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Original Article

Cognitive processes mediate the effects of insomnia treatment: evidence from a randomized wait-list controlled trial



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ABSTRACT

Introduction: Both guided online and individual face-to-face cognitive behavioral therapy for insomnia (CBT-I) are effective in improving insomnia symptoms and sleep efficiency. Little is known about the underlying mechanisms generating this effect. The present study tests the assumption that pre-sleep arousal, sleep-related worry and dysfunctional beliefs about sleep are mediators in the effect of cognitive behavioral treatment for insomnia.

Methods: A secondary analysis was performed on data previously collected from a randomized controlled trial ($N = 90$). In this trial, participants were randomized to either a face-to-face CBT-I condition, an internet-delivered CBT-I condition, or a wait-list group. This article reports on the efficacy of these interventions on pre-sleep arousal, sleep-related worry, and dysfunctional beliefs. Furthermore, we investigated whether these measures mediated the treatment effect on insomnia severity and sleep efficiency.

Results: Both treatment modalities were efficacious for these cognitive measures; however, face-to-face treatment showed superiority over the online treatment. All three cognitive measures mediated the effect on insomnia severity. Sleep-related worry and pre-sleep arousal mediated the effect on sleep efficiency, but dysfunctional beliefs did not.

Conclusion: Overall, these results point toward the importance of cognitive processes in the treatment of insomnia, implying that psychological treatments for insomnia may best be guided by (also) targeting these cognitive processes.

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1. Introduction

Chronic insomnia is a disorder with a prevalence of 6–10% in the general adult population [1]. Insomnia is characterized by dissatisfaction with sleep quantity and quality, coupled with difficulties getting to sleep and/or maintaining sleep for at least three months for three nights per week. These sleep problems need to significantly affect daytime functioning in order to satisfy the formal criteria of the sleep disorder insomnia [2].

Insomnia has a serious negative impact on quality of life [3] and comes with high societal costs [4]. Insomnia is related to several daytime complaints such as problems with memory and

concentration [5], and is a risk factor for cardiovascular disease [6], and psychiatric disorders such as anxiety and depressive disorders [7].

Cognitive behavioral therapy for insomnia (CBT-I) is currently the treatment of choice for insomnia [8,9]. CBT-I is more effective in the long term than sleep medication [10,11]. Furthermore, several meta-analyses have demonstrated large treatment effects for CBT-I [12–14]; for instance, van Straten et al., [14] reported a wait-list controlled effect size of $d = 1.0$ for insomnia severity.

Unfortunately, there is a shortage of trained CBT-I therapists [15]. For this reason, CBT-I is now increasingly provided via the internet where therapist time may be reduced, and less training may be required. Guided internet-delivered CBT-I is effective with moderate to large treatment effects (Cohen's $d = 0.6–1.1$) [16], and is preferably offered with some form of guidance [17].

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Guided internet-interventions have shown equivalent treatment effects compared to regular face-to-face CBT for other somatic and psychological disorders (Cohen's $d = -0.01$ /Hedges $g = 0.05$) [18,19]. However, for insomnia, the findings on the comparative efficacy of CBT-I between guided online and face-to-face CBT-I are scarce and inconclusive. A recent study of Blöm and colleagues [20] showed no difference in effect between guided-online and face-to-face CBT-I (Cohen's $d = 0.1$ on insomnia severity. In our work, we compared guided-online and face-to-face treatment to a wait-list control condition. Compared to the wait-list, we observed large treatment effects for guided-online and face-to-face treatment on insomnia severity ($d = 1.0/d = 2.3$) and sleep efficiency ($d = 0.6/d = 0.8$). In this trial, face-to-face CBT-I was superior over the online condition on insomnia severity (Cohen's $d = 0.9$) [21].

Despite these generally effective treatments, a central problem remains that even after the most effective treatments, 20–30% of insomnia sufferers still do not achieve a therapeutic response [5]. In order to improve treatments, it is essential to know more about the underlying or maintaining factors that bring about the treatment effects [22]. In recent years there have been some attempts to find mechanisms and cognitive processes appear to be among the most promising candidates for explaining the efficacy of insomnia treatments [23].

Several models have put forward the importance of cognitive processes in insomnia (eg, [24,25]). The cognitive model of Harvey [26] is specifically focused on this aspect. The model posits that an interplay between dysfunctional beliefs and safety behaviors leads to excessive negatively toned cognitive activity about sleep (such as worry). The excessive negatively toned cognitive activity about sleep then leads to arousal and distress, selective attention, and increased monitoring of sleep-related threats (all interrelated in the model). These processes build up to a distorted perception of the deficit, which in turn causes the actual impairment in sleep and daytime functioning. In the cognitive model, all these processes are supposed to play a unique part in maintaining the insomnia disorder. For this paper, we focus on three parts of the cognitive model of Harvey: dysfunctional beliefs, excessive negatively toned cognitive activity, and 'arousal and distress.' If these cognitive processes are indeed perpetuating factors of insomnia, ameliorating these should lead to a decline of the insomnia complaints as well. Stated differently, if these cognitive processes are important treatment mechanisms, they will mediate the treatment effect for insomnia disorder.

Recently, several articles have appeared that report on cognitive processes as mediators in the CBT-I treatment of insomnia, with inconsistent findings. For example, it was observed that a decline in insomnia severity was mediated by a decline in dysfunctional beliefs about sleep [27–31]; however, Okajima and colleagues did not observe this mediational effect [32]. Furthermore, we found that dysfunctional beliefs mediated the effects of CBT-I for sleep efficiency [30] although Espie and colleagues did not observe this relationship [28].

For arousal and excessive negatively toned cognitive activity, the evidence for mediators is scarcer. Pre-sleep arousal was observed to mediate both the decline in insomnia severity and sleep efficiency by Vincent and Walsh [33]; however, Sunnhed and Jansson-Fröjmark [34] found no evidence for pre-sleep arousal as a mediator for insomnia severity. For negatively toned cognitive activity, we were only able to identify two studies that conducted analyses on the relationship of the decline in sleep-related worry and the decline of insomnia severity; one study indeed identified sleep-related worry as a mediator [29], while the other study showed an association between the two constructs [34].

To conclude, even though several research groups on both a theoretical level and on an empirical level state the importance of

cognitive processes in insomnia, there have been very few studies that investigated whether the changes in these cognitive processes indeed mediate the treatment effects of CBT-I. The limited number of studies that have been published report inconclusive results, especially for pre-sleep arousal and sleep-related worry. To extend this literature, we performed a secondary data-analysis on an earlier randomized controlled trial comparing online to face-to-face treatment for insomnia [21]. In line with the results of that trial and the data about cognitive processes, we predicted:

1. Both face-to-face and guided internet-delivered CBT-I are more effective than a wait-list on dysfunctional beliefs about sleep, negatively toned cognitive activity (sleep-related worry), arousal and distress (pre-sleep arousal);
2. Face-to-face CBT-I is more effective than guided internet-delivered CBT-I on pre-sleep arousal, dysfunctional beliefs about sleep, and sleep-related worry;
3. Pre-sleep arousal, dysfunctional beliefs about sleep, and sleep-related worry mediate the treatment effects of CBT-I on insomnia severity and sleep efficiency.

2. Method

2.1. Participants

We performed a secondary data analysis on the study of Lancee and colleagues [21]. The primary study was approved by the internal Ethical Review Board of the University of Amsterdam and was registered at [Clinicaltrials.gov](https://clinicaltrials.gov) (NCT01955850). In this study, participants were recruited through a popular-science website from April 2013 to January 2014 (www.insomnie.nl) and through a Facebook campaign. The actual registration for the study took place in October 2013 and January 2014. A total of 183 participants completed the informed consent and the online questionnaires. Inclusion criteria for the study were as follows: (1) insomnia disorder according to the DSM-5 (difficulty initiating sleep and/or staying asleep; consequences during the day) [2]; (2) Insomnia Severity Index (ISI) score of 10 or higher [35]; (3) Sleep onset latency and/or wake after sleep onset for at least 30 min for at least three nights per week; and (4) at least 18 years or older. Exclusion criteria were: (1) sleep apnea (measured by a subscale of the SLEEP-50 cutoff > 15) [36]; (2) shift-work; (3) pregnancy; (4) earlier CBT-I; (5) start of psychotherapy in the past six months; (6) major depressive disorder based on the Structured Clinical Interview for DSM-IV disorders (SCID-I assessment via telephone); (7) alcohol abuse (more than three units alcohol per day for at least 21 days per month); (8) marijuana use more than once a week; (9) self-reported diagnosis of schizophrenia or psychosis; and (10) current suicidal plans.

Following these criteria, the online assessment resulted in the exclusion of 47 participants from the study. The remaining participants ($N = 136$) were invited to a telephone-screening interview for insomnia (DSM-5) and depression (SCID-I), conducted by a psychologist. Another 46 participants were excluded at this stage (see Fig. 1 for a flowchart). Of the final sample ($N = 90$), 81.1% ($n = 73$) was female, and participants had a mean age of 41.6 years old ($SD = 13.7$). About a third of the participants used prescribed sleep medication ($n = 28$; 31.1%), virtually no participants were currently in psychological treatment ($n = 3$; 3.3%), about half lived together with a partner ($n = 48$; 53.3%), most were employed ($n = 74$; 82.2%), and about half had insomnia for more than five years ($n = 47$; 52.2%). No differences between the groups on the demographic variables were observed at baseline. The participants were randomly distributed over the online treatment condition

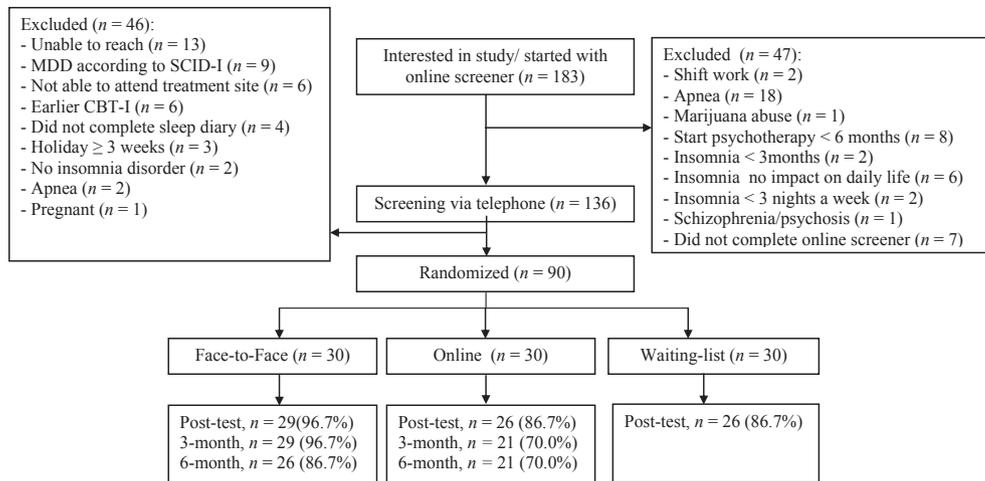


Fig. 1. Flowchart.

($n = 30$), the face-to-face treatment condition ($n = 30$) and the wait-list condition ($n = 30$).

2.2. Power

The sample size of the primary study was based on a power analysis for a within-between interaction effect size of $f = 0.20$ (alpha of 0.05 and a power of 0.80); for the analysis to detect this effect size, 22 participants per group were required. To account for prospective dropout, we recruited 30 participants per condition.

3. Materials

3.1. Mediators

Dysfunctional beliefs about sleep were measured using the 16-item Dysfunctional Beliefs and Attitudes about Sleep Scale (DBAS-16). The DBAS-16 has good internal consistency ($\alpha = 0.77 - 0.79$; $\alpha = 0.82$ in this sample) and good test-retest reliability ($r = 0.83$) [37]. The sum of the DBAS score was averaged so that the total score ranged from 0 (no dysfunctional beliefs) to 10 (severe dysfunctional beliefs).

Sleep-related worry was measured by the Anxiety and Preoccupation about Sleep Questionnaire (APSQ). The APSQ is a 10-item questionnaire with items that can be answered from 1 (strongly disagree) to 5 (strongly agree), with higher scores indicating more sleep-related worry. This self-report questionnaire has good internal consistency ($\alpha = 0.91$; $\alpha = 0.84$ in this sample) and is a valid measure to assess worry in people with insomnia [38].

Sleep-related arousal was measured with the Pre-sleep Arousal Scale (PSAS) [39]. The PSAS consists of 16 items that range from 1 ('not at all') to 5 ('extremely'), higher scores indicating more arousal. Originally, the PSAS consists of two 8-item subscales concerning either cognitive or somatic arousal ($\alpha = 0.76-0.81$). In correspondence with Vincent and Walsh [33], we report in this study on the sum-score of the PSAS ($\alpha = 0.84$ in this sample).

3.2. Dependent variables

Insomnia symptoms were measured using the Dutch version of the 7-item Insomnia Severity Index (ISI). This questionnaire generally has a good internal consistency ($\alpha = 0.74$) and is a valid measure to assess insomnia [40]. ISI items are answered on a 5-point Likert scale (range 0–4); the total score ranges from 0 to

28, higher scores indicating higher severity. As we reported in the primary study [21], the reliability of the ISI was low due to the first item of the questionnaire (about difficulty falling asleep ($\alpha = 0.22$ with an $\alpha = 0.57$ without the first item)). Furthermore, there is considerable conceptual overlap between the last item of the ISI ('how worried/distressed are you about your current sleep problem') and items from the APSQ. Therefore, we performed the mediation analyses for the ISI without items one and seven (the resulting scale had an $\alpha = 0.54$). Please see Footnote 1 for information about the mediation analyses with these items included.

All participants kept a Dutch translation of the Consensus Sleep Diary [41] for seven consecutive days. In this diary, they logged several aspects of their sleep. For this report, only the sleep efficiency (SE) is relevant. Sleep efficiency was calculated by: $SE = [TST/TIB] \times 100$, where TST = total sleep time and TIB = time in bed.

3.3. Other

Depressive symptoms were measured with the Dutch translation of the Centre of Epidemiological Studies Depression scale. The 20-item CES-D ranges from '0' (no depressive symptoms) to '60' (high depressive symptoms). The scale has shown strong reliability in both the original and Dutch language ($\alpha = 0.79-0.92$; $\alpha = 0.88$ in this sample) [42,43].

3.4. Intervention

In both the online and face-to-face CBT-I we employed a multicomponent treatment for insomnia that packaged: (a) psycho-education; (b) progressive muscle relaxation; (c) sleep hygiene and stimulus control; (d) sleep restriction (ie, restricting time in bed to actual sleeping time); (e) cognitive exercises (ie, challenging the misconceptions about sleep) [44]. The online treatment, first described by Lancee and colleagues [45], comprised of six weekly sessions, and participants received feedback by e-mail up to eight weeks. Feedback was provided by masters level psychology students, supervised by the first author. In the face-to-face condition, six 45-minute individual treatment sessions were administered by a psychologist specialized in insomnia treatment. For a more elaborate description of the intervention and the sleep restriction protocol, please see [21].

3.5. Procedure

Participants who met the study criteria first received a short information leaflet and an informed consent form with pertinent information about the study. After digitally signing the informed consent, participants completed the online questionnaires and sleep diary (for seven days). Subsequently, participants were randomly assigned to the guided-online treatment condition (online CBT-I), the individual face-to-face treatment condition (face-to-face CBT-I), or the wait-list condition. Neither experimenters nor participants were blind to treatment allocation. Twelve weeks after the randomization and start of the study (about four weeks after completing the treatment), all participants received a posttest assessment with the same questionnaires and the same seven-day sleep diary as the pretest. After that, participants in the wait-list condition received online treatment for insomnia. Participants in the two active treatment conditions also received measurements three and six months after the post-test (FU1 and FU2). For a more elaborate description of the study procedure, please see [21].

3.6. Statistical analyses

Generalized mixed (multilevel) regression analyses were carried out to evaluate the effects of the two interventions compared to the wait-list at post-test and their sustained effects over the course of the two follow-up assessments. Regular linear mixed regression with an identity link was used for normally distributed data (APSQ, DBAS, and pre-sleep arousal at post-test), negative binomial regression with a log link in case of skewness (pre-sleep arousal at follow up assessments). Data were analyzed in accordance with the intention-to-treat principle [46]. Assuming data to be missing as random, we used an unstructured covariance structure as the best fitting model for the data.

In the parent study [21] we observed that there were baseline differences in depression symptoms between the face-to-face and the wait-list condition. Furthermore, in the online condition, high depression scores were related to non-response to the post-test in the online condition. For this reason, short-term effects (pre-versus post-test) were examined using baseline depression scores (CES-D) [42]. Long-term effects (post-assessment-FU1-FU2) were examined using both the CES-D baseline and APSQ at baseline as covariates because baseline APSQ scores predicted non-response to the follow-up measurements in the face-to-face intervention group. Effect sizes reported are within-group Cohen's *d* effect sizes (based on multilevel estimated baseline and post-test means and pooled standard deviations at baseline) and between-group Cohen's *d* effect sizes (calculated by dividing the estimated difference in change scores between two groups by the pooled standard deviations at baseline; [47]).

For the mediation analyses, we decided to collapse the online and face-to-face CBT-I condition. We did not expect specific mediation effects for the online vs. face-to-face condition since two treatments employed the same intervention modules. Furthermore, collapsing the two conditions increased our statistical power. We do report the 'un-collapsed' mediation analyses in the Results section and Supplemental Table S6. We used the PROCESS tool in SPSS [48]. The PROCESS tool uses a bootstrapping method, which is a non-parametrical procedure that generates an estimate of the sample based on several re-samples. The reason for using this bootstrapping procedure was because this procedure does not require the sampling distribution of the indirect effect to be normally distributed. Mediation was tested by evaluating the 95% confidence interval of the indirect effect (see Fig. 2 for the mediation models). We employed a procedure similar to the one we described in our earlier study [30]. For the

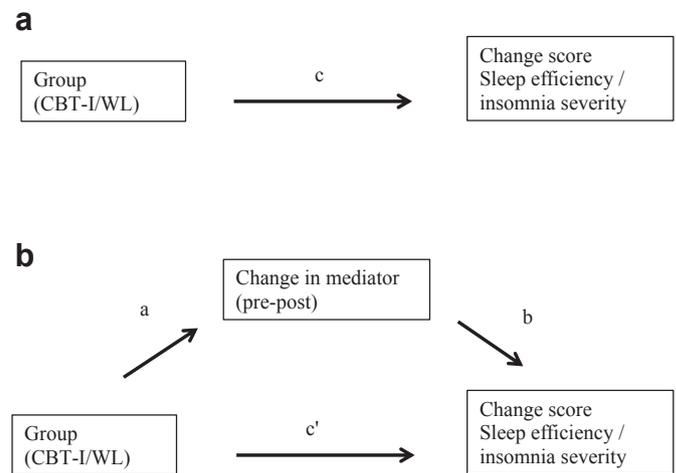


Fig. 2. Fig. 2a: path *c* is the total effect of group on the outcome variables without the influence of the mediator variable. Fig. 2b, path *a* is the effect of group on the mediator variable, path *b* is the effect of the mediator on the outcome variable. Path *ab* is the indirect effect (or mediation effect). Path *c'* is the effect of the group controlling for the mediator variables.

current analyses, we used $n = 50,000$ bootstrap re-samples and used change scores (pre-post) for both the mediator and the dependent variable. Moreover, because of baseline differences, we used baseline depression scores (CES-D) as a covariate in all the mediation analyses. We calculated the effect size of the indirect effect with $1 - c'/c$. With this formula, the proportion of the effect of the independent variable on the dependent variable, which is accounted for by the mediator, is calculated. Possible values lie between 0 and 1 [49].

4. Results

4.1. Multilevel analyses

At post-test, multilevel regression analyses showed significant Condition \times Time interactions for dysfunctional beliefs, $F(2, 78) = 25.69, p < 0.001$; sleep-related worry, $F(2, 77) = 19.90, p < 0.001$; and pre-sleep arousal, $F(2, 78) = 18.88, p < 0.001$. Specifically, participants undergoing face-to-face treatment showed larger decreases than wait-list participants in sleep related worry; $F(1, 54) = 54.73, p < 0.001$; in pre-sleep arousal, $F(1, 53) = 36.25, p < 0.001$; and dysfunctional beliefs, $F(1, 53) = 74.12, p < 0.001$. Online treatment also outperformed the wait-list condition on all variables: sleep related worry, $F(1, 49) = 9.93, p = 0.003$; pre-sleep arousal, $F(1, 48) = 12.63, p = 0.001$; and dysfunctional beliefs, $F(1, 49) = 13.28, p = 0.001$. Additionally, face-to-face treatment outperformed the online treatment on dysfunctional beliefs, $F(1, 54) = 8.91, p < 0.01$; on sleep-related worry, $F(1, 53) = 7.37, p < 0.01$; and on pre-sleep arousal, $F(1, 55) = 4.42, p = 0.04$. Please see Fig. 3 and Tables 1 and 2 for the estimated means and the corresponding Cohen's *d*'s (and Supplemental Table S1–S4 for the observed means and the multilevel regression coefficients). At follow-up, improvements in the two treatment effects were stable: no significant time effects or Condition \times Time interaction effects were found when comparing post-assessment vs. follow-up 1 vs. follow-up 2: sleep-related worry, $F(2, 46) = 0.20, p = 0.818$; dysfunctional beliefs, $F(2, 49) = 0.30, p = 0.742$; and pre-sleep arousal, $F(2, 43) = 0.29, p = 0.75$. The differences between face-to-face treatment and online treatment remained significant over time from post-test to the second follow-up, with face-to-face outperforming online treatment on sleep-

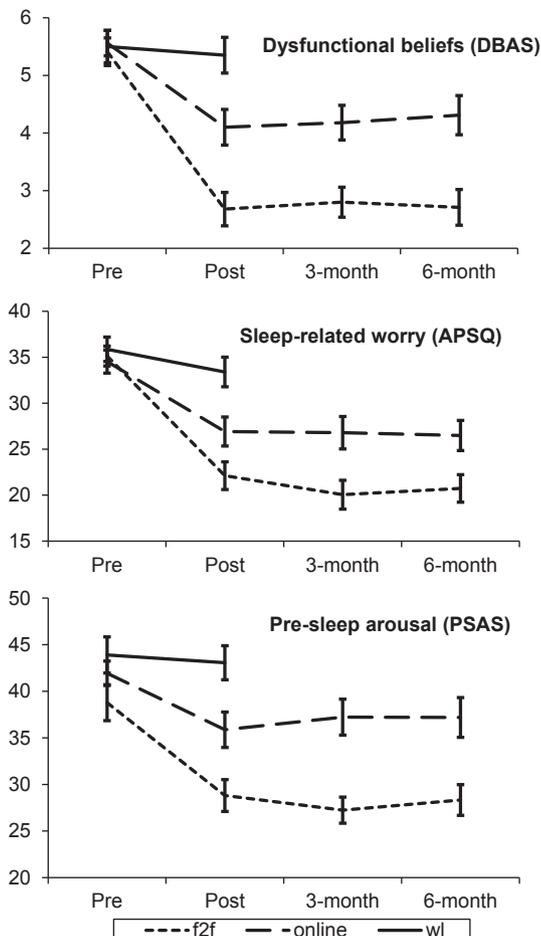


Fig. 3. Dysfunctional beliefs (DBAS), sleep-related worry (APSQ) and pre-sleep arousal (PSAS) scores on all time points for face-to-face, online and wait-list groups. The figure is based on the estimated means. Error bars represent SEM.

related worry, $F(1, 49) = 9.96$, $p = 0.003$, pre-sleep arousal, $F(1, 44) = 20.295$, $p < 0.001$, and dysfunctional beliefs, $F(1, 50) = 16.47$, $p < 0.001$.

4.2. Single mediation analyses

We conducted mediation analyses with the pre-post change scores of the Insomnia Severity Index (ISI) with sleep efficiency (SE) as dependent variables. The pre-to-post change scores of the dysfunctional beliefs (DBAS), sleep-related worry (APSQ), and pre-sleep arousal (PSAS) were included as mediator variables in the analyses, and the severity of depressive symptoms (CES-D) was added as a covariate. Please see Supplemental Table S5 for the zero-order baseline correlations.

¹ As described in the 'measurements' section we decided to use the ISI without item 1 & 7. We also ran the mediation analysis with either item 1 or 7 or all items included. The bootstrap CI changed slightly but the observed effects stayed within the 95% CI indicating that the observed effects were robust. The same applies, for the mediation analyses with the two Pre-arousal subscales (cognitive/somatic – with the exception that the somatic subscale was no mediator for sleep efficiency). We also carried out reciprocity mediation analyses (ISI/SE as mediator and DBAS/Pre-arousal/APSQ as outcomes). In these analyses we found roughly the same but smaller mediation effects, indicating bi-directional relationships: DBAS was mediated by the ISI (46% variance explained) but not by SE. APSQ was mediated by the ISI (42%) and SE (44%). Pre-arousal was mediated by the ISI (44%) but not SE.

As can be seen in Table 3, dysfunctional beliefs, $b = 3.57$, 95% BI [2.42, 4.97] (83% of the effect explained by the mediator), sleep-related worry, $b = 2.46$, 95% BI [1.34, 3.97] (58% explained variance), and pre-sleep arousal, $b = 2.84$, 95% BI [1.76, 4.28] (66% explained variance) mediated the treatment effects of CBT-I on insomnia severity. A decline in these cognitive measures was related to a decline in insomnia severity.¹ Additionally, sleep-related worry, $b = 5.51$, 95% BI [2.11, 10.06] (79% explained variance) and pre-sleep arousal, $b = 3.33$, 95% BI [0.25, 7.52] (48% explained variance) mediated the effects on sleep efficiency; the change in dysfunctional beliefs did not. A decline in sleep-related worry and pre-sleep arousal was related to an increase in sleep efficiency.

We also carried out the mediation analyses for the separate interventions (ie, non-collapsed; online vs. wait-list and face-to-face vs. wait-list). The same set of variables emerged as significant mediators, with the only exception that in the online condition, sleep efficiency was now mediated by dysfunctional beliefs and sleep-related worry but not by pre-sleep arousal. These mediation analyses can be found in Supplemental Table S6.

4.3. Multiple mediation analyses

In the multiple mediation analyses, all mediator variables were included in a single model (Table 3). For the Insomnia Severity Index, only dysfunctional beliefs and pre-sleep arousal remained as significant mediators. The three cognitive variables together explained 97% of the variance, $b = 4.40$, 95% BI [3.07, 6.19]. For sleep efficiency, only sleep-related worry survived the multiple mediation analyses. The three variables together explained 71% of the variance, $b = 4.93$, 95% BI [0.58, 10.11].

5. Discussion

This study examined the efficacy of face-to-face and guided online CBT-I on dysfunctional beliefs, sleep-related worry, and pre-sleep arousal and whether these cognitive processes mediate the treatment effect. As expected, we found a strong effect for both interventions: participants in the face-to-face and online CBT-I conditions reported moderate-to-large decreases in dysfunctional beliefs, pre-arousal, and sleep-related worry compared to the wait-list. The observed treatment effects of CBT-I on these cognitive measures are in line with previous research [27,30,34,50–52].

Furthermore, the effects on pre-arousal, sleep-related worry and dysfunctional beliefs were superior for face-to-face CBT-I compared to online CBT-I. This is in line with the primary study that demonstrated the superiority of face-to-face treatment over online treatment [21]. Whether face-to-face treatment is generally better than online treatment for insomnia remains an open, empirical question. As discussed in the introduction, there have been other sleep-related studies that did not observe differences between these two modalities [53], and relatedly, a recent meta-analysis found no difference between online and face-to-face treatment for psychiatric and somatic disorders [19].

Next, we considered the mediating role of sleep-related beliefs, negative cognitive activity, and pre-sleep arousal on insomnia severity and sleep efficiency (with both treatment conditions collapsed). For insomnia severity, as expected, pre-arousal (66% explained variance), sleep-related worry (58%) and dysfunctional beliefs (83%) all mediated the effect of treatment on insomnia severity: A larger decline in these measures meant a larger decline in insomnia severity. In the multiple mediation analyses, only dysfunctional beliefs and pre-sleep arousal remained mediators of the effects. These findings were in general consistent with the extant literature. For dysfunctional beliefs, five earlier studies also

Table 1
Pre- and (estimated) post-test means with corresponding between-group Cohen's d effect sizes for the online, face-to-face (f2f), and wait-list (wl) condition.

	Pre-test means (SE)			Post-test means (SE)			Cohen's d		
	f2f	Online	WL	f2f	Online	WL	f2f × online	f2f × WL	Online × WL
Dysfunctional beliefs (DBAS)	5.41 (0.24)	5.56 (0.22)	5.50 (0.28)	2.68 (0.29)	4.10 (0.31)	5.35 (0.31)	1.0**	1.8***	1.0**
Sleep-related worry (APSQ)	35.13 (1.08)	34.53 (1.26)	35.87 (1.33)	22.12 (1.51)	26.92 (1.58)	33.40 (1.61)	0.8**	1.6***	0.7**
Pre-sleep arousal (PSAS)	38.77 (1.93)	41.93 (1.31)	43.90 (1.94)	28.81 (1.71)	35.87 (1.90)	43.06 (1.83)	0.4*	0.9***	0.6**

Note. * = $p < 0.05$; ** = $p < 0.01$; *** = $p < 0.001$. APSQ = Anxiety and Pre-occupation about Sleep Questionnaire; DBAS = Dysfunctional Beliefs and Attitude about Sleep scale; PSAS = Pre-Sleep Arousal Scale. Please see Supplemental Table S1 for the observed means and Supplemental Table S3 for the multilevel regression coefficients. f2f = Face-to-face; WL = Wait-list.

Table 2
Estimated three- and 6-month follow-up means with corresponding Cohen's d scores for the online and face-to-face (f2f) condition.

	3-month FU means (SE)		6-month FU means (SE)		Cohen's d f2f × online	
	f2f	Online	f2f	Online	3-month	6-month
Dysfunctional beliefs (DBAS)	2.80 (0.26)	4.18 (0.30)	2.71 (0.31)	4.31 (0.34)	1.0***	1.1***
Sleep-related worry (APSQ)	20.05 (1.57)	26.79 (1.77)	20.73 (1.50)	26.49 (1.64)	1.1**	1.0**
Pre-sleep arousal (PSAS)	27.24 (1.40)	37.22 (1.93)	28.33 (1.65)	37.19 (2.13)	0.8***	0.6***

Note. * = $p < 0.05$; ** = $p < 0.01$; *** = $p < 0.001$. APSQ = Anxiety and Pre-occupation about Sleep Questionnaire; DBAS = Dysfunctional Beliefs and Attitude about Sleep scale; PSAS = Pre-Sleep Arousal Scale. Please see Supplemental Table S2 for the observed means and Supplemental Table S4 for the multilevel regression coefficients. f2f = Face-to-face; WL = Wait-list.

found that it mediated CBT-I effect on insomnia severity [27–31] and only one did not [32]. For sleep-related worry, there was one study identifying it as a mediator [29] and one study that found associations between a decline in sleep-related worry and insomnia severity [34]. For pre-arousal findings appear inconclusive, with one study reporting the effect [33] and one not [53].

For sleep efficiency, our results were somewhat mixed. In line with our hypotheses, the increase in sleep efficiency was mediated by a decline in sleep-related worry (explaining 79% of the variance) and pre-arousal (48%). The latter was also a mediator in an earlier study [33], but was absent in another [53]. Sleep-related worry was the only mediator that survived the multiple mediation analyses. We could not identify any earlier study that investigated it as a mediator for sleep efficiency, but the results are in line with earlier work with insomnia severity as an outcome [29]. Dysfunctional beliefs did not mediate the treatment effect of sleep efficiency in our sample, in contrast to our own earlier work [30], but in line with Espie and colleagues [28]. A possible reason why the findings on sleep efficiency are less consistent may be that the magnitude of its treatment effect is smaller than for insomnia severity and that mediation is, therefore, harder to detect.

Before further discussing the implications of these results we would first like to turn toward possible limitations of this study. First and foremost, this study was not set up as a mediation study. As a consequence, we did not have any midpoint measurements, and therefore we have no information on temporality or causality. Therefore, it is unclear whether the changes in the mediators preceded the changes in insomnia symptoms. This is especially notable since reciprocity tests showed that the cognitive measures and the two outcomes had bidirectional relationships (but smaller if the cognitive measures were inserted as outcomes – see Footnote 1). It is critical to identify the direction of these temporal relationships and, accordingly, there is a clear need for studies that are specifically designed to investigate these mediators.

Second, since we used a dataset of a study not specifically set up for testing mediation analyses, we were only able to test a subsample of cognitive measures. For instance, we did not include a measure for selective attention and monitoring nor did we include safety behaviors, both of which feature in the cognitive model of Harvey [26]. Some earlier studies have found selective attention [29] and safety behavior to be mediators [30] in the treatment effect, but other studies did not [31]. In any case, only two studies

Table 3
Dysfunctional beliefs (DBAS), sleep-related worry (APSQ) and Pre-sleep arousal (PSAS) as mediators of online CBT for insomnia outcomes.

Mediator	Dependent variable	Effect of independent variable on mediator (a)	Effect of mediator on dependent variable (b)	Indirect effect (ab), [95% BI]	Direct effect (c')	Total effect (c)
Single mediator analyses						
DBAS	ISI	$b = -2.33, t = -5.87^{***}$	$b = -1.53, t = -6.76^{***}$	$b = 3.57, [2.42, 4.97]$	$b = -0.73, t = 0.78^{ns}$	$b = -4.30, t = 4.36^{***}$
APSQ	ISI	$b = -9.80, t = -5.08^{***}$	$b = -0.25, t = -4.87^{***}$	$b = 2.46, [1.34, 3.97]$	$b = -1.84, t = 1.84^{\phi}$	$b = -4.30, t = 4.36^{***}$
PSAS	ISI	$b = -11.54, t = -5.34^{**}$	$b = -0.25, t = -5.53^{***}$	$b = 2.84, [1.76, 4.28]$	$b = -1.47, t = 1.49^{ns}$	$b = -4.30, t = 4.36^{***}$
DBAS	SE	$b = -2.41, t = -5.69^{***}$	$b = -1.37, t = -1.46^{ns}$	$b = 3.30, [-1.26, 8.28]$	$b = 3.66, t = 0.92^{ns}$	$b = 6.96, t = 2.13^*$
APSQ	SE	$b = -9.34, t = -4.70^{***}$	$b = -0.59, t = -3.10^{**}$	$b = 5.51, [2.11, 10.06]$	$b = 1.45, t = 0.41^{ns}$	$b = 6.96, t = 2.13^*$
PSAS	SE	$b = -10.53, t = -4.60^{***}$	$b = -0.31, t = -1.83^{\infty}$	$b = 3.33, [0.25, 7.52]$	$b = 3.63, t = 0.99^{ns}$	$b = 6.96, t = 2.13^*$
Multiple mediator analyses						
DBAS	ISI	$b = -2.33, t = -5.87^{***}$	$b = -1.09, t = -3.93^{***}$	$b = 2.54, [1.22, 4.09]$	$b = -0.10, t = -0.11^{ns}$	$b = -4.30, t = 4.36^{***}$
APSQ	ISI	$b = -9.80, t = -5.08^{***}$	$b = -0.03, t = -0.49^{ns}$	$b = 0.29, [-1.02, 1.48]$		
PSAS	ISI	$b = -11.54, t = -5.34^{**}$	$b = -0.13, t = -2.82^{**}$	$b = 1.57, [0.37, 3.14]$		
DBAS	SE	$b = -2.41, t = -5.69^{***}$	$b = 0.87, t = -0.71^{ns}$	$b = -2.10, [-8.70, 3.77]$	$b = 2.03, t = 0.52^{ns}$	$b = 6.96, t = 2.13^*$
APSQ	SE	$b = -9.34, t = -4.70^{***}$	$b = -0.66, t = -2.45^*$	$b = -6.12, [1.16, 12.85]$		
PSAS	SE	$b = -10.53, t = -4.60^{***}$	$b = -0.08, t = -0.42^{ns}$	$b = 0.91, [-3.16, 5.27]$		

Note. Independent variable = 'Group'; $\infty = p < 0.1$; * = $p < 0.05$; ** = $p < 0.01$; *** = $p < 0.001$; APSQ = Anxiety and Pre-occupation about Sleep Questionnaire; DBAS = Dysfunctional Beliefs and Attitudes about Sleep scale; ISI = Insomnia Severity Index; PSAS = Pre-sleep arousal scale; SE = Sleep efficiency.

[29,31] have looked at the full interplay of all the constructs of the cognitive model. These two studies provided a fascinating insight into the model, but both studies had specific limitations. Harvey et al. [29] did not include an inactive comparison condition, and Norell-Clarke et al. [31] did not observe treatment effects on several cognitive measures (possibly because of the active control condition and the comorbid sample). In our opinion, future studies should ideally include all relevant variables and employ a design that is optimally suited to investigate mediational effects and allows for a comprehensive test of the full model.

Third, we decided to collapse the two treatment conditions for reasons of statistical power and because we did not expect differential mediation effects between the two conditions (we expected the same mediators in online and face-to-face condition). Even though we deem it a plausible argument, the findings may be confounded by mode of delivery. This, however, seems unlikely: the mediation outcomes generally followed the same pattern when the conditions were analyzed separately (Supplemental Table S6).

Another limitation of the present study is that the sample consisted of self-referred patients who were recruited online. Possibly, this convenience sample may differ from patients who seek help from their general practitioner or from other clinical groups, which would limit the external validity of the present study. Furthermore, we did not include objective measures such as polysomnography and did not include an active control condition. The latter omission leaves open the question of whether or not the effects were due to the active treatment ingredients or non-specific factors.

Notwithstanding these limitations, we hold that this paper makes two main contributions: (1) It shows that several theoretically meaningful cognitive processes can be ameliorated by both online and face-to-face CBT-I, and (2) that these cognitive processes generally mediate the treatment effects. It has yet to be established which of these specific processes in the Cognitive model of Harvey [26] is most central. However, we think it firmly stands that the combination of these cognitive processes plays a major role in the maintenance of insomnia. This implies that psychological treatments for insomnia may best be guided by (also) targeting these cognitive processes.

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Conflicts of interest

None.

The ICMJE Uniform Disclosure Form for Potential Conflicts of Interest associated with this article can be viewed by clicking on the following link: <https://doi.org/10.1016/j.sleep.2018.09.029>.

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