Biased Processing of Sleep-Related Stimuli in Children of Parents With Insomnia

Jason G. Ellis, Amy Thomson, Alice M. Gregory & Annette Sterr

Northumbria Center for Sleep Research, School of Life Sciences, Northumbria University, Newcastle-upon-Tyne, England
Department of Psychological Medicine, University of Glasgow, Glasgow, Scotland
Department of Psychology, Goldsmiths, University of London, London, England
Department of Psychology, University of Surrey, Guildford, England

Accepted author version posted online: 02 Jan 2012. Published online: 12 Nov 2012.

To cite this article: Jason G. Ellis, Amy Thomson, Alice M. Gregory & Annette Sterr (2013) Biased Processing of Sleep-Related Stimuli in Children of Parents With Insomnia, Behavioral Sleep Medicine, 11:2, 108-119, DOI: 10.1080/15402002.2011.634050

To link to this article: http://dx.doi.org/10.1080/15402002.2011.634050
Disorder-specific cognitive biases have been observed in children whose parents suffer from psychological disorders. Despite those same biases being observed in individuals with insomnia, they have yet to be explored as an index of vulnerability in children of parents with insomnia. It was hypothesized that potentially vulnerable children would demonstrate cognitive biases to sleep-related cues, relative to controls. Following a “tired-state induction,” a sleep-related Emotional Stroop was completed by 2 groups: 38 children of parents with insomnia and 51 controls. Children also reported their observations about the content of the Stroop words. Results showed an attention bias in children whose parents have insomnia, but no interpretive bias. The results are discussed in terms of a predispositional vulnerability to insomnia.
It has long been established that individuals with a range of mental and emotional disorders display cognitive biases for stimuli relevant to their condition (for a comprehensive review, see Mathews & MacLeod, 2005). Despite these findings, the concept of sleep-related “cognitive biases” has only recently been investigated (Ellis, Mitchell, & Hogh, 2007). As such, research using computerized cognitive tasks, such as the Emotional Stroop (Gotlib & McCann, 1984) and Dot-Probe Task (MacLeod, Mathews, & Tata, 1986), has provided strong and conclusive support for an attention bias in people with insomnia (Jones, MacPhee, Broomfield, Jones, & Espie, 2005; Spiegelhalder, Espie, Nissen, & Riemann, 2008; Taylor, Espie, & White, 2003).

One of the questions critical to the potential mechanisms of an attention bias in insomnia concerns the role of physiological and psychological characteristics inherent to the condition (Spiegelhalder, Espie, & Riemann, 2009). Studies on individuals with delayed sleep phase syndrome (DSPS)—a disorder characterized by a physiological sleep disturbance with a minimal, if nonexistent, psychological pathway—have found no attention bias in this group (MacMahon, Broomfield, & Espie, 2006; Marchetti, Biello, Broomfield, MacMahon, & Espie, 2006). This absence of an attention bias effect in DSPS suggests that a physiological sleep disturbance alone is insufficient to elicit a response in terms of biased information processing. This interpretation is further supported by sleep deprivation studies showing sleep-related attention bias remains stable when normally sleeping participants are deprived of sleep for 36 hr (Sagaspe et al., 2006). In addition, it has been shown that polysomnographic parameters of poor sleep are not correlated with attention bias scores in people with insomnia (Spiegelhalder et al., 2010). Together, these data suggest that the attention bias in insomnia is primarily the result of psychological processes related to increased preoccupations about sleep and the daytime sleepiness (Espie, 2007).

Insomnia has also been investigated for interpretive biases, as another form of cognitive bias, albeit to a much smaller extent. So far, three studies have studied whether people with poor sleep are more likely to interpret ambiguous cues as sleep-related as opposed to non-sleep-related (Ellis, Gardani, & Hogh, 2010; Ree & Harvey, 2006; Ree, Pollitt, & Harvey, 2006). Again, the issue of teasing apart the physiological from psychological aspects of the insomnia, in relation to an interpretive bias, has been further examined. A study by Ree et al. showed an interpretive bias in people with poor sleep after controlling for levels of sleepiness and anxiety. In addition, Ree and Harvey demonstrated that levels of sleeplessness mediated the intensity of the interpretive bias. Together, these studies provide suggestive evidence that both the psychological (sleepiness and anxiety) and physiological (sleeplessness) aspects of the insomnia contribute to the interpretive bias.

Research in the area of mental and emotional disorders has become increasingly focused toward factors that make some more vulnerable to mental health problems than others. It is widely accepted that many mental and emotional disorders have a strong familial link (e.g., depression and bipolar disorder; Garber & Robinson, 1997; Goodman & Tully, 2008). In this context, children of parents with these disorders have been shown to be vulnerable for developing the disorder themselves and cognitive bias tasks have been explored as an index of this intergenerational vulnerability (Gibb, Benas, Grassia, & McGearry, 2009; Gotlib, Traill, Montoya, Joormann, & Chang, 2005; Kujawa et al., 2011; Schneider, Unnewehr, Florin, & Magraf, 2002). These studies are largely conclusive, although the strength and direction (avoiding vs. attending to the relevant stimuli) of the attention bias appears to be governed by an exposure to a salient state induction (i.e., a primer that activates relevant negative cognitive schemas that are thought to lie dormant) prior to the task (Joormann, Talbot, &
Gotlib, 2007; Mathews, Ridgeway, & Williamson, 1996; Murray, Woolgar, Cooper, & Hipwell, 2001; Schneider, Unnewehr, In-Albon, & Margraf, 2008). In terms of interpretive biases as an index of vulnerability toward other mental and emotional disorders, the evidence is scarce, although one study has shown that daughters of depressed mothers were more likely to interpret ambiguous stimuli as more negative, following a negative mood induction, than daughters of mothers who had never suffered from depression (Dearing & Gotlib, 2009). These combined findings do suggest that this change in the processing of disorder-relevant stimuli, as indexed by cognitive biases, may be generalizable across emotional and mental conditions and contribute to the mechanism of an intergenerational vulnerability. If this assumption is indeed correct, one could postulate that all disorders that are characterized by cognitive biases are likely to be subject to intergenerational vulnerability. With this study, we aimed to explore this possibility by investigating cognitive biases in children of parents suffering from insomnia. Specifically, this study used a “tired-state induction” in combination with an Emotional Stroop task to test the hypothesis that children whose parents have insomnia would demonstrate both attention bias and an interpretive bias toward sleep-related cues, relative to children whose parents do not have insomnia.

METHOD

Recruitment

Recruitment took place at the Glasgow Science Center (UK) over an 8-week period (February–March). Information leaflets were available at a recruitment stall for individuals to read and consider participation. The first section of the leaflet contained an initial self-screen to ensure parents in each group met the criteria for the inclusion of their own and their child’s data. The leaflet explained that potential participants were excluded if they, or their partner, had a history of or current sleep disorder other than insomnia or if any of the presenting child’s siblings had any sleep disorder. Insomnia was described to potential participants as a current sleep problem in getting to sleep, staying asleep, or waking earlier than they would like to for no apparent reason, which was causing distress, occurred at least 3 nights per week and had been present for at least 6 months—that is, based on Diagnostic and Statistical Manual of Mental Disorders (4th ed. [DSM–IV]; American Psychiatric Association, 1994) criteria for primary insomnia. Descriptions of the central features (taken from the DSM–IV) of five other sleep disorder types (i.e., breathing-related sleep disorders, parasomnias, restless legs syndrome, narcolepsy, and circadian rhythm disorders) were provided if a parent was unsure of their or their family’s sleep status. The second section of the leaflet outlined exclusion criteria to parents regarding the child taking part in the experiment. These were a reported diagnosis of color-blindness, a learning disability, attention deficit hyperactivity disorder (ADHD), or dyslexia. In addition, the child had to be between 9 and 12 years of age to be included (the age range was chosen to reflect the previous literature on intergenerational vulnerabilities for mental and emotional disorders). Parents were not asked about sleep problems in the child taking part in the experiment. If eligible, and interested, parents were asked to complete a checklist confirming that they did not meet any of the exclusion criteria for themselves or their family. Written informed consent was obtained from the parent, and assent was provided by the child. If a parent had more
than one child who was eligible and eager to take part, then all the children were allowed to complete the experiment; however, data from one child per family was included in the analysis. The choice of which child’s data to include, if from the same family, was randomly selected prior to testing. The protocol was approved by the University of Glasgow ethics committee.

Participants

A total of 94 parent–child pairs were initially recruited to the study. The child’s Stroop response data was examined for each participant individually. Errors and outliers (i.e., reaction times [RTs] of $< 300$ msec and $> 2,000$ msec) were excluded from the analysis based on recommendations from previous Stroop research (McNally et al., 1994; Taylor et al., 2003). These exclusions resulted in a loss of five datasets. The final sample was 89 children, predominately Caucasian (97.8%), and comprised of 51 (57.3%) girls and 38 boys aged 9 to 12 years (age, $M = 10.3$ years, $SD = 1.05$). In addition, 89 parents (55 women; 61.79%; age, $M = 41.45$ years, $SD = 4.60$) were included in the study. The children were allocated into two groups: those with a parent with a current episode of DSM–IV-defined insomnia (either from the presenting parent or reports regarding a partner in the household) and a control group with no parental history of problems with their own, their partner’s, or any of their other children’s sleep (32 girls and 19 boys). The final sample comprised 38 children of parents with insomnia (26 girls; 68.4%) and 51 children (26 girls; 50.98%) in the control group. In terms of the parents with insomnia, 36 of the 38 parents reported that they had insomnia themselves (23 mothers and 13 fathers) of which 4 parents reported having insomnia and also having a partner with insomnia. Two parents reported sleeping normally themselves, but having a partner (both men) with insomnia.

Procedure

Children were individually tested; the test took place in an enclosed computer area by a trained psychologist. The testing protocol for the children consisted of three consecutive stages: the tired-state induction, the Emotional Stroop task, and the self-report scales. For the tired-state induction phase, children first completed the Stanford Sleepiness Scale (SSS; Hoddes, Zarcone, & Dement, 1972) to obtain pre-induction sleepiness scores. Then, a series of progressive muscle relaxation, yawning, and breathing exercises (Matsumoto & Smith, 2001) were administered for 10 min, which was followed by a re-rating of the SSS to assess post-induction sleepiness. Children were told that they were completing a series of exercises to prepare them for the computerized experiment. Next, the Emotional Stroop was performed. The latter consisted of a practice trial of 20 words to ensure that children could read the words, determine the different colors, and correctly use the response box. The main experiment was comprised of 40 target words randomly presented in four colors (red, blue, yellow, and green) on screen. During the test, children wore earphones to reduce auditory interference. After the Stroop test, children completed an interview about the test itself. For this, they were first asked whether they noticed anything about the words and, if so, what? Subsequently, the Sleep Self-Report (SSR; Owens, Maxim, Nobile, McGuinn, & Msall, 2000) was administered. Finally, children were asked whether they were worried about either of their parent’s sleep or their own sleep.
During data collection with the children, parents completed a demographic questionnaire and the diagnostic screening interview covering additional diagnostic criteria for DSM–IV-defined insomnia for themselves and their family, to confirm group allocation. Upon completion, children and parents were debriefed together, and children were provided with token rewards (novelty stickers) for their participation.

Measures

**Stanford Sleepiness Scale (SSS).** The SSS was used to examine the effectiveness of the tired-state induction. The scale contains seven statements through which people rate their current level of alertness/sleepiness (e.g., 1 = “feeling ... wide awake” to 7 = “... sleep onset soon ...”). Scores range from 7 to 49, with higher scores indicating higher levels of sleepiness.

**Emotional Stroop—Child version.** As with the original Stroop task, the Emotional Stroop involves asking participants to name the color that words appear in on the screen. Unlike the words in the standard Stroop, Emotional Stroop words are selected to be either neutral or disorder-related. The words had been chosen based on previous research using an Emotional Stroop to examine a sleep-related attention bias (Taylor et al., 2003), and the words were piloted on a class of children aged 9 to 12 to ensure they were age appropriate (e.g., night, bedtime, dream, tree, bottle, and jumper). It is thought that mechanisms of selective attention will drive the preferential processing of concern-related information (i.e., in the content of the word) in such a way that interferes with performance on the color-naming task; thus, individuals will take longer to respond correctly to disorder-related words. In this case, interference scores were used as an index of the attention bias, and were calculated by subtracting the mean RT from all the sleep words against the mean RT from all the neutral words (i.e., MeanRTSleep – MeanRTNeutral). In these instances, higher positive scores indicated more interference or a larger attention bias.

**SSR.** The SSR was used to control for potential child sleep problems in the analysis. The SSR is a 33-item questionnaire designed for completion by children aged 7 to 12 years, which assesses four domains: (a) difficulty going to bed and falling asleep, (b) sleep duration, (c) night awakenings, and (d) daytime sleepiness. Total scores range from 13 to 39, with higher scores indicating more problematic sleep. The reliability of the measure in this sample was acceptable (Cronbach’s α = 0.73).

**Diagnostic screening interview.** The diagnostic screening interview was used to provide additional information from participants in terms of the presence of insomnia, particularly when reporting on behalf of a family member. Participants were asked a series of 11 questions that elaborated on differentiating the symptoms of insomnia from other sleep disorders (e.g., a self-reported sleep onset latency of 30 min or more not due to unpleasant or uncomfortable sensations in the legs, nocturnal awakenings accompanied by gasping for breath, or a sustained period of sleeping outside desired times, despite adequate opportunity). Of the parents who initially self-reported having insomnia, 100% still met the criteria for insomnia from the screening interview. In addition, all six parents who were reported to have insomnia by their
partner still met the criteria after the screening interview. No participants were excluded or reallocated to a different group on the basis of the screening interview. Parents were finally asked whether they were concerned about the sleep of their child that was taking part in the experiment (“yes/no” response format).

**Interpretive bias data.** To examine the presence of an interpretive bias, the children were asked, “Did you notice anything about the words [presented in the Stroop task]?” If the child said “Yes,” they were then asked, “What did you notice about the words?” To be classified as a positive interpretive bias response (yes vs. no), the child had to respond positively to the first question and state that the words related to sleep, or a derivative term (e.g., bedtime or nighttime). Where ambiguous answers were provided (e.g., daytime), the children were asked to recall the words that related to the category that they had noticed, and this was deemed as an interpretive bias if the majority of the words provided by the child were sleep-related words from the Stroop.

**RESULTS**

Analysis of the demographic data and the children’s reports of their own sleep revealed no differences between the children of parents with insomnia (age, $M = 10.29 \pm 1.09$) and the control group (age, $M = 10.32 \pm 1.03$) in age, $t(87) = 0.13$, ns; or gender, $\chi^2(1, N = 89) = 2.73$, ns. The percentage of parents reporting concerns about their child’s sleep was higher in the insomnia group (21.1%) compared to controls (13.7%), but this difference was insignificant, $\chi^2(1, N = 89) = 0.83$, ns. The percentage of children reporting worry about their own sleep was comparable, with 21.6% for children of parents with insomnia and 21.1% in controls, $\chi^2(1, N = 89) = 0.01$, ns. In addition, there was no significant difference in terms of parents’ age between the two groups (mean age of parents with insomnia = 42.16 ± 4.87; mean age of control parents = 41.26 ± 4.74), $t(87) = -0.88$, ns. Finally, mean SSR scores for children with parents with insomnia were 19.24 ± 3.97 and 20.05 ± 4.47 for controls, and did not significantly differ, $t(87) = 0.91$, ns (see Table 1).

**TABLE 1**

<table>
<thead>
<tr>
<th>Characteristics and Scores of Participants by Grouping</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Children of Parents</strong></td>
</tr>
<tr>
<td>With Insomnia$^a$</td>
</tr>
<tr>
<td>M</td>
</tr>
<tr>
<td>---</td>
</tr>
<tr>
<td>Child’s SSR total score</td>
</tr>
<tr>
<td>SSS pre-state induction</td>
</tr>
<tr>
<td>SSS post-state induction</td>
</tr>
</tbody>
</table>

*Note.* SSR = Sleep Self-Report; SSS = Stanford Sleepiness Scale.

$^a_{n = 38}$, $^b_{n = 51}$. 

---

\[\sum\text{Note}\]
Children’s worries about their parent’s sleep were comparable between groups (9.8% control vs. 21.1% children of parents with insomnia), $\chi^2(1, N = 89) = 2.21, ns$, suggesting that any differences between the groups was not a result of current concern over their parents sleep.

Tired-State Induction

There were no significant between-group differences on the SSS either pre-tired-state induction, $t(87) = -0.62, ns$; or post-tired-state induction, $t(87) = -0.78, ns$. To examine the effectiveness of the state induction, a repeated-measures $t$ test was used, $t(87) = 6.56, p < .001$. This showed that the tired-state induction had been effective in both the children of parents with insomnia group, $t(50) = 5.11, p < .001$; and the control group, $t(37) = 4.08, p < .001$. Children in both groups reported being significantly sleepier following the state induction, although in terms of actual increases in scores on the SSS, this was small (see Table 1).

Stroop Data

Control children responded to neutral stimuli within $954.63 \pm 213.93$ msec and sleep-related stimuli within $942.36 \pm 194.01$ msec, whereas children of parents with insomnia responded to neutral stimuli within $949.44 \pm 187.02$ msec and sleep-related stimuli within $970.82 \pm 187.02$ msec (see Figure 1). A one-way, between-groups ANCOVA, controlling for child’s age, gender, and self-reported sleep problems (SSR scores), was used to determine whether

![Figure 1](image-url)  
**FIGURE 1** Mean response latencies to neutral and sleep-related words.
there were differences in interference scores by group (children of parents with insomnia vs. controls). There was a significant group difference, $F(1, 87) = 4.03$, $p < .05$; with higher interference scores in children of parents with insomnia ($21.38 \pm 114.52$ msec) than controls ($-12.27 \pm 91.73$), although the variance explained was small (partial $\eta^2 = .05$).

**Children’s Interpretive Bias**

When asked, the majority of children from both groups (92%) reported noticing something about the words. When prompted, the children detailed a variety of different word characteristics, including the following: “They were different colors,” “One of them was tree,” “Some of them were about sleep,” and “They were about bedtime.” Overall, 68.4% of children of parents with insomnia and 60.8% of the control children reported that the words were about sleep. A Group (parental insomnia status) $\times$ Reported Sleep Content (yes or no) chi-square test did not reach statistical significance, $\chi^2(1, N = 89) = 0.55$, ns.

**DISCUSSION**

Despite a growing literature suggesting that (a) attention and interpretive biases can be observed within both clinical and non-clinical samples of people with insomnia, and (b) cognitive biases are evident in children of parents with a range of other mental and emotional disorders, to date, there have been no studies that have examined whether children of parents with insomnia have cognitive biases toward sleep. Based on the assumption that the mechanisms of vulnerability, indexed by cognitive biases, may apply in insomnia, we hypothesized that attention to sleep-related stimuli and biased interpretation of stimuli as sleep-related would be evident in children of parents with insomnia. To test this hypothesis, we used a mixed design, where cognitive biases were assessed following a tired-state induction in children with parents with insomnia and controls. Data obtained through the Emotional Stroop confirmed that children of parents with insomnia show greater interference effects than controls. The effects in the Stroop task, therefore, suggest that an attention bias for sleep exists in children whose parents have insomnia. Based on attention bias research in other health conditions, we propose that the attention bias observed in the offspring of insomnia patients is a potential index of vulnerability in this group. Although further research is clearly necessary to ascertain the latter proposition, the results of this study are interesting not only because they demonstrate attention bias in children of parents with insomnia for the first time, but also because this finding holds the potential for novel preventative intervention strategies. For example, it is conceivable that children identified as vulnerable for insomnia, by a cognitive bias index, could be given a series of management tools to circumvent the potential transition from acute to chronic insomnia. However, it must first be established, presumably through longitudinal research, whether children who demonstrate this attention bias go on to develop insomnia.

In contrast to our findings on attention bias, our hypothesis that children of parents with insomnia would also show an interpretive bias was not confirmed. Thus, we found no differences between children of parents with insomnia and controls. There are two possible explanations for this finding: The interpretive bias seen in children “at risk” of depression (Dearing & Gotlib,
Elliott, Thomson, Gregory, and Sterr (2009) does not transfer within the context of insomnia, or the sleepiness induction was not sensitive enough to elicit or detect an interpretive bias.

This study suggests that cognitive biases might transfer from parent to child. Based on the theory of intergenerational transference by Beck (1967), one could postulate that this transfer may be mediated by disordered schemata that are developed through observing the signs and symptoms of the disorder in the parent. Although this mechanism of transfer is well-documented and plausible in the case of depression, it may not be so apparent in insomnia. For example, insomnia may not impact on a parent’s behavior to an extent that would interfere with their functioning within the family and, therefore, be evident to their child. A parent’s struggle to initiate and maintain sleep may be confined to a time when their children are already asleep. Therefore, the children may simply not be provided with sufficient observations of their parent’s disorder from which to develop these disorder-related schemata. This is an interesting point, especially when considering that only a small percentage (21.1%) of the children of parents with insomnia reported being worried about their parent’s sleep, despite 100% of the families in the insomnia group having at least one parent who met the DSM–IV diagnosis of insomnia and 10.5% of the families in the insomnia group reporting both parents having insomnia. The main contention to this explanation is that unlike the non-significant interpretive bias, a significant, albeit small, attention bias was found. Here, an interesting distinction emerges that may explain these differences. Unlike the nighttime symptoms of the insomnia, which may not be observable because the child would be asleep, it is more likely that the child would be exposed to the daytime sleepiness and other daytime symptoms experienced by the parent. Going back to the differences between the attention biases and interpretive biases observed in the insomnia literature, whereas both sleeplessness and sleepiness contribute to an interpretive bias (Ree & Harvey, 2006; Ree et al., 2006), levels of sleeplessness are unrelated to an attention bias (Marchetti et al., 2006; Sagaspe et al., 2006). As such, it may be that intergenerational cognitive biases work in the same way in that, unless children are witness to the actual sleep loss of their parents (i.e., the signs of the disorder), there will be no observable interpretive bias. Here, it would have been interesting to do a subgroup analysis of the attention and interpretive bias scores based on the amount of time the children spent with a parent who had insomnia compared to the parent that did not (or in 4 cases, both parents with insomnia); however, the sample size precluded this level of analysis.

The main methodological issue that could explain the absence of an interpretive bias is the tired-state induction. Where previous studies on cognitive vulnerability in children have indicated that the inclusion of a relevant mood induction is important (Schneider et al., 2008), this study used a tired-state induction. The tired-state was chosen to activate the salient trigger representation of insomnia by mimicking the main reported daytime symptom (i.e., sleepiness; Ellis et al., 2007). To create this sleepiness, a set of progressive muscle relaxation, breathing, and yawning exercises were used due to their applicability and evidenced use in inducing sleepiness in young adults (Matsumoto & Smith, 2001) and reducing physiological and psychological arousal in therapeutic interventions with children (Christophersen & Mortweet, 2001). This manipulation appeared to have worked, as demonstrated by the increased scores on the SSS. That said, the post-induction scores were not outside the “normal” range seen in adult samples; and the SSS has, to our knowledge, never been used with this age group. As such, it is difficult to ascertain whether the scale was valid in this sample and whether the observed increases were, in fact, meaningful (Hoban & Chervin, 2001). However, modest correlations...
between actigraphically defined sleep and self-reported sleepiness, as measured on a 4-point scale ranging from 1 (very alert) to 4 (very sleepy), which is broadly similar in structure to the SSS, has been demonstrated in 7- to 11-year-old children (Sadeh, Raviv, & Gruber, 2000). Future research may wish to explore the assessment of children's sleepiness, within the context of cognitive biases, using more objective measures of sleepiness, such as the Multiple Sleep Latency Test. There also remains the broader question of whether increasing sleepiness is the most appropriate method to elicit an interpretive bias. As insomnia develops, it may not be that sleepiness is the most salient component of the acute sleep disturbance. It may be something else (e.g., increased sleep preoccupation or increased anxiety over the sleep loss). To account for this in future research, it may be worth exploring a manipulation that not only increased sleepiness, but one that also creates anxiety. That said, it may simply be the case that an interpretive bias is not a marker for a vulnerability to insomnia; and, where the tired-state induction activated cognitive schemata, as evidenced by the changes in attention bias scores, there is simply no relation between interpretive biases and cognitive schema in insomnia.

This study should be viewed in light of its limitations. Primarily, parents were asked to self-report on their partner’s sleep history, as well as their own. It may be that some parents reported their partner as never having had an episode of insomnia when, in fact, they may have; or, they may have reported the partner as not having a current episode of insomnia when, in fact, the partner currently did. Conversely, participants may have reported their partners as having insomnia when, in fact, they did not. That said, if it were the case that some individuals had been misclassified into the control group, then it would be more likely to skew the results against an observed attention bias in the children of parents with insomnia group. In addition, as only two parents with insomnia were identified as such by a normal sleeping partner, the impact of misclassification in these cases would have been small. Further, all of the adult participants went through several screening procedures to ensure group allocation was as “pure” as possible. It could also be suggested that as children with sleep problems were not excluded, the child’s own sleep status may explain the observed attention bias. However, the child’s sleep was controlled for in the analysis, and there were no between-group differences on SSR scores. In addition, as scores on the SSR were similar in both groups and of a level normally observed in good-sleeping children (Gregory et al., 2008), it is unlikely that this was the case. Finally, the use of the term worried may have created confusion when the children were asked to self-report issues with their own and their parents’ sleep. A child may believe their parent to have a sleep problem, but not have been worried by this. That said, as the same wording was used with both groups of children, any ambiguity would have equally influenced reporting patterns.

Overall, the results demonstrate that there was an attention, but not interpretive, bias in children whose parents have insomnia. Although these findings provide a preliminary indication of a method to assess a potential pre-existing vulnerability to insomnia, replication is needed to ensure the validity of the findings.

**ACKNOWLEDGMENT**

Alice M. Gregory is currently supported by a Leverhulme Research Fellowship.
REFERENCES


