. Thus, inability to sleep can be conceptualized as a significant threat as well as an anxiety generator (Espie, 2002). We believe that attentional biases favoring sleep-related stimuli will, during an acute insomnia phase, lead to persistent insomnia in two ways: by promoting sleep preoccupation and by driving heightened sleep effort. This hypothesis is consistent with the reported association between insomnia and both excessive presleep cognitive activity (Wicklow & Espie, 2000) and effortful attempts to control sleep onset (Broomfield & Espie, 2003). Together, these two processes are likely to prevent successful cognitive and somatic de- arousal, which will inhibit the recovery of normal sleep (Espie, 2002). Harvey (2002) makes parallel predictions. In her cognitive model, worries regarding inability to sleep, and the consequent daytime impact of sleep loss, are thought to promote autonomic arousal and anxiety that in turn drive selective attention toward internal– external sleep-related threat cues. Again, it is proposed that sleep disturbance is maintained by an attentional bias favoring sleep-relevant cues.

Second, although differential attentional biases have generally been sought between those who have a diagnosed disorder and those who do not, efforts to understand alcohol consumption variability show that a systematically changing differential attentional bias toward alcohol-related stimuli might be present along the consumption continuum (of use, misuse, abuse, dependence), not just at its clinical pole. For example, a differential attentional bias has been found with (a) “alcoholic” drinkers (using “nonalcoholic” controls; Johnsen, Laberg, Cox, Vaksdal, & Hugdahl, 1994), (b) “problem” drinkers (“nonproblem” controls; Sharma et al., 2001), (c) “heavy” social drinkers (“light” social drinking controls; Bruce & Jones, 2005), and (d) “frequent social” drinkers (“occasional social” drinking controls; Townshend & Duka, 2001). Moreover, even at the clinical pole itself, Ryan (2002) and Jones, Bruce, Livingstone, and Reed (in press) have shown that the extent of attentional bias exhibited by problem drinkers in treatment is positively related to their problem severity. Applying Williams et al.’s (1996) model to the anxiety generated within the context of the negative expectations (or expectancies) of future consumption held by those at the clinical pole (B. T. Jones, 2004; B. T. Jones, Corbin, & Fromme, 2001) helps explain this differential bias.

Moreover, it has been found in social drinkers, using explicit (e.g., Lee, Grealley, & Oei, 1999; McMahon, Jones, & O’Donnell, 1994) and implicit (Gadon, Bruce, McConnachie, & Jones, 2004; Gadon & Jones, 2002; Leigh & Stacy, 1998) methodologies, that the number, range, and severity of negative expectancies increase as consumption increases along the continuum from infrequent and lighter to frequent and heavier drinker—predicting a systematically changing attentional bias along the continuum. It is important to note that the progressive increase in negative expectancies would not necessarily restrain consumption (a macrobehavior) until a threshold was exceeded (B. T. Jones, 2004; B. T. Jones & McMahon, 1998) but that while subthreshold, it would progressively impact on microbehaviors such as emotional Stroop performance. While subthreshold, consumption would be driven by positive expectancies (e.g., Goldman, Del Boca, & Darke, 1999) and because an attentional bias would prime positive expectancies, the apparent paradox is resolved that at levels of social drinking, increases in anxiety caused by increases in negative expectancies would increase consumption.

Little comparable research (either at the clinical pole or along the sleep–problems continuum) has been carried out on sleep-related attentional bias, which is surprising because interest in the control that sleep-related objects might have over sleep behavior is long established (Bootzin, 1972). For example, within a conditioning framework, bedroom environment objects might become discriminative stimuli for sleep (e.g., Bootzin, Epstein, & Wood, 1991), but when the bedroom–sleep contingencies are broken, they might become discriminative stimuli for wakefulness. Meta-analyses have suggested that treatments for insomnia that purport to modify the stimulus control the bedroom environment has over sleep are indeed effective (Morin et al., 1999; Murtagh & Greenwood, 1995; Smith et al., 2002)—to the extent that they are recommended by the American Academy of Sleep Medicine as the standard nonpharmacological intervention (Cheson et al., 1999). Yet, evaluations of the principles of stimulus control within the sleep environment have produced inconsistent data (see Espie, 2002, for a review), which raises the possibility that considerations of stimulus control extend beyond the conditioning framework and that treatments might have other active ingredients.

In an effort to more fully understand the impact that the bedroom environment might have on sleep, this article goes beyond the conditioning framework described above (which addresses one form of stimulus control) to a framework of attentional bias (which addresses another). Two previous studies have explored sleep-related attentional bias using a textual Stroop paradigm. In one, Lundh, Froding, Gyllenhammer, Broman, and Hetta (1997) found that individuals with primary insomnia showed no effect. In the other, Taylor et al. (2003) found an attentional bias in cancer patients with persistent insomnia (12–18 months) as compared with cancer patients with acute insomnia (0–3 months). In the former study, however, only one fourth sleep-related words represented bedroom objects and, in the latter, only 3/20; consequently, although informative, neither study addressed the possible influence of the bedroom environment on sleep.

Rather than use the textual Stroop paradigm, we explore sleep-related attentional bias with digitized objects using a flicker paradigm (Rensink, O’Regan, & Clark, 1997; Simons & Levin, 1997) featuring a perceptual phenomenon called induced change blindness (ICB; Rensink, 2002; Simons, 2000; Simons & Rensink,
This research reveals that when a change is made to a visual scene (and the process of change is hidden from view), it is more difficult to detect than might be expected. Normally in this paradigm, a single feature of a visual scene is changed between successively repeated brief presentations until the change is detected. Change-detection latency is explained by a change’s “grabiness” (O’Regan & Noe, 2000), and this depends not just on the object’s physical features that carry the change but also on the viewer’s history in relation to that object. Using this technique, researchers (B. C. Jones, Jones, Blundell, & Bruce, 2002; B. T. Jones, Jones, Smith, & Copely, 2003) have found differential attentional biases between two levels of social use of alcohol and cannabis. Here, we extend their approach to explore differential attentional biases along the sleep-problems continuum.

We postulate that a systematically changing attentional bias will be found (at different points of the sleep-problems continuum) to exist toward changes made to bedroom environment objects as compared with changes made to other objects. We predict it will be greatest in poorest sleepers (having primary insomnia diagnosed), reduced in moderate sleepers (not having a clinical diagnosis but having more sleep problems than good sleepers), and absent in good sleepers (having no or few sleep problems). We propose that (a) anxiety is a mediator of sleep-related attentional bias at the clinical pole (Espie, 2002; Harvey, 2002), just as with alcohol-related attentional bias at the clinical pole of the consumption continuum, and (b) that different subclinical points of the sleep-problems continuum will generate different levels of anxiety, just as with subclinical levels of alcohol problems. Through Williams et al.’s (1996) model, we predict an increasing sleep-related attentional bias toward bedroom environment objects from good through moderate to poor sleepers. We use a highly representative set of bedroom objects and test this hypothesis with a change carried by the one most representative.

**Method**

**Participants**

Students, staff, and visitors on the campus of a large city university were asked to take part in a 15-min experiment in a nearby quiet room. Each of the opportunistically recruited 302 volunteers were tested singly. Using the procedures described below, we selected 192 for analysis (M age = 32.1 years, SD = 12.6).

**Measures**

Each participant completed a computer task that measured his or her visual change-detection latency (the dependent variable) to changes in photographic scenes described below. They then completed three questionnaires (used for participant exclusion and retrospective group assignment with age-matching). First, the Beck Depression Inventory (BDI; Beck & Steer. 1987) was given, followed by the Pittsburgh Sleep Quality Index (PSQI; Buysse, Reynolds, Monk, Berman, & Kupfer, 1989). The PSQI provides a global measure of sleep quality by aggregating the scores on a 0–3 subscale (3 is the negative pole) of seven different areas of sleep: subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleep medication, and daytime dysfunction. The PSQI is a widely used, internally consistent (Cronbach’s alpha = .83) screening instrument for the detection of significant sleep disturbance (using a threshold score of 6). Recent, independent study has validated this cut-off and confirmed reliability (Cronbach’s alpha = .85, test–retest r = .84; Backhaus, Junghanns, Broocks, Riemann, & Hohagen, 2002). Finally, a local questionnaire was given that collected information on age, gender, and whether individuals had suffered from any psychopathology or other illness known to affect sleep or had any sleep complaint.

The global PSQI score has been shown to be effective in discriminating between nonclinical (0–5) and clinical (6–21) sleepers (e.g., Backhaus et al., 2002), and we use it for this purpose. In addition, however, we seek to explore the possible continuity of attentional bias along the sleep-problems continuum by using the PSQI’s continuous measure of sleep problems to further classify nonclinical sleepers using the end regions (0–2 and 4–5) of the nonclinical PSQI range (0–5). We identify participants as “good” and “moderate” sleepers, respectively, discriminating them from “poor” sleepers and thereby creating three points on the sleep-problems continuum that can be compared. We acknowledge that our concept of moderate sleeper has, perhaps, more limitations than good sleeper, but we justify inclusion of this group for two reasons. First, epidemiological data clearly demonstrate that prevalence of insomnia varies depending on the question asked. Whereas 25–30% of the adult population report dissatisfaction with their sleep, the true prevalence of the clinical disorder is around half that rate, 10–15% (Ford & Kamerow, 1989; Gallup Organization, 1991; Oyahun, Caullet, & Guilleminault, 1997). Second, because (a) there were clear criteria for establishing a poor sleeper category, (b) the distance between the poor sleeper and good sleeper category was substantial, and (c) we wished to test the hypothesis that there would be systematically modified attentional bias across the sleep-problems continuum, we felt that the conceptual delineation of a middle category would be appropriate.

**Design**

The hypothesis tested was that participants with PSQI-indicated sleep problems will detect a change made to one of a collection of sleep-related objects quicker than a change made to one of a collection of neutral objects, as compared with participants with fewer PSQI-indicated sleep problems. An entirely between-participants design was adopted with the following three factors: nature of change to be detected (two levels: sleep-related and neutral), gender (two levels: male and female), and PSQI-indicated sleep quality (three levels: good, moderate, and poor). The dependent variable was change-detection latency.

Sleep problems approximately double from adulthood to later life, and women present about twice as often as men (e.g., Espie, 2002; Ford & Kamerow, 1989; Li, Wing, Ho, & Fong, 2002). Accordingly, groups were matched for age (on an individual basis rather than a group-mean basis), and gender was included as a factor in the design.

A target of approximately 300 participants was estimated to be necessary to allow for the application of the exclusion and matching procedures described below. Each participant was given the flicker ICB change-detection task followed by the BDI, the PSQI, and a local questionnaire. Once recruited into the experiment, participants were randomly assigned to one of four groups generated by crossing the factors of nature of change and gender. Group sizes of 75 or 76 were planned. For each of the four groups, participants who (a) self-reported an illness known to affect sleep (i.e., 2), (b) scored above 9 on the BDI (10–18 indicates moderate-mild depression [Beck & Steer, 1987]; no participants were found to be in this category), or (c) failed to satisfy the task requirements (i.e., to correctly identify the change to which they were exposed—9 participants) were excluded from the analyses. For analyses purposes, the remaining participants of each of the four groups were retrospectively assigned to one of the three levels of the factor of sleep quality, on the basis of their PSQI scores: good sleepers (PSQI score 0–2 inclusive), moderate sleepers (4–5 inclusive), and poor sleepers (> 5). Assignment to the newly formed 12 groups (2 × 2 × 3) was constrained by matching for age (to within 4 years) and the goal of equal group sizes (to obtain the best estimate of between-groups variation). Retrospective group assignment was blind to the dependent variable of the analyses: change-detection latency. Unmatched participants
were excluded from the analyses. Analyses were carried out on the 192 remaining participants, with a group size of 16.

**Apparatus and Stimuli**

An Apple iBook (MacOS 9.1) and the experiment-generation package SuperLab 1.75 (Cedrus Corporation, San Pedro, CA) were used to implement the flicker paradigm. Photographic stimuli were presented centrally and almost filled the iBook’s 28-cm (diagonal) screen. The viewing distance was approximately 65 cm normal to the screen.

A different flicker pair of stimuli were used for each of the two levels of the nature of change factor (sleep-related and neutral). Each of the two pairs contained the same original stimulus (OS) shown in Figure 1. The OS was a full-color photograph (1280 × 960 pixels) taken in natural daylight of seven sleep-related objects and an equal number of neutral objects arranged in two collections on either side of the scene midline. The second stimulus of each pair was identical to the OS but for one small change: a sleep-related change was introduced for the “sleep” level of the nature of change factor (stimulus conditioned stimulus [CS-S]) and a neutral change for the “neutral” level of the same factor (stimulus CS-N). The two changed stimuli are shown in Figure 1 along with their common originating stimulus. Within a flicker paradigm, the two stimuli of a pair (OS/CS-S for the six sleep change groups and OS/CS-N for the six neutral change groups) are presented in continuous succession (each replacing the other, in register, on the iBook screen) until the change from the original to the changed stimulus (or from the changed stimulus to the original stimulus) is detected (see Figure 2). In the current experiment, following usual practice to suppress visual transients produced by the change process, a mask comprising a rectilinear matrix of Xs was presented on the screen between the OS and the changed versions (CS-S or CS-N). The OS and either the CS-S or the CS-N were presented for 250 ms, and the mask was presented for 80 ms with no other interstimulus interval and no intertrial interval (B. C. Jones et al., 2002; B. T. Jones et al., 2003).

Precautions were taken to ensure that the seven sleep-related objects included in the OS were highly representative of the sleep environment and that the object carrying the sole sleep-related change to be detected throughout the experiment was the most representative of the seven. This was done in three stages. First, by asking 60 students (50% female) to list at least 5 objects that they would associate with “going to bed to sleep.” Their lists were compiled to identify the 12 most listed objects. Second, examples of these objects were photographed singly, and the set of 12 objects were embedded in another set of 12 photographs of neutral objects (thought not to be bedroom and sleep-related by the experimenters and none of which had appeared in the 60 lists of sleep-related objects collected earlier). This created a 4 × 6 matrix of photographs of 12 sleep-related and 12 neutral objects. Finally, another 30 students (50% female) were asked to rate the 24 photographs of the objects on a 1–10 “sleep-relatedness” scale (1 = highly sleep-related, 10 = not sleep-related at all). The 7 objects with the highest mean sleep-relatedness score (mean range = 1.2–5.8) were selected for the sleep-related collection of objects in the OS, and the 7 with the lowest mean score (all had means of 10) were selected for the neutral collection.

The sleep-related objects were teddy bear, pajamas, pillow, alarm clock, hot water bottle, hand cream, and pair of slippers. The pair of slippers had the highest sleep-related score. The neutral objects were rucksack, journal issue, files, bottle of ink, office tray, telescoped umbrella, and pair of gloves. The CS-S was made by removing one of the pair of slippers (the most representative sleep-related object) from the OS and photographing the remaining scene, and the CS-N was made by removing one of the pair of gloves from the OS. (In the absence of differential sleep-relatedness scores in this group, this object was chosen because it was physically most similar to the object carrying the sleep-related change.)

In earlier applications of the flicker paradigm to explore attentional biases in the social use of alcohol and cannabis, B. C. Jones et al. (2002) and B. T. Jones et al. (2003) used both normal and mirror-reversal versions of the original and changed stimuli as a between factor. This was done to control for possible carry-over effects from, for example, lifelong reading practices generating side preferences in visual processing. In the four studies they reported, however, B. C. Jones et al. (2002, 2003) found no significant main effects for or interactive effects implicating the factor of normal/mirror-reversal. For this reason, mirror-reversals of stimuli OS and CS-S/CS-N were not included in the current experiment.

**Procedure**

Potential participants were approached throughout the campus and asked to take part in an experiment. Care was taken during the recruitment period
to ensure that the sleep-related nature of the experiment was not apparent. Those who agreed to take part were taken to a nearby quiet room and asked to read the instructions on an iBook screen on what they should expect. Participants were then asked to press the space bar when they were ready, at which time they would see a visual scene presented on the screen for less than a second. It would keep appearing and disappearing for as long as it took them to spot a small change made to the scene between successive presentations. When they had spotted the change, participants were asked to immediately press the space bar, which would be recorded by the computer, and then to say what the change was. Participants were then asked to complete the BDI, the PSQI, and the local questionnaire. It would only have been through reading the contents of the PSQI and the local questionnaire that participants would have been explicitly exposed to the sleep-related nature of the experiment. Once the questionnaires had been completed and passed to the experimenter, the true purpose of the experiment was explained. Participants were also provided with contact information in case they wanted to be informed of the outcome of the project when it was completed.

Results

As indicated earlier, age and BDI-indicated depression associates with sleep problems. Consequently, two analyses of variance (ANOVAs) were carried out prior to the main analyses. A matching-check for age across the 12 groups was carried out using an ANOVA that was identically structured to the main analysis, that is, a three-factor between-participants ANOVA ($N = 192, n = 16$) but with age as the dependent variable and with the following factors and levels: nature of change (sleep-related, neutral), gender (male, female), and sleep quality (poor, moderate, good). With age as the dependent variable, there were no main, two-way, or three-way interactive effects. This indicates a good degree of equivalence in age between the 12 different groups. A second identical ANOVA was carried out with BDI scores as the dependent variable; this analysis also showed no main or interactive effects (overall BDI score: $M = 3.1, SD = 2.1$), indicating a corresponding equivalence.

Analysis 1: ANOVA

The number of flickers to correct change detection for each participant was entered into a three-factor between-participants ANOVA (structured as above; $N = 192, n = 16$). There were no significant main effects. Sleep-related change-detection latencies were no different from neutral change-detection latencies ($Ms = 17.7$ and $19.0$, respectively; $SDs = 12.3$ and $11.1$, respectively), $F(1, 180) = 0.51, p > .05$; women’s change-detection latencies were no different from mens’ ($Ms = 18.2$ and $18.5$; $SDs = 7.9$ and $9.4$, respectively), $F(1, 180) = 0.07, p > .05$; and there were no differences between the change-detection latencies of poor, moderate, and good sleepers ($Ms = 18.4, 16.9$, and $19.8$, respectively; $SDs = 7.8, 7.1$, and $9.7$, respectively), $F(2, 180) = 1.35, p > .05$. Of the one 3-way and three 2-way interactions, only the Change $\times$ Sleep Quality interaction was significant, $F(2, 180) = 7.12, p < .01$ (see Figure 3). The interaction was interpreted using tests for simple main effects. This was done in two ways.

First, for the neutral change, no differences in change-detection latency were found between the poor, moderate, and good sleepers ($Ms = 21.9, 18.4$, and $16.5$, respectively; $SDs = 12.1, 10.9$, and $6.8$, respectively), $F(2, 180) = 1.69, p > .05$. By contrast, differences in change-detection latency were found between poor, moderate, and good sleepers for the sleep-related change ($Ms = 14.5, 15.5$, and $23.1$, respectively; $SDs = 9.4, 10.7$, and $7.6$, respectively), $F(2, 180) = 6.59, p < .01$. These differences were located using $t$ tests. It was found that poor sleepers detected the sleep-related change significantly more quickly than good sleepers ($M = 14.5$ vs. $M = 23.1$; $SDs = 8.5$ vs. $7.6$), $t(191) = 3.33, p < .01$, and moderate sleepers detected it significantly more quickly than good sleepers ($M = 15.5$ vs. $M = 23.1$, $SDs = 9.1$ vs. $10.1$), $t(191) = 2.90, p < .01$. There was no difference in the change-detection latency for the sleep-related change between poor and moderate sleepers, however ($M = 14.5$ and $M = 15.5$, respectively; $SDs = 9.4$ and $10.7$, respectively), $t(191) = 0.34, p > .05$.

Second, poor sleepers detected the sleep-related change significantly more quickly than the neutral change ($Ms = 14.5$ and $21.9$, respectively; $SDs = 9.4$ and $12.1$, respectively), $F(1, 180) = 7.11, p < .01$, displaying a sleep-related attentional bias. Although moderate sleepers showed the same direction as poor sleepers, the difference between sleep-related and neutral change detections was not significant ($Ms = 15.5$ and $18.4$, respectively; $SDs = 10.7$ and $10.9$, respectively), $F(1, 180) = 1.29, p > .05$. By contrast, good sleepers detected the change within the neutral objects significantly more quickly than within the sleep-related objects ($Ms = 16.5$ and $23.1$, respectively; $SDs = 6.8$ and $7.6$, respectively), $F(1, 180) = 6.21, p < .05$, showing a bias toward neutral rather than sleep-related objects. The effect size for poor sleepers (see Figure 4) was significant and “moderate” using Cohen’s (1992) classificatory scheme for effect size comparisons (Cohen’s $d = 0.60$; 95% confidence limits of 0.07 and 1.06) as compared with a “small” and nonsignificant effect size for moderate sleepers ($d = 0.21$; 95% confidence limits of −0.17 and 0.51). The effect size of the opposite bias shown by the good sleepers was significant and “large” (Cohen’s $d = −0.91$; 95% confidence limits of −0.41 and −1.46).

The foregoing is consistent with poor, moderate, and good sleepers, together responding equivalently to the neutral change but responding differently to the sleep-related change. Further, no difference between the neutral and sleep-related change is found for the moderate sleepers but for the poor sleepers, there is a decrease in change-detection latency whereas for good sleepers, there is an increase.

Analysis 2: Regression

Two hierarchical regression analyses were carried out for the two different levels of the nature of change factor (sleep-related and neutral) in order to test the relationship between change-detection latency and a continuous representation of the global PSQI score (rather than a categorical representation, as was used in the ANOVA). With change-detection latency as the dependent variable, age, gender (for reasons outlined earlier), and the BDI score were first added to the regression model and then the PSQI score. Although BDI scores above 9 represent the severity of a clinical diagnosis associated with increasingly poor sleep, this does not necessarily mean that the BDI score range 0–9 can be used to represent states increasingly proximate to a clinical diagnosis (and with commensurate associations with sleep quality). Nevertheless, entering the “subclinical” BDI score into the regression model prior to the PSQI score appears to be a sensible precaution. The incremental variance explained when the PSQI score is added to
the model already containing age, gender, and the BDI score is the critical test of the relationship.

In both models (sleep-related change and the neutral change), there was no significant variance explained in change-detection latency when age, gender, and the BDI scores were added. When the PSQI score was added to the model for the sleep-related change, however, the model deviated from the null, $F(4, 92) = 2.51, p < .05$. The incremental variance explained on the basis of adjusted $R^2$ was 10.6% ($p < .05$), and the beta for PSQI was $0.310$ ($p < .05$). For the neutral change, the model did not deviate from the null when the PSQI score was added, $F(4, 92) = 2.29, p > .05$.

Thus, there is no relationship between PSQI-indicated sleep problems and change-detection latency for the neutral change, but for the sleep-related change, change-detection latency decreases as sleep problems increase.

**Discussion**

Espie’s (2002) and Harvey’s (2002) models of primary insomnia identify attentional bias toward sleep-related concepts as contributing to the perpetuation of primary insomnia. In both models, the heightened arousal accompanying insomnia and the belief that the night’s sleep problems will negatively impact on next day’s activities generate anxiety. Williams et al. (1996) postulated that anxiety raises the resting activity level of network inputs representing concepts with which the anxiety temporally occurs. The raised resting activity level is the basis of the attentional bias we predict will be found for bedroom objects in primary insomnia. This means that representations of the bedroom environment will be more likely to enter current cognitions, generate anxiety, and disrupt sleep.

Our results support this prediction, showing an attentional bias toward a representative bedroom object in sleepers who satisfy the minimal criteria for primary insomnia (our poor sleepers); change-detection latencies in poor sleepers were quicker when the change was made to the sleep-related object than to the neutral object. There was also found a marked differential attentional bias between poor sleepers and the good-sleeping controls. However, whereas the poor sleepers showed a bias toward the sleep-related object as predicted, the good sleepers showed a bias toward the neutral object. This part of our prediction, therefore, does not appear to be supported—namely, that because good sleepers would be reporting few if any sleep problems, there would be little anxiety generated within the sleep-problems context, negligible changes in the resting activity levels of the sleep-related network input units, and therefore a negligible sleep-related attentional bias. There are two explanations why the good-sleeping component of the differential attentional bias might occur.

First, it is possible that the good sleepers’ choices are driven by the physical properties (physical saliencies) of the sleep-related and neutral objects themselves, rather than semantic saliencies. Particularly in complex stimuli (e.g., real-world objects collected
into a scene), values of the objects’ physical properties will compete for attention resources and contribute to, or even drive, what is attended to and what reaches consciousness. Physical properties such as object size, color, brightness, complexity, and overlap as well as relative positional and configurational aspects will be important in this respect. This means that any single attentional bias measure (e.g., the comparison between the change-detection latencies of the group of good sleepers given the sleep-related change with the other group of good sleepers given the neutral change) is ambiguous because it confounds the sleep salience manipulation (i.e., the sleep-related change vs. the neutral change) with a range of other nonsleep saliences. If the good sleepers have comparatively problem-free sleep (a group assignment feature of the design), there will be little associated anxiety and, consequently, negligible sleep-related attentional bias. In addition, in such a case, any attentional bias would be driven by the physical saliencies of the grouped objects. We would conclude, therefore, that either the group of neutral objects in which the neutral change was embedded, or the neutral objects that carry the neutral change had more “grabby” physical properties than did the corresponding sleep-related objects; this would explain the neutral attentional bias shown by the good sleepers. When measuring differential attentional bias, however (i.e., the difference in attentional bias between poor sleepers and good sleepers), we are able to control the physical saliencies (and any other nonsleep semantic saliencies) through giving the poor and good sleeping groups the same sleep.
stimulus presentations. The interaction representing the differential attentional bias between poor and good sleepers is, therefore, the properly controlled measure of the effect of the PSQI-derived sleep-problem manipulation on sleep-related attentional bias. Indeed, it follows from this explanation that the apparent sleep-related attentional bias shown by the poor sleepers will be an underestimate of the real sleep-related attentional bias and will only be provided through the measure of differential attentional bias.

Second, there is an alternative explanation for the neutral bias found in good sleepers that makes no reference to physical saliencies. It is possible that our good sleepers were low in anxiety-sensitivity (Reiss, 1997), given that, using a dot probe paradigm, Keogh et al. (2001) have shown that participants with low anxiety-sensitivity under some circumstances orient away from threatening stimuli rather than toward them—in which case, our good sleepers should orient away from the set of bedroom objects if they were low in anxiety-sensitivity. If this occurred, it would assist neutral change detection but hinder sleep-related change detection and explain the bias toward the neutral set of stimuli that good sleepers show. Although there might be grounds for suggesting that good sleepers might indeed be low in trait anxiety (because this disposition might either contribute to or derive from their good-sleeping status), there is less to suggest that they might be low in anxiety-sensitivity and, as Keogh et al. noted, it is the latter rather than the former that would be an important feature of this type of explanation. Nevertheless, it remains a possibility.

However the differential attentional bias between two poles of the sleep-problems continuum is explained, it remains that the poor sleepers appear to attend to a sleep-related object more than a neutral object. If our finding with one sleep object generalizes to others, the attentional bias that it represents will feed the processes that maintain or perpetuate primary insomnia described by Espie (2002) and Harvey (2002). There is some indication that attentional bias might be an agent for not just the perpetuation of primary insomnia but also its precipitation—this possibility is raised through a consideration of the moderate sleepers’ performance. On the surface, the moderate sleepers do not appear to show an attentional bias toward the sleep-related object as compared with the neutral object. If, however, the explanation of the performance of the good sleepers is indeed based on physical saliencies (as described above), then the good-sleepers performance represents a baseline condition against which the performance of the moderate sleepers can be judged, with all nonsleep saliencies controlled. When this comparison is made, good and moderate sleepers do not treat the neutral change differently (and neither do poor sleepers); by critical contrast, however, the moderate sleepers detect the sleep-related change significantly more quickly than do the good sleepers. The apparent lack of attentional bias in moderate sleepers, derived from comparing only their detection latencies to the sleep-related and neutral change, should not obscure the fact that a differential attentional bias is revealed from good to moderate sleepers when more appropriately controlled comparisons are made. If moderate sleepers do indeed exhibit a sleep-related attentional bias—generated by the anxiety accompanying acute sleep problems, not diagnosed as the more chronic primary insomnia—it could help maintain these sleep problems in much the same way as Espie (2002) and Harvey (2002) suggested that attentional bias maintains primary insomnia.

If sleep problems are maintained beyond an acute phase by attentional bias, however, and if the length of time satisfies the criterion for primary insomnia, it suggests that attentional bias might not only be implicated in perpetuating primary insomnia but also in precipitating it.

Our study is the first to report a sleep-related differential attentional bias between good and poor sleepers and a possible corresponding differential bias between good and moderate sleepers. It also identifies a likely source perpetuating primary insomnia and a possible precipitating factor. We find only partial support for a continuity of sleep-related attentional bias corresponding to the sleep-problems’ continuum, however, because of the failure to find a differential attentional bias between the moderate and poor sleepers.

Our findings and conclusions are limited by a number of considerations. First, the clinical pole (primary insomnia) was represented by participants with PSQI scores greater than 5. The incorporation in future research of an additional “primary insomnia” group with a higher inclusion threshold would provide an additional opportunity to compare more distant clinical and subclinical points on the sleep-problems continuum as well as points within the diagnostic category—providing a more extensive test of the hypothesis of continuity of attentional bias. Second, only a single sleep-related change and neutral change were used, and this might compromise generalizing the findings and conclusions to other bedroom and neutral objects. Care was taken, however, to ensure that all the objects used were either highly representative of the bedroom environment or not representative at all. Further, the most representative bedroom object was chosen to carry the sleep change. Finally, Bruce and Jones (2005) have recently shown with social drinkers that change detection performance in a flicker paradigm is almost entirely driven by the set of different objects in which the single object carrying the change (also a set member) is embedded rather than by the single object itself. If this finding extends to our sleep study (which uses similar stimulus configurations), this limitation is reduced because seven sleep objects make up the sleep set and another seven make up the neutral set. Nevertheless, our finding requires replication, and future research should extend the range of objects used. Furthermore, we do not have anxiety and anxiety-sensitivity data, and the possibility that good sleepers low in anxiety sensitivity might be orienting away from the sleep-related objects cannot be evaluated. Future research needs to pay attention to this potential explanatory source by collecting anxiety data.

Although it is some 30 years since stimulus control was proposed as offering an explanatory, and potentially modifiable, paradigm in insomnia (Bootzin, 1972), contemporary sources agree that its role in the development and amelioration of persistent insomnia remains unclear (Edinger & Wohlgemuth, 1999; Espie, 2002; Morin et al., 1999). In large part, this is because research effort has focused on treatment efficacy derived from stimulus control intervention rather than on experimental studies. We are now in the position where a psychological rather than pharmacological approach is seen as the treatment of choice for persistent primary insomnia (Espie, 1999; Smith et al., 2002) and stimulus control instructions meet American Academy of Sleep Medicine criteria for “standard treatment” (Chesson et al., 1999). The present study, which uses a novel experimental approach well suited to the systematic investigation of the stimulus control par-
adigm, illustrates one way forward for research in this area. Evidence presented of attentional bias toward the bedroom environment, and our previously reported evidence of attentional bias toward textual representations of sleep and sleeplessness (Taylor et al., 2003), raises the possibility that dysfuntional stimulus control in insomnia develops from the conditioning of nonverbal and verbal signals as threat cues that impact on selective attention through the principles of Williams et al.'s (1996) model—starting a vicious cycle. The feasibility of objectively and accurately measuring this bias, therefore, yields new opportunity for both experimental and clinical investigations of the development of primary insomnia during remission and treatment response.

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