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INITIAL INSOMNIA AND PARADOXICAL INTENTION: AN EXPERIMENTAL INVESTIGATION OF PUTATIVE MECHANISMS USING SUBJECTIVE AND ACTIGRAPHIC MEASUREMENT OF SLEEP

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Abstract Paradoxical Intention (PI) is a cognitive treatment approach for sleep-onset insomnia. It is thought to operate by eliminating voluntary sleep effort, thereby ameliorating sleep performance anxiety, an aroused state incompatible with sleep. However, this remains untested. Moreover, few PI studies have employed objective sleep measures. The present study therefore examined the effect of PI on sleep effort, sleep anxiety and *both* objective and subjective sleep. Following a seven-night baseline, 34 sleep-onset insomniacs were randomly allocated to 14 nights of PI, or to a control (no PI) condition. Consistent with the performance anxiety model, participants allocated to PI, relative to controls, showed a significant reduction in sleep effort, and sleep performance anxiety. Sleep-onset latency (SOL) differences between PI participants and controls using an objective sleep measure were not observed, although an underlying trend for significantly lowered subjective SOL amongst PI participants was demonstrated. This may relate to actigraphic insensitivity, or more probably confirms recent suggestions that insomniacs readily overestimate sleep deficit, due to excessive anxiety about sleep. Together, results help determine putative mechanisms underlying PI, have important implications for the clinical application of PI, and emphasize the need for further PI research within an experimental cognitive framework.

Keywords: Insomnia, paradoxical intention, cognitive-behaviour therapy, performance anxiety, actigraphy.

Introduction

Evidence has converged suggesting pre-sleep cognitive activity is a key maintaining factor in sleep-onset insomnia (Espie, 2002; Harvey, 2002). Despite this, research examining stand-alone cognitive insomnia interventions remains rare. One exception to this is Paradoxical Intention (PI). Single case (Ascher, 1975; Ascher & Efran, 1978; Espie & Linday, 1985) and randomized-controlled studies (Ascher & Turner, 1979, 1980) support the utility of PI in the management of sleep-onset insomnia, and its equivalence to stimulus control and relaxation (Espie, Lindsay, Brooks, Hood, & Turvey, 1989; Ladoucer & Gros-Loius, 1986; Turner & Ascher, 1979). Indeed, PI is now regarded as a ''probably efficacious'' insomnia

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intervention according to APA criteria (Chesson, Anderson, Littner, Davila, & Hartse, 1999; Morin et al., 1999).

Despite these outcome data, and its now routine use within multicomponent CBT (e.g. Espie, Inglis, Tessier, & Harvey, 2001), the mechanisms underlying PI remain unclear. PI is a cognitive approach, which requires poor sleepers give up voluntary effort to control sleep. The treatment achieves this by encouraging insomniacs to relax with the lights out at bedtime, keeping their eyes open. Paradoxically, the likelihood of staying awake is reduced by encouragement to do so (Espie & Lindsay, 1985).

Sleep is a behaviour, which cannot be placed under full voluntary control. One hypothesis therefore is that direct attempts to control sleep will fail, because sleep effort inhibits relaxation and sets up performance anxiety, an aroused state incompatible with sleep. Consistent with this, Ascher and Turner (1979) have argued that by eliminating voluntary sleep effort, PI minimizes sleep performance anxiety, thereby promoting rapid sleep-onset. Similarly, Espie (2002) has suggested that by diverting attention away from sleep performance, PI facilitates cognitive/affective de-arousal and promotes sleep. To date, however, these performance anxiety conceptualizations remain untested. This study, therefore, is *not* an evaluation of the efficacy of PI. Rather, it aimed to clarify whether PI institutes sleep change via sleep effort/performance anxiety reduction.

There also, at present, remains a marked lack of PI research using objective sleep measures. This is problematic. Insomniacs' self-report sleep data can be unreliable (Carskadon et al., 1976), and objective and subjective sleep measures may reflect differing response systems (Wicklow & Espie, 2000). To date, only one PI study has employed objective sleep measurement (Ott, Levine, & Ascher, 1983), and the method employed only estimated sleep-onset latency (SOL) objectively to within five minutes. A second aim of the present study was therefore to examine and compare objective (actigraphy) and subjective (self-report diary) sleep outcome following brief (14 night) PI using a reliable sleep measurement system.

A reliable and minimally intrusive objective sleep measure is the actigraph – a small wrist attachment that records the wearer's movements. There is recognition that movement is a good predictor of wakefulness, whilst lack of movement is a good predictor of sleep (American Sleep Disorders Association, 1995; Mullaney, Kripke, & Messin, 1980). Moreover, actigraphic measures correlate highly with polysomnographic (PSG) data for sleep duration and total wake time (e.g. Sadeh, Hauri, Kripke, & Lavie, 1995; Mullaney et al., 1980).

To summarize then, a study was conducted examining the performance anxiety model of PI. Sleep-onset insomniacs were randomly assigned to two weeks of PI, or a control (no PI) condition, following a one-week baseline. Voluntary sleep effort and sleep anxiety data were collected, alongside actigraphic and subjective sleep data. A One Between (Condition: Paradoxical Intention [PI], Control) and One Within factor (Time: Baseline, Week One, Week Two) design was employed. Relative to the control condition, it was predicted allocation to PI would (i) reduce sleep effort after Weeks One and Two; (ii) reduce sleep performance anxiety after Weeks One and Two; (iii) reduce objective and subjective SOL after Weeks One and Two; (iv) and raise objective and subjective sleep efficiency after Weeks One and Two. Importantly, the research was *not* designed as a treatment study, rather as an experimental examination of putative mechanisms underlying PI, as well as objective/subjective sleep outcome following the procedure.

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Method

Participants

Participants were recruited using the University e-mail system and via notices placed locally. The Health Authority and University granted ethical approval. Prior to participation potential participants completed screening questionnaires assessing sleep (Pittsburgh Sleep Quality Index [PSQI] – Buysse, Reynolds, Monk, Berman, & Kupfer, 1989; Sleep History Questionnaire – Morin, 1993), anxiety (Spielberger Trait Anxiety Inventory – STAI; Spielberger, Gorsuch, & Lushene, 1970), worry (Penn State Worry Questionnaire – PSWQ; Meyer, Miller, Metzger, & Borkovec, 1990) and depression (Beck Depression Inventory – BDI; Beck, Steer, & Brown, 1996).

Respondents were included if they were between 16 and 65 years, complained of clinically significant problems falling asleep according to the revised version of the International Classification of Sleep Disorders (American Sleep Disorder Association, 1997; i.e. SOL greater than 30 min at least 4 nights per week, with or without disruption to other sleep variables), and scored in excess of 5 on the PSQI, the recognized cut-off for identifying clinically significant sleep disturbance (Buysse et al., 1989). Participants were excluded if they experienced intermittent awakenings without sleep-onset difficulties, were receiving treatment for sleeping difficulties, or were suffering any medical or psychopathological disorder impacting on sleep.

Forty-six participants were identified, all meeting criteria. Thirty-four completed the experiment (74%; mean age 25.2 years, average sleep disturbance 6.35 years). A further seven failed to attend the initial meeting, three withdrew during baseline, and two were excluded due to unreliable diary completion.

Measures

A daily sleep diary (Espie, 1991), completed upon rising for 21 days, provided measures of subjective SOL and sleep efficiency (based on time to bed, rise time, time to fall asleep, and total sleep time responses). Effort to sleep data were recorded in the diary using a 7-point scale (‘‘I tried hard to get to sleep last night’’; anchor points 0 ‘‘not at all’’, 6 ‘‘very much’’).

Two self-report scales measured sleep-related performance anxiety: the Sleep Anxiety Scale (SAS; Fogle & Dyall, 1983, see p. 26 for internal consistency data); and a specially developed scale, the Sleep Performance Anxiety Questionnaire (SPAQ). The latter comprised seven components of dysfunctional sleep monitoring (sleep effort, sleep control, sleep avoidance, bedtime worry, performance failure, anticipatory anxiety, and daytime worry). Piloting indicated the SPAQ readily distinguished good ($N = 4$; mean = 7.75, $SD = 0.96$) from poor ($N = 4$; mean = 15.50, $SD = 3.69$) sleepers. Cronbach’s alpha data for both scales are presented in the results.

Wrist actigraphic recording using the ‘‘Actiwatch’’ (Model AW2; Cambridge Neurotechnology Ltd) provided objective estimates of SOL and sleep efficiency. As noted, actigraphic measures correlate highly with polysomnographic (PSG) data for sleep duration and total wake time (Sadeh et al., 1995; Mullaney et al., 1980), and movement is a good predictor of wakefulness, whilst lack of movement is a good predictor of sleep (American Sleep Disorders Association, 1995; Mullaney et al., 1980).

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Participants wore the actigraph continuously on their non-dominant hand except during wet activities. An event marker was depressed at lights out, and upon rising. Epoch length was set at 1 min. (Wicklow & Espie, 2000), with ''sleep'' or ''wakefulness'' determined by the program's algorithm.

Procedure

Following a telephone interview, participants were sent screening questionnaires (see participants section), an information sheet, and an informed consent slip. These were completed and returned by post. Participants then received a sleep diary and wrist actigraph, and were told the next seven days was a baseline measurement week.

Seven days later, participants completed the two sleep anxiety scales (Baseline), and were issued with further copies to be completed after seven days (Week One). Participants were then randomly allocated to experimental condition.

PI participants were introduced to the rationale of PI, and instructed, at lights out, to stay awake for as long as possible by keeping their eyes open. The need to resist sleep-onset gently but persistently in an environment conducive to sleep was emphasized. The use of active methods to stay awake (e.g. reading, physical movement) was discouraged. Patient expectations can influence response to PI (Epsie & Lindsay, 1985). In an attempt to control for this, half of PI participants were told to expect immediate sleep improvement (positive demand), whereas half were told to expect sleep improvement only at Week Two (counter demand). Control participants were told to continue wearing their actiwatch, and to continue completing their sleep diary.

After 14 nights, all participants returned their diaries and actigraph, and completed two final sleep anxiety scales (Week Two), and a compliance-rating sheet. They were then debriefed and thanked, and issued with the ''Good Sleep Guide'', a leaflet describing behavioural advice for home practice prepared by the second author (National Medical Advisory Committee, 1993).

Results

Participant characteristics

Participants had a mean age of 25.2 years, and mean sleep disturbance duration of 6.35 years. PI participants and controls were equitable in terms of gender ($p > .10$, NS), and did not differ on age, duration of sleep problem, trait anxiety, worry, depression or sleep quality (all $p > .10$; see Table 1 for data).

Compliance with experimental instructions

Participants reported correctly following experimental instructions (diary completion, actiwatch use) on mean = 19.2 nights. Mean compliance rating (scale 0–6) was 5.03, SD = 0.79. Non-compliance included forgetting to press the actiwatch, or to replace the actiwatch after wet activities. This was unusual.

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Table 1. Demographic and questionnaire data, and sleep anxiety, sleep effort, objective and subjective sleep-onset latency (mins) and sleep efficiency (%) data at Baseline (B), Week One (W1) and Week Two (W2), for paradoxical intention (PI) and control (C) participants

	Age	Females: Males	Insomnia duration (years)	STAI-T	PSWQ	BDI	PSQI
Condition							
PI	26.00	9:8	5.93	37.35	40.58	7.94	11.18
C	24.35	10:7	6.77	37.29	40.11	9.88	10.59

		SAS			SPAQ			Sleep effort		
		B	W1	W2	B	W1	W2	B	W1	W2
PI	*M*	16.18	13.59	12.29	15.41	12.53	11.41	2.33	1.11	1.10
	SD	4.05	3.78	4.03	2.87	3.89	3.61	1.37	1.20	1.30
C	*M*	15.76	16.35	16.05	14.88	15.18	14.76	2.28	2.21	2.14
	SD	3.83	3.12	3.60	2.71	2.43	2.51	1.44	1.12	1.28

		Objective SOL			Objective efficiency			Subjective SOL			Subjective efficiency		
		B	W1	W2	B	W1	W2	B	W1	W2	B	W1	W2
PI	*M*	29.92	27.23	24.47	80.87	81.84	80.65	65.74	41.76	38.24	80.87	86.10	85.79
	SD	17.16	15.06	17.20	6.67	7.76	7.57	33.97	19.62	25.28	13.49	11.85	11.07
C	*M*	26.62	25.19	25.03	78.93	79.80	80.64	54.67	58.82	55.33	80.04	82.46	82.82
	SD	21.83	16.37	16.49	7.93	7.34	8.95	25.67	32.98	29.16	11.85	10.32	12.40

Using PI

All 17 PI participants said they lay awake with their eyes open when using PI. None used active methods (reading, television) to stay awake.

Outcome variables

All data were first examined for kurtosis and skewness and fell within acceptable limits. Data also showed homogeneous variance, following Hartley's Fmax test (Winer, 1971). Analyses then relied on a Two (Condition: Paradoxical Intention vs. Control) by Three (Time: Baseline vs. Week One vs. Week Two) ANOVA design based on weekly means. Alpha level was set at .05, two-tailed, throughout. Means and standard deviations are presented in Table 1.

Sleep effort

ANOVA revealed a main effect of Time ($F[2, 64] = 9.05$, $p = .0001$), and a Time × Condition interaction ($F[2, 64] = 6.37$, $p = .003$). In order to clarify this, Bonferroni corrected simple main effects analyses for Condition were completed across the Time variable (critical p value = .017; Keppel, 1993). As is evident in Figure 1 panel A, this indicated relative to controls, PI participants showed significantly lower sleep effort at Week One ($F[1, 33] = 7.72$, $p = .009$; critical p value = .017) and near significant lower effort at Week Two ($F[1, 33] = 5.55$, $p = .025$; critical p value = .017). No differences were observed at Baseline ($F[1, 33] = 0.01$, $p > .1$, NS).

Sleep performance anxiety

Separate ANOVAs were run for the two performance anxiety scales employed, based on participant scores at Baseline, Week One and Week Two.

Sleep Anxiety Scale (SAS). ANOVA revealed a main effect of Time ($F[2, 64] = 6.69$, $p = .002$), and an interaction effect of Time × Condition ($F[2, 64] = 9.84$, $p = .0001$). As is evident in Figure 1 panel B, simple main effects indicated PI participants displayed significantly lower sleep anxiety at Week Two ($F[1, 33] = 8.26$, $p = .007$), and a near significant trend for lower sleep anxiety at Week One ($F[1, 33] = 5.41$, $p = .026$; critical p value = .017). No differences were observed at Baseline ($F[1, 33] = 0.09$, $p > .1$, NS).

In order to examine the internal consistency of scale data, baseline item scores across participants were subjected to appraisal using Cronbach's alpha. Overall alpha coefficient was 0.86, with range of alpha values, if item deleted, of 0.83–0.87. The mean corrected item-total correlation was 0.62.

Sleep Performance Anxiety Questionnaire (SPAQ). Analysis revealed a main effect of Time ($F[2, 64] = 15.68$, $p = .0001$), and a Time × Condition interaction ($F[2, 64] = 15.48$, $p = .0001$), displayed in Figure 1 panel C. As is evident, simple main effects indicated PI participants showed significantly lower sleep anxiety at Week Two ($F[1, 33] = 9.89$, $p = .004$), and a trend for lower sleep anxiety at Week One ($F[1, 33] = 5.66$, $p = .023$). No differences were observed at Baseline ($F[1, 33] = 0.30$, $p > .1$, NS). Overall alpha coefficient

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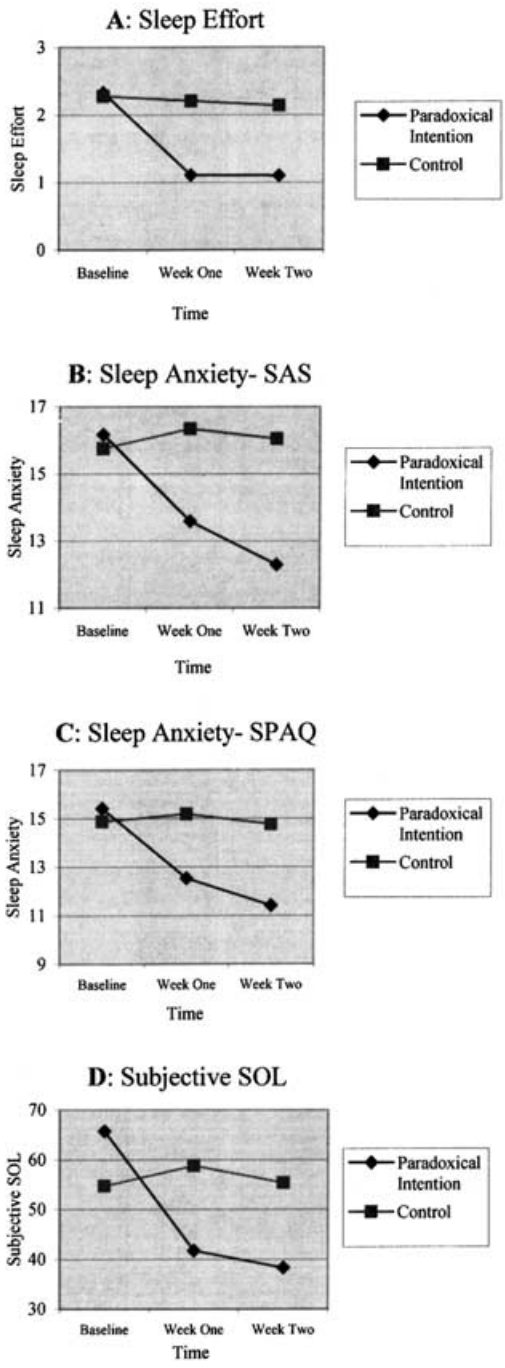

Figure 1. Mean sleep effort, sleep anxiety and subjective SOL for PI and control participants, as a function of Time

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for the SPAQ was 0.70, with the range of alpha values, if item deleted, of 0.63–0.73. The mean corrected item total correlation was 0.42.

Finally, Pearson correlation coefficients were computed across participants' baseline scores on the SAS and SPAQ. This indicated correlation was ''moderate'' ($r = 0.62$, $p < .01$), representing 38.5% shared variance.

Sleep

Two participants' actigraph data were lost due to faulty equipment. Analyses were therefore based on 16 participants per condition.

Counterdemand instructions. A series of Two (Instructions: Counterdemand vs. Positive Demand) by Three (Time: Baseline vs. Week One vs. Week Two) ANOVAs for PI participants *only*, on objective and subjective SOL and sleep efficiency, revealed no significant Instruction × Time interaction effects (all $p > .1$, NS; see Table 2). Primary sleep analyses therefore compared PI participants with controls.

Objective sleep (actigraphy). ANOVA for objective SOL revealed non-significant main effects of Time ($F[2, 60] = 0.48$, $p > .1$, NS) and Condition ($F[1, 30] = 0.06$, $p > .1$, NS). The Time × Condition interaction was also non-significant ($F[2, 60] = 0.13$, $p > .1$, NS). Objective SOL remained at approximately 25–30 minutes across condition (see Table 1).

Similarly, ANOVA for objective sleep efficiency data failed to reveal any significant effects of Time ($F[2, 60] = 0.53$, $p > .1$, NS), Condition ($F[1, 30] = 0.67$, $p > .1$, NS), or Time × Condition ($F[2, 60] = 0.19$, $p > .1$, NS). Objective efficiency remained at approximately 80% (see Table 1).

Subjective sleep (diary). ANOVA revealed a main effect of Time ($F[2, 64] = 6.72$, $p = .002$), and an interaction of Time × Condition ($F[2, 64] = 9.16$, $p = .0001$), displayed in Figure 1 panel D. Simple main effects analyses could be indicative of an underlying trend for lower SOL amongst PI participants, relative to controls, at Weeks One ($F[1, 33] = 3.36$,

Table 2. Mean and standard deviation objective and subjective sleep-onset latency (mins) and sleep efficiency (%) at Baseline (B), Week One (W1) and Week Two (W2), for counterdemand (C) and positive demand (P) instructions

	Objective				Subjective			
	SOL		Efficiency		SOL		Efficiency	
Condition	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
BC	20.73	10.52	78.31	16.96	62.03	20.97	78.30	16.96
BP	37.10	19.15	83.15	10.00	69.03	39.96	83.15	9.99
W1 C	22.41	13.71	81.82	16.04	46.66	18.41	81.82	16.04
W1 P	28.48	18.11	89.92	4.61	37.41	20.69	89.92	4.60
W2 C	19.32	10.35	82.53	14.53	39.41	28.01	82.53	14.53
W2 P	29.61	21.59	88.70	6.36	37.19	24.26	88.69	6.35

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p = .076) and Two (F[1, 33] = 3.34, p = .077). However, test statistics on small samples can be unstable. No differences were observed at Baseline (F[1, 33] = 1.15, p > .1, NS).

Allocation to PI resulted in a 41.85% reduction in SOL, compared to a 1.21% increase amongst controls (see Table 1). Treatment effect size was ''moderate'' (d = 0.61; Cohen, 1988). 70.6% of PI participants reported PI helped them get to sleep quicker.

Analysis of subjective sleep efficiency data revealed a main effect of Time (F[2, 64] = 6.65, p = .024), although the Time × Condition interaction failed to reach significance (F[2, 64] = 0.73, p > .1, NS).

The relationship between objective and subjective sleep measures. As inspection of Table 1 reveals actigraphic SOL at baseline was 29.92 minutes for PI participants, and 26.62 minutes for controls. At Week Two, these scores were unchanged for both PI participants (mean = 24.47 minutes) and controls (mean = 25.03 minutes). No significant objective SOL change following PI was therefore observed.

Subjective SOL scores at baseline were much higher than objective SOL scores for PI participants (mean = 65.74 minutes) and controls (mean = 54.67 minutes). This suggests all participants *overestimated* SOL *relative to* actigraphic assessment as an objective criterion at baseline. The discrepancy between subjective (mean = 38.24 minutes) and objective (mean = 24.47 minutes) SOL scores reduced for PI participants by Week Two. Control subjects showed no discrepancy reduction (mean subjective SOL = 55.33 minutes, mean objective SOL = 25.03 minutes). This suggests allocation to PI reduced participants' tendency to overestimate subjective SOL.

Pearson correlation coefficients across all participants examined the association between objective and subjective SOL, and objective and subjective sleep efficiency (baseline – week 2). Correlations were ''low'' for SOL (r = 0.25), and ''low'' for sleep efficiency (r = 0.17).

The association between sleep effort, sleep performance anxiety and subjective sleep onset latency

To explore whether reduced sleep effort or sleep anxiety best predicted reduced *subjective* SOL, change scores for each variable (baseline – week 2) were computed and subjected to Pearson correlational analyses. SOL change was signficantly associated with sleep anxiety change (SAS: r = 0.36, p =.035; SPAQ: r = 0.43, p = .01), and with effort change (r = 0.56, p = .001). Comparing explained variance (SAS r^2 = 0.13; SPAQ r^2 = 0.18; Effort r^2 = 0.31), the association between SOL change and effort change is strongest.

Since baseline sleep anxiety was significantly associated with sleep effort (SAS; r = 0.56, p = .001), (SPAQ; r = 0.58, p = .0001), partial correlations were computed for PI participants *only* to reveal the individual effect of effort change, and anxiety change (both scales), on subjective SOL change. Effort change significantly correlated with SOL change when sleep anxiety was partialled out (SAS: r_p = 0.48, p = .029; SPAQ: r_p = 0.44, p = .045). In contrast, when effort change was partialled out, neither measure of sleep anxiety was significantly associated with SOL change (SAS: r_p = –0.2, p > .1, NS; SPAQ: r_p = –0.1, p > .1, NS). This suggests SOL reduction amongst PI participants was most strongly associated with sleep effort reduction.

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Discussion

The present study has several limitations. The sample employed was non-treatment seeking, and the study timescale was short, only 21 nights. The effect of PI in the longer term is therefore unclear. The design lacked a credible but otherwise inert control treatment. So the effect of non-specific factors on the findings is unknown. Sample size was small, which underpowered the counterdemand-positive demand comparison, and the measure of sleep effort employed was not validated prior to the study.

Relative to controls, PI participants showed reduced sleep effort, reduced sleep performance anxiety and reduced SOL measured subjectively. SOL and anxiety change were significantly correlated, although SOL change amongst PI participants was most strongly associated with sleep effort change. Together, therefore, findings support a mediational performance anxiety model of PI. In other words, sleep effort and sleep anxiety are mechanisms of change in PI (cf. Ascher & Turner, 1979), and inhibitory to normal sleep function (cf. Espie, 2002).

Findings are also consistent with Wegner's model of ironic mental control (Wegner, 1994). As noted, there was a strong association between reduced sleep effort and reduced subjective SOL amongst PI participants. Thus, for insomniacs high in sleep anxiety, SOL was reduced when attempts were made to stay awake. This raises the possibility that, under high cognitive load (i.e. sleep performance anxiety), with the intentional operating system undermined, PI caused the ironic monitoring process to detect restful cognitions, leading to a perception of faster sleep onset (cf. Ansfield, Wegner, & Bowser, 1996; Wegner, 1994).

As well as supporting current theoretical conceptualizations of PI, findings may have important clinical implications. If, as the data suggest, PI institutes sleep improvement by reducing sleep effort and sleep anxiety, the approach may prove particularly beneficial to insomniacs high in these variables at assessment. There is already some evidence of a link between elevated performance anxiety and response to PI in the social anxiety literature (Ascher & Schotte, 1999). Research should now clarify more closely the association between pre-treatment sleep effort, sleep anxiety, and subsequent response to PI. Clinical assessment of these constructs is certainly feasible, and if elevated sleep effort/anxiety does predict outcome, this raises the prospect of routine screenings for stand-alone PI.

Turning to the sleep data, recognizing the lack of PI research employing objective measures, the present study obtained actigraphic *and* subjective sleep data. Contrary to hypotheses, no significant objective SOL reduction or sleep efficiency increase was observed amongst PI participants, relative to controls. The subjective sleep data did, however, indicate a marginal trend for reduced SOL amongst PI participants, and the treatment effect size was ''moderate'' (cf. Cohen, 1988). One interpretation of this may relate to actigraphic measurement error. Although actigraphic measures of sleep duration and total wake time correlate highly with Polysomnography (PSG) data (Mullaney et al., 1980; Sadeh et al., 1995), lower agreement rates for SOL have been reported (Blood, Sack, Percy, & Pen, 1997; Hauri & Wisbey, 1992). The accuracy of actigraphy in distinguishing sleep from wakefulness has also been questioned (Pollak, Tryon, Nagaraja, & Dzwonczyk, 2001; Verbeek, Klip, & Declerck, 2001). Perhaps quiet wakefulness in the pre-sleep phase was coded actigraphically as ''sleep'', lowering objective SOL scores, relative to subjective values (see Table 1), washing out any possible objective SOL effect.

Alternatively, it is possible PI shifted participants' perceptions of their sleep deficit.

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Lower objective relative to subjective SOL scores were observed amongst *both* participant groups at baseline (see Table 1). Interestingly, this apparent overestimation of SOL was *only* observed amongst controls at Week 2. This raises the intriguing possibility that PI reduced participants' tendency to overestimate subjective SOL relative to objective criterion. Harvey (2002) has suggested that excessive sleep anxiety, and an attentional bias for sleep-related threat triggered by this anxiety, causes insomniacs to overestimate their sleep deficits. Perhaps by lowering sleep anxiety, PI reduced participants' tendency to overestimate time to sleep (cf. Harvey, 2002). This alteration in perception may also have involved reduced activation of metacognitive sleep beliefs (e.g. ''thinking about sleep means I'm a poor sleeper''), as the less sleep anxious PI participants experienced fewer sleep-related intrusions (Wells, 2001). Further research within an experimental cognitive framework will be needed to determine whether the present findings are indeed due to a shift in PI participants' estimations of SOL or arose due to actigraphic insensitivity.

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