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# Correcting distorted perception of sleep in insomnia: a novel behavioural experiment?

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## Abstract

Patients with primary insomnia overestimate their sleep onset latency (SOL) and underestimate their total sleep time (TST). The present study aimed to test the utility of a novel behavioural experiment designed to correct distorted perception of sleep among patients diagnosed with primary insomnia. Individuals with primary insomnia were asked to wear an actigraph and keep a sleep diary for three nights. On the following day, half were shown the discrepancy between the data recorded on the actigraph and their sleep diary (Shown-Discrepancy Group), the other half were not shown the discrepancy (No-Demonstration Group). Participants were then asked to wear the actigraph and keep a sleep diary for three further nights. Following the behavioural experiment, the Shown-Discrepancy Group estimated their SOL more accurately and reported less anxiety and preoccupation about sleep compared to the No-Demonstration Group. The theoretical and clinical implications of these findings are discussed.

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*Keywords:* Insomnia; Distorted perception; Sleep; Behavioural experiment; Anxiety; Preoccupation

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The prevalence of insomnia in the United States is 33% with 9% reporting insomnia on a regular nightly basis (Ancoli-Israel & Roth, 1999). As such, insomnia is among the most prevalent psychological health problems. By definition, patients with chronic insomnia honestly and persistently complain of not getting enough sleep (American Psychiatric Association, 1994). However, the objective documentation of these has been somewhat illusory in that the majority of empirical investigations have not been able to detect clinically significant differences in polysomnographically measured total sleep time (TST) between patients with insomnia and good sleepers (e.g., Mendelson, 1993; Rosa & Bonnet, 2000; Spiegel, 1981). In studies where significant statistical

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differences were detected (e.g., Adam, Tomney, & Oswald, 1986; Gillin, Duncan, Pettigrew, Frankel, & Snyder, 1979; Knab & Engel, 1988), the degree of sleep deficit has often been regarded as unremarkable (Adam et al., 1986; Coates & Thoresen, 1981). For example, in a review of 14 related studies, Chambers and Keller (1983) reported the average difference in objective total sleep time between individuals with insomnia and normal sleepers to be only 35 min. This finding suggests that there is a significant proportion of patients for whom the severity of the complaint is not commensurate with the amount of sleep obtained. Consistently, there is robust evidence that a tendency toward overestimating sleep onset latency (SOL) and underestimating total sleep time is ubiquitous among patients with insomnia (e.g., Adam et al., 1986; Carskadon et al., 1976; Coates et al., 1982; Frankel, Coursey, Buchbinder, & Synder, 1976; Friedman et al., 2000; see Perlis, Giles, Mendelson, Bootzin, & Wyatt, 1997 for review; Spielman, Sadkin, & Thorpy, 1987; Wicklow & Espie, 2000).

In a recent cognitive model, we proposed that this characteristic distorted perception of sleep is one of the core processes that serves to maintain insomnia (Harvey, 2002). We suggest that if a patient with insomnia consistently *perceives* that he/she is not getting enough sleep, sleep related anxiety and preoccupation will be fuelled (e.g., “I’m losing control”, “I’m not going to cope”, “I will lose my job”). Escalating anxiety and preoccupation contribute to maintenance because they are not conditions conducive to optimal sleep onset (Espie, 2002) nor to optimal daytime performance (Sarason, 1984).

To date, cognitive behavioural treatments for insomnia have not specifically targeted distorted perception of sleep and the accompanying anxiety and preoccupation. The aim of the present experiment was to pilot the efficacy of a novel *behavioural experiment* with a view to filling this gap. Cognitive therapists employ two main methods to change distorted perceptions and unhelpful beliefs (Clark, 1999; Salkovskis, 1999). The first is a discussion technique. Through discussion, therapists provide information to directly challenge distortion in perceptions and logic of the patient’s beliefs (Beck, 1976; Ellis, 1962). However, as discussion is typically not sufficient (Teasdale, 1999), an alternative approach, behavioural experiment, has been developed. A behavioural experiment involves arranging real life experiences to provide a crystal clear and memorable demonstration to the patient that the perception or belief is not plausible, and from which the patient can derive corrective feedback (Beck, 1995; Clark, 1999; Salkovskis, 1999).

Based on the cognitive model of insomnia, we predicted that a behavioural experiment designed to correct distorted perception of sleep should be associated with (1) more accurate subjective perception of sleep and (2) reduced sleep related anxiety and preoccupation. Based on the null findings in comparisons of objectively measured sleep between good sleepers and individuals with insomnia (e.g., Chambers & Keller, 1993), we did not predict a change to objective sleep estimates. In an uncontrolled case series, an attempt to correct distorted perception of sleep among individuals with insomnia showed promise (Downey & Bonnet, 1992) but is not amenable to clinical practice as the procedure was intrusive (involving waking the patient 27 times on the training night) and required access to technically trained staff and expensive equipment (polysomnography).

## 1. Method

### 1.1. Participants

Participants were staff and students at two local universities in Oxford. A total of 52 individuals, aged 18–46 years, responded to an advertisement and attended a screening session. In the absence of a psychometrically validated alternative, a structured clinical interview, the Insomnia Diagnostic Interview (IDI), was used to identify the presence of insomnia. The IDI comprises five sections designed to carefully assess the presence of each DSM-IV (American Psychiatric Association, 1994) criteria for primary insomnia: (a) the predominant complaint is a difficulty in initiating or maintaining sleep or non-restorative sleep for at least 1 month (Cluster A); (b) the complaint causes clinically significant distress or impairment (Cluster B); (c) the insomnia does not occur exclusively as a result of another sleep or mental disorder (Cluster C and D); and (d) the insomnia is not due to the effects of a substance or illness (Cluster E). To be included in the present study, participants needed to have met all the criteria above. Additionally, participants' sleep disturbance must have been present at least three nights per week for at least one month (Morin, 1993). Ten participants were excluded for experiencing insomnia one or two nights a week ( $n = 4$ ) or not being clinically distressed or impaired ( $n = 6$ ). Two further participants were excluded from the final analyses due to unsuccessful experimental manipulation. Hence, the final sample comprised 40 individuals diagnosed with primary insomnia. The structured clinical interview for the DSM-IV (SCID; Spitzer, Williams, Gibbon, & First, 1996) was administered. Fifteen participants (37.5%) met criteria for one or more DSM-IV Axis 1 diagnoses in the past (major depression = 11, generalized anxiety disorder = 1, panic disorder = 2, post-traumatic stress disorder = 1). Two participants (5%) met criteria for one current DSM-IV Axis 1 diagnosis (generalized anxiety disorder, specific phobia). As insomnia is commonly comorbid with a range of psychological disorders (Harvey, 2001b), individuals with comorbid problems were not excluded as (1) the occurrence of comorbid psychological problems does not necessarily render insomnia as a secondary diagnosis (Harvey, 2001b), and (2) selecting 'pure' cases reduces the representativeness of the sample (McCrae & Lichstein, 2001; Morin, Stone, McDonald, & Jones, 1994). Instead, we made every effort to ensure that the sleep disturbance was currently the most distressing and disabling problem (Di Nardo, Moras, Barlow, Rapee, & Brown, 1993), and that the sleep disturbance did not occur exclusively as a result of the comorbid psychological disorder (American Psychiatric Association, 1994).

### 1.2. Design and procedures

Participants were randomly allocated to either the Shown-Discrepancy Group ( $n = 20$ ) or the No-Demonstration Group ( $n = 20$ ) and attended three sessions.

#### 1.2.1. Session one

After obtaining informed consent, the IDI and the SCID (Spitzer et al., 1996) were administered. Participants then completed the Pittsburgh Sleep Quality Index (PSQI; Buysse, Reynolds, Monk, Berman, & Kupfer, 1989), the Penn State Worry Questionnaire (PSWQ; Meyer, Miller, Metzger, & Borkovec, 1990), the Beck Anxiety Inventory (BAI; Beck, Epstein, Brown, & Steer,

1988), and the Beck Depression Inventory (BDI; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961). To index baseline distorted perception, participants were given an actigraph and a sleep diary. They were instructed to wear the actigraph, on their non-dominant wrist, from two hours prior to bedtime until rising in the morning for the next three consecutive nights and to complete the sleep diary immediately on waking for the next three consecutive mornings (Days 1–3). Both groups of participants were told that the purpose of the study was to observe the sleep/wake schedule over a six-day period.

### 1.2.2. *Session two*

When participants returned for the second session, the actigraph and the sleep diaries for Days 1–3 were collected and the Anxiety and Preoccupation about Sleep Questionnaire (APSQ) was administered. The Shown-Discrepancy Group then engaged in a behavioural experiment that involved two parts. First, participants were shown and taught to read their actigraph recordings from the last three days, after the experimenter had downloaded their sleep data from the actigraph to a computer. The experimenter then gave each participant a short tutorial in which the participant was informed that the data were generated from their wrist movement. The participant was then orientated to the method of calculating their own SOL and TST. No further verbal interaction ensued between the experimenter and the participants. Second, the participant completed a data summary sheet on which the sleep diary and the actigraph recordings were juxtaposed. Participants then calculated the discrepancy between the self-rated and objectively recorded sleep estimates. The aim of this exercise was to provide a clear and memorable demonstration of the discrepancy between how much participants *thought* they slept and how much the actigraph estimated they had slept. The procedure was identical for the No-Demonstration Group except that they did not do the behavioural experiment. To index the impact of the behavioural experiment, participants were instructed to wear the actigraph for the next three nights and complete a sleep diary for the next three mornings (Days 4–6).

### 1.2.3. *Session three*

During the final session, the actigraph and the sleep diaries for Days 4–6 were collected. All participants were asked to complete the APSQ with reference to the last three days. As a manipulation check, the Shown-Discrepancy Group was asked to rate the extent to which they believed the discrepancy between the sleep diary and actigraph recordings (1 “believe”, 10 “do not believe”). Participants were debriefed as to the purpose of the study.

## 1.3. *Materials*

### 1.3.1. *Anxiety and Preoccupation about Sleep Questionnaire*

In the absence of an existing questionnaire to index our second hypothesis, we developed the APSQ. The 10 items that comprise the APSQ were derived from statements made by patients with primary insomnia as documented in previous research (Borkovec, 1982; Harvey, 2001a; Watts, Coyle, & East, 1994). Participants were required to rate, on a 10-point scale, how true each of the statements was for them over the past three days (1 “not true”, 10 “very true”). A total score for the APSQ was obtained by summing across each rating. Example statements include: “I worry about the amount of sleep I am going to get every night” and “I put great effort into

rectifying my sleep problems”. The reliability and validity of the questionnaire was established on a sample of 110 university students (41 of whom had PSQI global score  $>5$ , a cut off identifying a clinically significant sleep disorder with 90% specificity; Buysse et al., 1989). The internal consistency (Cronbach’s alpha) of the total scale was 0.92. The correlation with PSQI ( $r = 0.44$ ,  $p < 0.0001$ ) and BAI ( $r = 0.37$ ,  $p < 0.0001$ ) indicated that higher scorers on the APSQ were also higher scorers on the PSQI and BAI.

### 1.3.2. Objective sleep estimates

An actigraph is a small, wrist-watch like device widely used in sleep research for the evaluation of sleep–wake cycles. Embedded within is a miniaturized piezoelectric acceleration sensor that detects and stores information about physical motion. Movement data can later be downloaded into compatible software to generate an estimation of the sleep/wake cycle. In the present study, the Mini Motionlogger Actigraph Basic (Ambulatory Monitoring Inc.) was used to provide an objective estimate of SOL and TST. Data were collected in Zero-Crossing Mode at 60-s intervals. Conversion of movement data in sleep parameters was performed by Action W using the Cole–Kripke algorithm (Cole, Kripke, Gruen, Mullaney, & Gillin, 1992). To facilitate more accurate scoring of sleep, participants were instructed to depress the event marker on the actigraph when they turned the light off to go to sleep and to depress the event marker for the second time when they wake up the following morning. The reliability and validity of the use of actigraphy has been widely investigated. According to a comprehensive review by the American Sleep Disorders Association (Sadeh, Hauri, Kripke, & Lavie, 1995), actigraphy is considered as a useful, acceptable instrument to measure sleep–wake schedule and sleep quality. Compared to polysomnographically measured sleep, the ‘gold standard’, there was a strong agreement rate (above 90%; Jean-Louis, Kripke, Mason, Elliott, & Youngstedt, 2001; Sadeh et al., 1995) in normal adults. However, the agreement rate is likely to be lower in individuals who lie immobile for long periods (e.g., clinically depressed patients and insomnia patients) as the technology is less accurate in differentiating quiet pre-sleep wakefulness from sleep (ASDA, 1995). For the use of actigraphy on insomnia patients, the epoch by epoch agreement rate ranges from 78.20% to 89.71% (Cole et al., 1992; Jean-Louis et al., 1997; Mullaney, Kripke, & Messin, 1980; Sadeh, Alster, Urbach, & Lavie, 1989). Whilst there are concerns about the merits of actigraphy in providing a precise estimate of sleep onset, the advantage of using actigraphy in the context of the present study was that (1) it is non-intrusive, (2) it allows sleep to be monitored in the participants’ home, a natural sleeping environment, (3) it is relatively inexpensive, and most importantly of all, (4) actigraphic data are quickly downloaded and are presented in a clear and easily digestible manner such that participants can easily calculate how long it took them to fall asleep and how long they slept in total.

### 1.3.3. Subjective sleep estimates

The daily sleep diary comprised four questions asking participants to record their bedtime, SOL, TST, and wake time.

## 2. Results

### 2.1. Participant characteristics

Table 1 presents the participant characteristics. There were no differences between the Shown-Discrepancy Group and the No-Demonstration Group for sex composition (analysed with chi-square), age, subjectively estimated SOL, subjectively estimated TST, PSQI, BAI, BDI, PSWQ and insomnia duration (analysed with independent sample *t*-tests).

### 2.2. Baseline distorted perception of sleep

At baseline (Days 1–3), participants as a whole overestimated their SOL by 37 min and underestimated their TST by 46 min, indicating that this sample of participants did exhibit distorted perception of sleep. Further, at baseline, the mean objective sleep data (SOL = 23.6 min, TST = 7.2 h) were within the range recorded from good sleepers (SOL < 30 min, TST = 7–8 h; Morin, 1993), indicating that this sample of participants did not exhibit a ‘substantial deficit in nocturnal sleep time’ (Chambers & Keller, 1993, p. 649).

The analyses below involved a series of repeated measures ANOVAs with Group (Shown-Discrepancy Group vs. No-Demonstration Group) as the between subjects factor and Session (pre-experiment vs. post-experiment) as the within subjects factor. To explore significant interactions, *t*-tests were conducted with a Bonferroni adjustment to control for multiple comparisons ( $p < 0.0125$ ).

Table 1  
Participant characteristics

	Shown-Discrepancy Group	No-Demonstration Group	$\chi^2$ (1)
Sex			
Female	15	11	1.76
Male	5	9	
			<i>t</i> (38)
Age	22.8 (5.2)	24.6 (7.0)	−0.92
sSOL	67.6 (33.9)	76.5 (55.3)	−0.61
sTST	6.3 (0.9)	6.5 (1.4)	−0.53
PSQI	11.0 (3.1)	10.1 (3.7)	0.84
BAI	14.8 (8.0)	11.2 (10.0)	1.28
BDI	12.5 (8.6)	12.2 (10.9)	0.10
PSWQ	55.3 (15.4)	48.9 (15.1)	1.33
Insomnia duration	5.2 (4.6)	4.9 (4.9)	0.19

*Note:* Mean values are reported with standard deviations in parentheses. Age is reported in years; sSOL = subjectively estimated sleep onset latency in the last month (in minutes); sTST = subjectively estimated total sleep time in the last month (in hours); PSQI = global score on the Pittsburgh Sleep Quality Index; BAI = total score on the Beck Anxiety Inventory; BDI = total score on the Beck Depression Inventory; PSWQ = total score on the Penn State Worry Questionnaire; Insomnia duration is reported in years

### 2.3. The effect of the behavioural experiment on subjective sleep perception

#### 2.3.1. Subjective SOL estimates

Fig. 1a depicts the change in subjective SOL for the two groups before and after the behavioural experiment. There was no significant main effect for Group. There was a significant main effect for Session,  $F(1, 38) = 13.78$ ,  $p < 0.001$ , such that the SOL estimates were shorter after the behavioural experiment compared to before the behavioural experiment. There was a significant Group by Session interaction,  $F(1, 38) = 4.51$ ,  $p < 0.05$ . Follow up tests indicated that the Shown-Discrepancy Group estimated their SOL to be shorter after the behavioural experiment relative to before the behavioural experiment,  $t(19) = 3.52$ ,  $p < 0.002$ . This difference was not observed for the No-Demonstration Group.

#### 2.3.2. Subjective TST estimates

Fig. 1b depicts the change in subjective TST for the two groups before and after the behavioural experiment. There was no significant main effect for Group. There was a significant main effect for Session,  $F(1, 38) = 14.47$ ,  $p < 0.001$ , such that the TST estimate was longer after the behavioural experiment compared to before the behavioural experiment. There was no interaction.

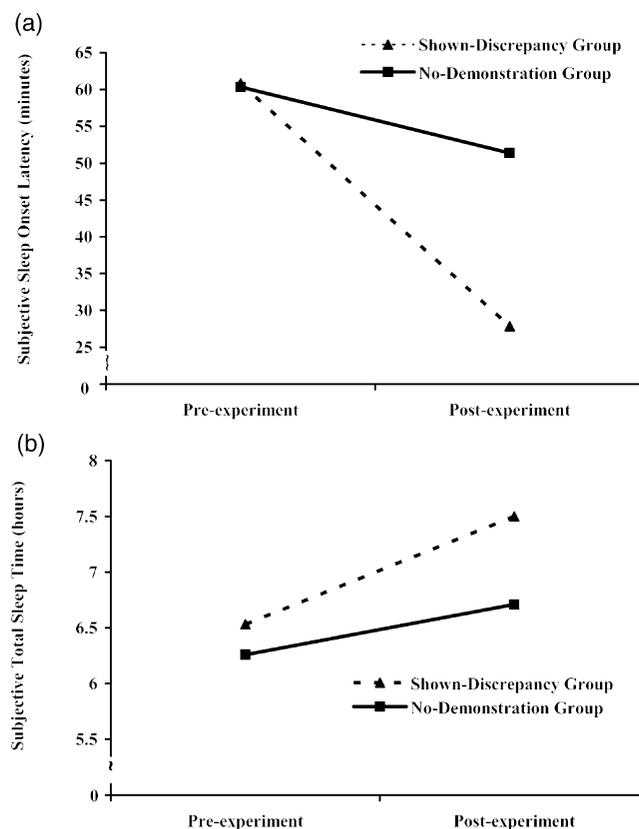


Fig. 1. (a) Subjective sleep onset latency before and after the behavioural experiment. (b) Subjective total sleep time before and after the behavioural experiment.

#### 2.4. The effect of the behavioural experiment on sleep related anxiety

The mean APSQ scores for the two groups before and after the behavioural experiment are depicted in Fig. 2. There was no significant main effect for Group. There was a significant main effect for Session,  $F(1, 38) = 4.09, p < 0.05$ , such that the APSQ score was lower post-experiment relative to pre-experiment. There was also a significant Group by Session interaction,  $F(1, 38) = 13.15, p < 0.001$ . Follow up tests indicated that the Shown-Discrepancy Group reported lower sleep-related anxiety and preoccupation after the behavioural experiment compared to before the behavioural experiment,  $t(19) = 3.83, p < 0.001$ . This difference was not observed for the No-Demonstration Group. Effect sizes were calculated so that a positive effect size denoted a reduction in anxiety and preoccupation about sleep [ $d = (M_1 - M_2) / \sigma_{pooled}$ ]. In order to give a more precise estimate of the population variance, we used the pooled standard deviation,  $\sigma_{pooled} = \sqrt{(\sigma_1^2 + \sigma_2^2) / 2}$  instead of the standard deviation of either group. Whilst the effect size for the No-Demonstration Group was  $-0.13$ , that for the Shown-Discrepancy Group was  $0.48$ , a medium effect size according to Cohen's (1988) definition.

#### 2.5. The effect of the behavioural experiment on objective sleep

##### 2.5.1. Objective SOL estimates

Before the behavioural experiment, the mean objective SOL for the Shown-Discrepancy Group was 23.4 min (SD = 14.7) and for the No-Demonstration Group was 23.8 min (SD = 23.7). After the behavioural experiment, the mean objective SOL for the Shown-Discrepancy Group was 17.9 min (SD = 12.3) and for the No-Demonstration Group was 26.5 min (SD = 21.3). No significant effects were observed on this variable.

##### 2.5.2. Objective TST estimates

Before the behavioural experiment, the mean objective TST for the Shown-Discrepancy Group was 7.1 h (SD = 1.0) and for the No-Demonstration Group was 7.2 h (SD = 1.7). After the behavioural experiment, the mean objective TST for the Shown-Discrepancy Group was 7.5 h

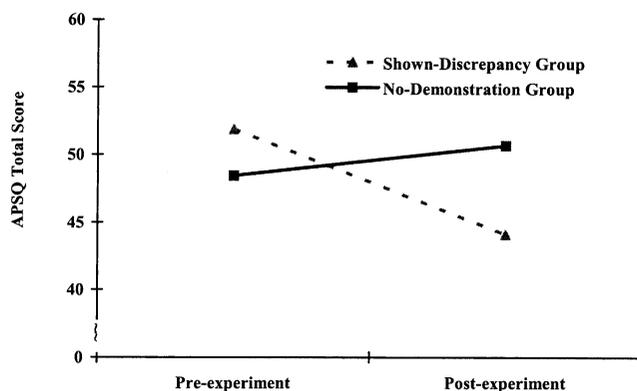


Fig. 2. Anxiety and Preoccupation about Sleep Questionnaire (APSQ) total score before and after the behavioural experiment.

(SD = 1.1) and for the No-Demonstration Group was 7.2 h (SD = 1.5). No significant effects were observed on this variable.

### 2.5.3. Manipulation check

The mean belief rating for the Shown-Discrepancy Group was 3.4 (SD = 1.9), indicating that participants readily accepted the information conveyed during the behavioural experiment and that, from the participants' point of view, the behavioural experiment had credibility. Pearson correlation analyses (2-tailed) were performed to examine the relationship between the credibility of the behavioural experiment, as reflected in the belief rating, and reduction in anxiety and preoccupation about sleep, improvement in SOL and TST estimation. No significant correlations were detected. This is not surprising as the variability of the belief rating was limited by the exclusion of individuals ( $n = 2$ ) who scored more than 7.

## 3. Discussion

The first prediction tested was that the behavioural experiment would be associated with improved subjective sleep estimates. In support, the Shown-Discrepancy Group estimated their SOL to be shorter after, compared to before, the behavioural experiment. This difference was not significant for the No-Demonstration Group. It is worth noting that after the behavioural experiment the Shown-Discrepancy Group's subjective SOL estimate ( $M = 27.9$  min) improved to within 30 min, the cut-off for 'normal' SOL (Morin, 1993; Morin et al., 1999).

Interestingly, whilst the behavioural experiment was successful in correcting perception of SOL, it was not as potent for TST. One possible explanation for this result was that perhaps the discrepancy for SOL was demonstrated with more clarity: SOL was easily estimated at a glance, whereas TST involved subtracting periods of wakefulness after sleep onset. It should be noted that for TST there was a significant main effect for Session, suggesting that both groups slept better across time. This result is likely to be attributable to a diary keeping effect, such that an "enhanced awareness" of sleep patterns may reduce anxiety over sleep loss and thus contribute to better sleep (Morin, 1993, p.71).

The second prediction was that the behavioural experiment should be associated with reduced sleep related anxiety and preoccupation. Consistent with this prediction, after the behavioural experiment, the Shown-Discrepancy Group reported significantly lower levels of anxiety and preoccupation about sleep compared to before the behavioural experiment, an improvement that was not observed for the No-Demonstration Group. This finding is consistent with the view that distorted perception of sleep serves to fuel anxiety and preoccupation with sleep (Harvey, 2002). Several alternative explanations of the results need consideration. First, perhaps the therapeutic effect seen in the Shown-Discrepancy Group was a response to demand characteristics inherent to the experimental procedure. However, as great care was taken throughout the study to avoid giving participants any indication that they were expected to show any improvement in their sleep, this account is not entirely convincing. In future research, consideration should be given to employing a proactive counterdemand procedure. Second, perhaps the therapeutic effect seen in the Shown-Discrepancy Group was an artefact of the interaction between the participant and experimenter during the behavioural experiment. Since interpreting actigraphic data was novel to

all participants, some verbal exchange was inevitable. However, this is an unlikely account of the results as the verbal exchange was minimal. Third, perhaps the therapeutic effect seen in the Shown-Discrepancy Group was a placebo effect. That is, perhaps participants would have shown a reduction in sleep-related anxiety and preoccupation if they were simply shown any feedback. Whilst the current design does not allow us to eliminate this possibility, it would be interesting to test in future research whether or not participants given false feedback would show comparable therapeutic effect. Nevertheless, it should be emphasised that during the session it was common for participants to check that they were viewing their *own* data typified by comments such as “I remember I was woken up by my dog at 5 a.m. this morning and from then onward I couldn’t go back to sleep. Wow, I can see that here (pointing to the screen)” and “Oh it’s interesting... I did go to the toilet twice last night... I can see where I got up here (pointing to the screen) and here (pointing to the screen)”.

There are several limitations to this study. First, the sample was drawn from the university population. However, it should be noted that all participants met strict DSM-IV criteria for primary insomnia, the mean PSQI global score for the sample was 10.5, and the average duration of the insomnia for the sample was 5 years. Nonetheless, replication with a clinical sample is a crucial “next step”. In particular, it would be interesting to check whether the behavioural experiment has potential to improve objective sleep in more severe samples. Second, as participants were followed up for three days, the long term durability of the behavioural experiment is not known. Third, whilst the obvious advantage of actigraphy is that it is non-intrusive and inexpensive, the disadvantage is that it provides an indirect measure of sleep based on movement and as such, some error is inevitably introduced. For instance, actigraphy might have over scored sleep in individuals who experienced long periods of quiet wakefulness before sleep onset. Nonetheless, it should be noted that the epoch by epoch agreement rate between polysomnography and actigraphy for patients with insomnia is overall reasonably high (78.20–89.71%; e.g., Sadeh et al., 1995) and the design of the experiment has contributed to keeping the error rate constant across the two groups tested. Importantly, when our participants examined their *own* sleep data during the behavioural experiment, they rated the actigraphic data to be a plausible objective representation of their sleep. Fourth, the sample was not objectively assessed for comorbid sleep disorders such as sleep apnea, periodic limb movement syndrome and narcolepsy. However, the IDI includes a section designed to carefully screen for the key symptoms of comorbid sleep disorders. No participant endorsed any screening question. According to the Standards of Practice report by the American Academy of Sleep Medicine, full overnight polysomnographic assessment is not mandatory for the routine evaluation of chronic insomnia, unless there is a valid rationale to support the use of it (Chesson et al., 2000).

After treatment, patients with insomnia often demonstrate improvements on subjective indices of sleep despite a lack of change in objectively measured sleep (e.g., Engle-Friedman, Bootzin, Hazlewood, & Tsao, 1992 used polysomnography; Friedman et al., 2000 used actigraphy). This pattern of findings is consistent with those of the present study and adds weight to the argument that subjective perception of sleep plays a central role in insomnia. As is reflected in the current diagnostic criteria, insomnia is essentially a subjective complaint of insufficient sleep and is identified primarily on the basis of the patient’s self-report. Future research may benefit from greater consideration of the clinical significance of the role of subjective perception of sleep in the context of insomnia.

In conclusion, a novel behavioural experiment designed to reduce distorted perception of sleep was associated with improved subjective perception of SOL and reduced sleep related anxiety and preoccupation. Theoretically, the findings support the proposal that distorted perception of sleep functions to maintain insomnia by fuelling anxiety and preoccupation with sleep (Harvey, 2002). Clinically, the findings highlight the possible benefits of targeting inaccuracy in the perception of the amount of sleep obtained when treating insomnia and provides a non-intrusive, easy to administer method to do this. Future research is required to correct the limitations discussed above and to determine whether such an intervention might enhance the already moderately effective cognitive behavioural intervention for insomnia (average effect size = 0.42–0.88; Morin, Culbert, & Schwartz, 1994).

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