Exploring the Interplay of Insomnia, Depression and Mediating Factors: An analysis of Pre-Sleep Arousal and Dysfunctional Beliefs and Attitudes about Sleep

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Abstract

This study delves into the relationship between insomnia and depression, examining the mediating roles of pre-sleep arousal and dysfunctional beliefs and attitudes about sleep. The research aims to contribute insights on mediating factors that might partially explain the relationship between insomnia and depression. The comprehensive investigation involved an examination of the direct and indirect effects of insomnia on depression. Utilising self-reported questionnaires, the study explored the mediating impact of both Pre-Sleep Arousal (PSA) and dysfunctional beliefs and attitudes about sleep (DBAS) among 362 Dutch participants. Three hypotheses were formulated. Insomnia (x) has a positive effect (c) on depression (y) (H1). PSA(M1) mediates the relationship between insomnia (x) and depression (y) (H2). DBAS (M2) mediates the relationship between insomnia (x) and depression (y) (H3). Two mediation analyses were run showing significant partial mediations for both PSA and DBAS. All hypotheses were confirmed. Limitations include the lack of differentiation in subscales for PSA and HADS, restricting a nuanced understanding of influential factors. Additionally, the use of questionnaires raises questions about the generalizability of the findings to clinical populations. Future research should consider physiological measures alongside self-reported data. The study advocates for the consideration of further research about pre-sleep arousal and dysfunctional beliefs when treating individuals with both insomnia and depression. These mediating variables hold promise in disrupting the connection between these common syndromes. The findings call for further clinical implementations and additional research funding to replicate and explore these connections more specifically and in greater detail.

Keywords

Insomnia, Depression, Pre-Sleep Arousal, Dysfunctional Beliefs, Sleep

Introduction

Sleep is fundamental to mental health: chronic sleep loss has been linked to a range of psychiatric outcomes, including depression and suicidality (Bei et al., 2018; Olgiati & Serretti, 2022). Insomnia, defined as self-reported challenges in initiating or sustaining sleep, is a major risk factor for persistent depressive symptoms (Meaklim et al., 2023). It is proposed to be associated with depression (Neckelmann et al., 2007), defined as a persistent loss of pleasure or interest in activities, accompanied by a melancholic mood (WHO, 2023). Previous findings indicate that in the case of comorbidity between insomnia and depression, the former is not just a symptom of depression, but a condition that requires its dedicated treatment (Blom et al., 2015). Co-occurrence between insomnia and major depressive disorders is common and brings a worse outcome for both conditions (Bei et al., 2018), making insomnia an important risk factor for persistent depressive disorder (Meaklim et al., 2023). When sleep is chronically disrupted, mood, motivation, and cognitive functioning are all compromised, which can deepen depressive symptoms and, in severe cases, contribute to risk of self-harm (Olgiati & Serretti, 2022). Therefore, understanding the mechanisms behind the connection of these two concepts is of critical value. The connection between insomnia and depression is widely established. For instance, depression was found to be correlated with insomnia severity (Park et al., 2012; Doos et al., 2014), and the betterment of insomnia was closely linked to improvements in depression (Cirino et al., 2019). Hence, detecting early changes in insomnia could be useful to foresee outcomes of depression. At the same time, greater and sustained betterments in depression reliably corresponded to lower levels of insomnia (Bei et al., 2018).

One crucial transdiagnostic process that links insomnia with depression is pre-sleep arousal (PSA). Pre-sleep arousal refers to heightened mental activity, uncontrollable worries, racing thoughts, distressing physical sensations like a racing heart, muscle tension, and jittery nervousness occurring during the pre-sleep period (Nicassio et al., 1985; Hoyniak et al., 2021). Although PSA frequently co-occurs with insomnia, it is conceptually and empirically distinct: insomnia describes the experience of disturbed sleep itself, whereas PSA captures a hyperaroused cognitive–somatic state that precedes and contributes to that disturbance (Nicassio et al., 1985; Palagini et al., 2017). Support for a model of insomnia that includes pre-sleep arousal is well-founded, as the two are significantly correlated (Doos et al., 2014). However, the impact of pre-sleep arousal extends beyond sleep difficulties; it has also been found to increase the risk of clinically significant depressive symptoms (Meaklim et al., 2023). Its function as an underlying mechanism is further evidenced by findings that identified it as a significant partial mediator between depression and fatigue, suggesting it acts as a predictor of symptoms in clinically depressed individuals (Karlson et al., 2010). Depression is a strong predictor of both pre-sleep arousal and sleep quality (Gorgoni et al., 2021). When levels of nocturnal cognitive arousal are lower due to mindfulness-based insomnia therapy, they are predictors of a reduction in depression (Kalmbach et al., 2023). Moreover, when it is tested whether pre-sleep arousal explained the association between insomnia and suicidal ideation, a full mediation of cognitive pre-sleep arousal is found (Olgiati et al., 2022).

Beyond pre-sleep arousal, a second cognitive factor that has consistently been implicated in both insomnia and depression is dysfunctional beliefs and attitudes about sleep (DBAS), which have been shown to correlate highly with both insomnia severity and depressive symptoms. Dysfunctional beliefs about sleep involve faulty beliefs and biased perceptions where insomnia sufferers hold unrealistic sleep expectations and excessive worry, leading to emotional distress and perpetuating the cycle of insomnia (Morin et al., 2007). During dCBT-I (digital

Cognitive Behavioral Therapy to treat insomnia), DBAS partially mediated the effects of dCBT-I on insomnia and changes in the DBAS fully mediated changes on the Hospital Anxiety and Depression Scale (HADS) (Faaland et al., 2023). There is evidence that beliefs and attitudes towards sleep may be involved in sustaining insomnia (Morin et al., 1993) and that changes in dysfunctional beliefs mediate improvements in symptoms of insomnia during CBT-I treatment (traditional CBT to treat insomnia) (Parsons et al., 2021). Not only is there a positive correlation between dysfunctional sleep-related cognitions and insomnia severity, but it was shown that the latter acts as a significant determinant for both sleep-related cognitions and depression (Park et al., 2012). On their own, dysfunctional sleep related beliefs and attitudes were found to predict clinically significant depression (Meaklim et al., 2023). Previous research also found that individuals suffering insomnia were found to have higher levels of both DBAS and PSA when compared to sound sleepers (Palagini et al., 2017). Taken together, these findings suggest that DBAS, like PSA, is closely entangled with both insomnia and depression and may therefore be a plausible candidate mechanism in the relationship between the two.

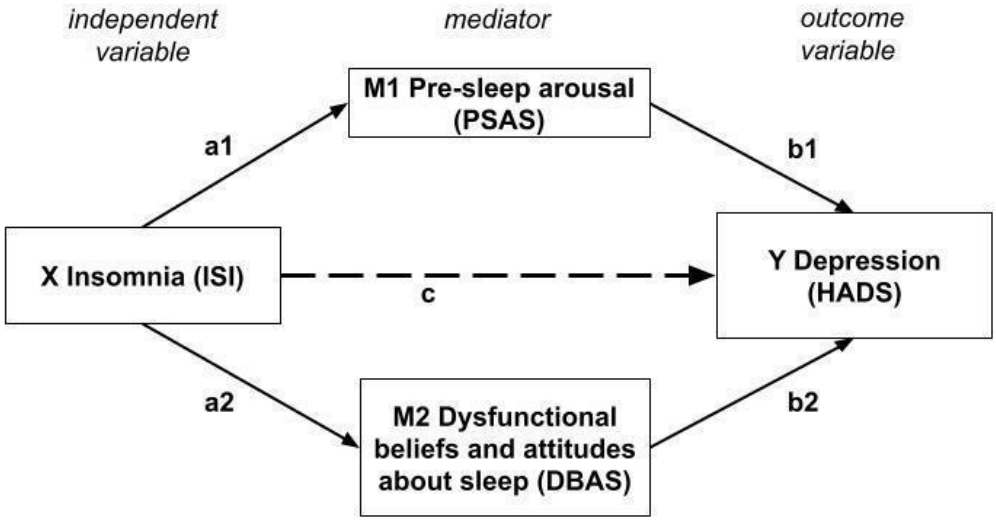
Although the co-occurrence of insomnia and depression is widely established, the specific mechanisms linking the two remain a critical area for investigation. Among the many candidate mechanisms discussed in the literature, pre-sleep arousal (PSA) and dysfunctional beliefs and attitudes about sleep (DBAS) stand out for two reasons. First, both have been independently linked to insomnia severity and to depressive symptomatology (Nicassio et al., 1985; Morin et al., 1993; Palagini et al., 2017). Second, both are explicit targets of evidence-based interventions for insomnia, including cognitive behavioural therapy for insomnia (CBT-I), making them clinically actionable rather than purely descriptive constructs (Parsons et al., 2021; Faaland et al., 2023). However, their specific roles in statistically accounting for the association between insomnia and depression are not yet fully clarified. Clarifying these mechanisms is societally relevant: insomnia and depression are among the most prevalent and disabling mental-health conditions worldwide, and treatments that fail to address the cognitive and arousal-based processes that link them risk leaving patients with persistent residual symptoms. The present study therefore examines the relationship between insomnia and depressive symptoms, with a specific focus on PSA and DBAS as potential mediators. Examining these variables may contribute to a more nuanced understanding of how insomnia and depression co-occur, and may inform future research aimed at identifying clinically relevant intervention targets.

On the basis of previous findings, it was hypothesised that**:**

- **H1:** Insomnia (x) has a positive effect (c) on depression (y)
- **H2:** PSA Scale (M1) mediates the relationship between insomnia (x) and depression (y)
- **H3:** DBAS (M2) mediates the relationship between insomnia (x) and depression (y)

Figure 1

Proposed Mediation Models for the Relationship Between Insomnia and Depression Implicated in the Development and Maintenance of Depression



Path c is the total effect of insomnia on depression without the influence of the potential mediator variable.

Path a1 is the effect of insomnia on the mediator variable PSA Scale and path b1 is the effect of PSA Scale on depression. Path a1*b1 is the indirect effect (including the mediator PSA Scale).

Path a2 is the effect of insomnia on the mediator variable DBAS and path b2 is the effect of DBAS on depression. Path a2*b2 is the indirect effect (with the mediator DBAS).

Methodology

Participants

Four hundred and sixty-nine dutch-speaking adults were recruited from 30th of November until 17th of December 2021. Criteria for inclusion were being of age 18 or above, proficient in Dutch and having an email and internet connection.

Materials

After signing informed consent, participants completed a single online survey hosted on Qualtrics. The survey opened with a short demographic block (age, gender, education level, living situation, and occupational status) and was followed by four self-report questionnaires of interest, measuring insomnia, depressive symptoms, pre-sleep arousal, and dysfunctional beliefs and attitudes about sleep. The four questionnaires were presented in a fixed order for all participants. While randomisation of questionnaire order would have been preferable to control for order effects, this was not implemented in the original data collection, which is acknowledged as a limitation in the Discussion. .

Insomnia Severity Index (ISI)

Insomnia was measured using the Insomnia Severity Index (ISI) developed by Morin (1993). The test includes 7 items designed to assess: (a) the seriousness of difficulties in falling asleep (initial), (b) challenges in maintaining sleep (middle), (c) problems with early morning awakening (terminal), (d) contentment with the existing pattern of sleep, (e) disruptions to daily activities, (f) perceptibility of impairment linked to the sleep issue, and (g) the extent of distress caused by the sleep complaint. Answers can be input on a 5-point Likert-scale ranging from 0 (Very satisfied) to 4 (Very dissatisfied). A sample item is: "How SATISFIED-DISSATISFIED are you with your CURRENT sleep pattern?". A total score is calculated from all items. Total scores range from 0–7 indicating no clinically significant insomnia, to 22–28 hinting Clinical insomnia (severe). The Insomnia Severity Index (ISI) demonstrates sufficient and stable internal reliability with a Cronbach's alpha between 0.76 at baseline and 0.78 at followup and serves as a dependable self-report tool for assessing perceived sleep challenges (Bastien et al., 2001). In this study a high level of internal consistency was found (7 items, α = .82).

Hospital Anxiety and Depression Scale (HADS-D)

Symptoms of depression were measured with the Depression subscale of the Hospital Anxiety and Depression Scale (HADS-D), developed by Zigmond and Snaith (1986). Although the full HADS comprises 14 items measuring both anxiety and depression, only the 7 depression items (HADS-D) were used in the present study, as the focus was on depressive symptoms. The anxiety subscale was therefore not analysed or reported. An example of one of the items is: "Lately I feel like everything is getting more difficult." Responses are given on a 4-point Likert scale ranging from 0 (not at all) to 3 (almost always). The sum of all items is the total score and the HADS-D total score ranges from 0 to 21, with a score between 0 and 7 indicating no clinically relevant depression, and a score between 11 and 21 as a threshold for suspected depression. The HADS-D has demonstrated effectiveness in evaluating the severity of symptoms and the presence of depression across somatic, psychiatric, and primary care patients, as well as in the general population with a Cronbach's alpha values that for the HADS-D ranged from .67 to .90 (average.82) (Bjelland et al., 2002). In this study a high level of internal consistency was found (14 items α = .88).

Pre-Sleep Arousal Scale (PSAS)

To measure pre-sleep arousal the Pre-sleep arousal scale (PSAS) developed by Nicassio (1985) was used. The scale is made of 16 items where participants have to give an input. An example item in the questionnaire is: "A jittery, nervous feeling in your body". Participants have to give an answer on such items on a 5-point Likert scale ranging from 1 (not at all) to 5 (extremely). A total score is calculated from the input on all items. The first 8 items measure somatic pre-sleep arousal with a sum score (PSAS-somatic), and the 8 items that follow measure cognitive pre-sleep arousal (PSAS-cognitive). The clinically relevant threshold for PSAS-somatic and PSAS-cognitive are of a value of ≥14 for the somatic items and ≥20 for the cognitive items. Cronbach's alpha of 0.87 for the cognitive scale and of 0.83 within the somatic scale were found (Gorgoni et al. 2021) In this study a high level of internal consistency was found within the two scales combined (16 items α = .89).

Dysfunctional Beliefs and Attitudes about Sleep Scale (DBAS)

A scale developed by Morin (1993) was used to quantify dysfunctional beliefs and attitudes about sleep (DBAS). The questionnaire is made of 28 items where participants have to fill in. An item from the questionnaire can be: "I am sometimes afraid of dying in my sleep". Participants are required for each statement, to indicate the

level of agreement or disagreement on a visual analog scale. Each item is answered on a 100-mm line where respondents indicate with a sign the level of agreement with statements about sleep (0="strongly disagree"/ 100= "strongly agree"). Scores are created by recording the millimetres of distance from the start of the line to the response given. A final score is calculated with the average score of all items together, with greater values showing greater dysfunctional beliefs and attitudes. Some researchers are in favour of visual analogue scales and their sensitivity to the smallest differences, some have argued that certain items of the DBAS show low average and variance and they discussed it as evidence of a low sensitivity to individual differences. Cronbach alpha of 0.77 for clinical and 0.79 for research samples were found (Morin et al., 2007). In this study a high level of internal consistency was found (16 items α = .87)

Procedure

The pool of participants was created by unifying two different samples of individuals. Participants for the "insomnia sample" were recruited through www.insomnie.nl, where they voluntarily signed up and consented to be contacted for research. Then, they received an email with a link to the survey on Qualtrics. For the "student sample," a convenience sample was collected by bachelor's students in 2021 using various communication channels, including email, WhatsApp, and Reddit.

The recruitment followed a chronological order. Participants first received an information brochure, followed by a link to the ethically approved questionnaire by the University of Amsterdam (CE number: 2021-COP-14104). The questionnaire, requiring approximately 45 minutes for completion, was not rewarded with research credits. Participants received a reward in the form of a PDF containing self-help sleep tactics for their involvement.

Analysis plan

Only participants who completed every item across all four questionnaires were included in the statistical analysis, ensuring that no values were missing. For each respondent, individual item responses were aggregated into total scores. The primary analysis utilised the total scores of every participant across all measures for the questionnaires of Insomnia Severity Index (ISI), Hospital Anxiety and Depression Scale (HADS-D) and Pre-sleep arousal scale (PSAS), whereas an average score was calculated for the Dysfunctional Beliefs and Attitudes about Sleep scale (DBAS). The two groups were initially recruited through different channels and were therefore expected to differ in their baseline rates of insomnia and depressive symptoms (the website group was self-selected for sleep complaints, whereas the student group was a convenience sample). However, because the present study was not designed to compare the two groups but rather to test within-person associations between insomnia, depression, PSA, and DBAS, group membership was not a relevant grouping variable for the planned mediation analyses. The two samples completed exactly the same set of questionnaires under the same online conditions, and pooling them therefore did not introduce procedural heterogeneity. Combining the two sources also increased statistical power and broadened the range of insomnia severity captured in the data, allowing for a more reliable estimation of the relationships among the variables of interest. As a result, the two groups were treated as a single, unified sample. In the Hospital Anxiety and Depression Scale (HADS-D), items 1, 3, 5, 6, 8, 10, 11 and 13 are reverse-scored, meaning that the values of that item were reversed and only then they could be included in the statistical analysis.

The sample was tested for outliers with the use of boxplots created in SPSS analysing the total and average score of the participants within each questionnaire. If any input from a participant was revealed outside the lower or upper quartile and beyond the 1.5 interquartile range, it was considered an outlier and the input of that participant was removed altogether from the sample (Field, 2017). Descriptive statistics were calculated for the variables of interest and the demographics after removing outliers. The presence of multicollinearity was also checked by means of zero-order correlation. Cronbach's alpha was calculated within SPSS in order to verify the internal reliability of each questionnaire (ISI, HADS-D, PSAS and DBAS).

The hypotheses being tested are that the pre-sleep arousal scale (PSAS) and dysfunctional beliefs and attitudes about sleep (DBAS) mediate the relationship between insomnia and depression. This was investigated with two independent mediation models, the first one with the pre-sleep arousal scale (PSAS) as a mediator (Figure 1: H2/a1*b1) and the second one instead with dysfunctional beliefs and attitudes about sleep (DBAS) as a mediator (Figure 1: H3/a2*b2).

The assumptions necessary to run a mediation analysis were checked. The dependent, independent, and mediator variables should be continuous. These variables of interest should also show a linear relationship, and this was verified by means of a scatter plot. This has been inspected with the Regression Standardised residual on Regression Standardised Predicted Value Plot (Field, 2017).

Mediation analysis was performed in SPSS using the PROCESS tool developed by Hayes in 2012. PROCESS is an add-on of SPSS, a tool that facilitates the modelling and computation of mediation (Hayes, 2012). The two mediation analyses were tested with the same criterion. It was evaluated if the 95% confidence interval was significant for the indirect effect, namely the path a1*b1 and path a2*b2 for the first and second mediation analysis respectively (Figure 1). Partial mediation was established if there was a significant connection not only between the mediator and the dependent variable but also a direct significant link between the independent and dependent variables.

Results

Descriptive Statistics

100 participants were removed from the sample because they did not respond to all items necessary for the study. Within Table 1 the mean and the standard deviation of both main variables and mediators are reported.

Table 1

Correlations, means and standard deviations of model variables

	Mean	Sd	1.	2.	3.	4.
1. HADS	27.13	7.15	.88[a]			
2. ISI	15.12	5.96	.41***	.82[a]		
3. PSAS	35.58	12.20	.68***	.50***	.89[a]	
4. DBAS	4.67	1.79	.45***	.59***	.48***	.87[a]

Note: *p<.05, **p<.01, ***p<.001, [a] Cronbach's alpha

Demographic characteristics of included participants are exposed in Table 2 (Means and standard deviations of demographics for age) and Table 3 (Frequencies of Demographics).

Table 2

Means and standard deviations of age

	Mean	SD	Min	Max
Age	47.39	16.79	18	80

Table 3

Frequencies of Demographics

Demographic Characteristics	n *(N* = 362)	%
Insomnia		
Without	86	23.8
Less than a month	3	.8
1-3 months	6	1.7
Longer	276	73.8
Sex		
Male	104	28.7
Female	258	71.3
Educational level		
None	3	.8
Primary education	3	.8
MAVO/VMBO or comparable	27	7.5
MBO or comparable	66	18.2
HAVO/VWO or comparable	55	15.2
HBO or comparable	129	35.6
Scientific education	79	21.8
Residence		
I live with my partner (married or unmarried)	214	59.1
I live without a partner but with others (housemates, parents, children)	66	18.2
I live alone	78	21.5
Otherwise	4	1.1
Job		
Yes, a paid job	218	60.2
No	106	29.3
Yes, own company	38	10.5
Symptoms duration (insomnia)		
Less than a month	3	.8
1-3 months	6	1.7
Longer than 3 months (namely)	267	73.8

According to the boxplots seven outliers were identified and removed from the sample. This resulted in a total sample size of 362 participants that met all requirements.

Assumption Checks

The assumptions of linearity were met and homoscedasticity was violated only for PSAS. This problem was solved by running the mediation analysis including PSAS with heteroscedasticity robust standard errors. The process analysis was run with a Huber-White standard error. The presence of multicollinearity was also checked by means of zero-order correlation that can be verified in Table 1. The zero-order correlation of the variables was not greater than .80 or .90 so there are no signs of a violation of the assumption of multicollinearity (Field, 2017).

Finally, the Shapiro-Wilk test was used to assess whether the residuals of the regression models were approximately normally distributed. The Shapiro-Wilk test revealed that the normality assumption was met for the model predicting PSAS from ISI, $W(362) = 1.00$, $p = .604$, but was violated for the models predicting HADS from PSAS, $W(362) = .99$, $p = .035$, and HADS from DBAS, $W(362) = .99$, $p = .004$. However, according to the central limit theorem, since the dataset was larger than 30 respondents, the analysis was robust for not normally distributed residuals (Field, 2017).

Hypothesis testing

To test H1, H2, and H3, two parallel mediation analyses were conducted in SPSS using the PROCESS macro (Hayes, 2012), with insomnia (ISI) as the independent variable, depression (HADS-D) as the dependent variable, and PSA and DBAS as mediators in separate models. Each analysis was structured to evaluate (a) the total effect of insomnia on depression, (b) the path from insomnia to the mediator, (c) the path from the mediator to depression after accounting for insomnia, (d) the residual direct effect of insomnia on depression after including the mediator, and (e) the indirect (mediated) effect with 95% bootstrapped confidence intervals based on 5,000 resamples. Full statistics for both models are reported in Table 4 and visualised in Figure 2.

Hypotheses 1 and 2: Pre-Sleep Arousal as a Mediator

H1 stated that insomnia would have a positive effect on depression, and H2 stated that pre-sleep arousal (PSA) would mediate this relationship. To test these hypotheses, the first mediation model was estimated with Huber–White (heteroscedasticity-consistent) standard errors, given that the homoscedasticity assumption was violated for PSAS. First, insomnia significantly predicted PSA, indicating that higher insomnia severity was associated with higher pre-sleep arousal, $R^2 = .25$, $F(1, 360) = 128.15$, $p < .001$. Second, when both insomnia and PSA were entered as predictors of depression, the overall model was significant, $R^2 = .46$, $F(2, 359) = 145.15$, $p < .001$. Within this model, insomnia remained a significant predictor of depression, $B = .11$, $t(359) = 1.98$, $p = .048$, indicating a significant direct effect after accounting for PSA. The indirect effect of insomnia on depression through PSA was significant, indirect effect = .38, 95% bootstrapped CI [.29, .47]. Because both the direct effect and the indirect effect were significant, PSA was identified as a partial — rather than full — mediator: a meaningful portion of the association between insomnia and depression is statistically accounted for by elevated pre-sleep arousal, but a residual direct association remains that PSA does not capture. Therefore, H1 and H2 were both supported.

Hypothesis 3: Dysfunctional Beliefs and Attitudes about Sleep as a Mediator

H3 stated that DBAS would mediate the relationship between insomnia and depression. The second mediation model was estimated with conventional (non-robust) standard errors, since the homoscedasticity assumption was met for DBAS. First, insomnia significantly predicted DBAS, $R^2 = .35$, $F(1, 360) = 195.69$, $p < .001$. Second,

when both insomnia and DBAS were entered as predictors of depression, the overall model was significant, R^2 = .48, $F(2, 359) = 53.73$, $p < .001$. Within this model, insomnia remained a significant predictor of depression, B = .26, $t(359) = 3.81$, $p < .001$, indicating a significant direct effect after accounting for DBAS. The indirect effect of insomnia on depression through DBAS was also significant, indirect effect = .22, 95% bootstrapped CI [.13, .32]. As with PSA, the simultaneous significance of the direct effect and the indirect effect indicates partial mediation: dysfunctional sleep-related beliefs explain part, but not all, of the association between insomnia and depression. H3 was therefore supported.

Table 4

Hierarchical regression outputs for the mediation analysis

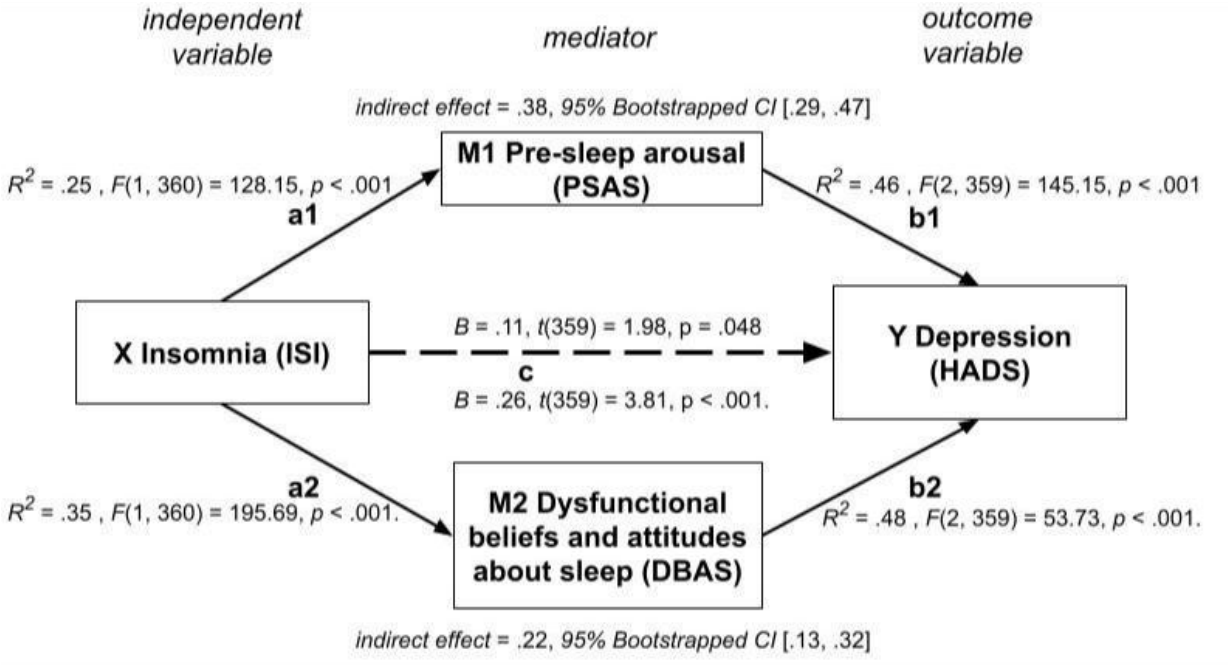
	PSAS	**Depression** (total effect)	**Depression**	**DBAS**	**Depression** (total effect)	**Depression**
	B^h	B^h	B^h	B	B	B
Constant	23.07	19.75	11.24	1.97	19.75	17.25
ISI	1.03***	.49***	.11*	.18***	.49***	.26***
PSA			.37***			
DBA						1.27***
Indirect effect 95% bootstrap		.38 [.29, .47]			.22 [.13, .32]	
N	362	362	362	362	362	362
Total R^2	.25***	.17***	.46***	.35***	.17***	.23***
F	128.15	71.43	145.15	195.69	71.40	53.73

Note: *p<.05, **p<.01, ***p <.001 normal standard errors are used in the analyses except where there is a ^{h} Huber–White (heteroscedasticity consistent) standard errors are used

Figure 2 visualises both mediation models side by side. The figure shows that insomnia is positively associated with each mediator (paths a_1 and a_2), each mediator is positively associated with depression after controlling for insomnia (paths b_1 and b_2), and a smaller but still significant direct path from insomnia to depression remains in both models (path c′). Together, the two figures and Table 4 indicate that PSA and DBAS each carry a meaningful share of the insomnia–depression association, with PSA accounting for a somewhat larger indirect effect than DBAS in this sample.

Figure 2

Results of the Two Mediation Analyses



Discussion

This study examined the association between insomnia and depressive symptoms in a Dutch adult sample, and tested whether pre-sleep arousal (PSA) and dysfunctional beliefs and attitudes about sleep (DBAS) statistically account for that association. Across two parallel mediation models, all three preregistered hypotheses were supported: insomnia severity was positively associated with depressive symptoms (H1), and both PSA (H2) and DBAS (H3) partially mediated this association. The two mediators carried meaningful but non-overlapping shares of the relationship, with the indirect effect through PSA (.38, 95% CI [.29, .47]) being somewhat larger than that through DBAS (.22, 95% CI [.13, .32]). In both models a residual direct effect of insomnia on depression remained, indicating that PSA and DBAS each explain part — but not all — of the link between disturbed sleep and depressive symptomatology.

Insomnia and Depressive Symptoms (H1)

Building on prior work showing that chronic insomnia is a robust predictor of depressive symptomatology (Neckelmann et al., 2007; Bei et al., 2018; Meaklim et al., 2023), it was predicted that ISI scores would be positively associated with HADS-D scores. The data were consistent with this prediction: insomnia severity was significantly associated with depressive symptoms in both mediation models, with a small-to-moderate residual direct effect remaining after the inclusion of each cognitive mediator. Substantively, this replicates the well-documented co-occurrence of these conditions in non-clinical adult samples and supports the view that insomnia is not merely a downstream symptom of depression but a clinically meaningful correlate that deserves attention in its own right (Blom et al., 2015).

Pre-Sleep Arousal as a Mediator (H2)

Drawing on cognitive–behavioural models of insomnia (Nicassio et al., 1985; Palagini et al., 2017) and on findings linking pre-sleep arousal to depressive and suicidal symptoms (Karlson et al., 2010; Olgiati & Serretti, 2022; Meaklim et al., 2023), it was predicted that PSA would mediate the insomnia–depression association. The findings were consistent with this prediction: insomnia severity was strongly associated with PSA, PSA was associated with HADS-D scores after adjusting for insomnia, and the bootstrapped indirect effect was significant. The pattern is best characterised as partial rather than full mediation, meaning that elevated nocturnal cognitive and somatic arousal statistically accounts for a sizeable portion of why people with worse insomnia also report more depressive symptoms, but does not fully explain the relationship. This is consistent with theoretical accounts in which hyperarousal is one — but not the only — transdiagnostic process linking sleep disturbance to mood (Kalmbach et al., 2023).

Dysfunctional Beliefs and Attitudes about Sleep as a Mediator (H3)

Given prior evidence that dysfunctional sleep-related cognitions sustain insomnia (Morin et al., 1993, 2007) and predict depressive symptomatology (Park et al., 2012; Meaklim et al., 2023), and that change in DBAS mediates change in psychological distress during cognitive behavioural therapy for insomnia (Parsons et al., 2021; Faaland et al., 2023), it was predicted that DBAS would mediate the insomnia–depression association. This prediction was also supported, with a significant indirect effect and a smaller, but still significant, residual direct effect. The smaller indirect effect for DBAS relative to PSA suggests that, in this sample, beliefs about sleep account for somewhat less of the insomnia–depression association than nocturnal arousal does. One possible reading is that PSA reflects an immediate state-level mechanism (in-the-moment hyperarousal at bedtime), whereas DBAS reflects a more stable trait-like cognitive pattern; both contribute, but they may operate on different time scales. Importantly, both mediators are explicit targets of CBT-I, which strengthens the clinical relevance of the present findings.

Strengths

Beyond replicating the insomnia–depression association, the present study has several strengths. First, by testing PSA and DBAS as parallel mediators in the same sample, it allows a direct comparison of the relative contribution of each mechanism, rather than evaluating them in isolation as much of the prior literature has done. Second, the sample combined a self-selected insomnia-prone group with a more general convenience sample, which broadened the range of insomnia severity captured by the ISI and increased the statistical power available to detect indirect effects. Third, the use of bootstrapped confidence intervals via the PROCESS macro provides a more robust test of mediation than older causal-steps approaches and reduces reliance on assumptions of normally distributed indirect effects. Together, these features add credibility to the conclusion that PSA and DBAS each carry a meaningful share of the insomnia–depression relationship.

Limitations and Future Research

Several limitations should be noted. First, the PSAS and HADS were analysed only at the total-score level, even though both have meaningful subscales (somatic vs. cognitive PSA; HADS-A vs. HADS-D). This precludes any conclusion about which subcomponent drives the observed indirect effects. Distinguishing PSAS-somatic

from PSAS-cognitive matters because cognitive arousal in particular has been singled out as the active ingredient in mindfulness-based and CBT-I interventions for insomnia and depression (Kalmbach et al., 2023; Olgiati & Serretti, 2022), and may therefore be the more clinically actionable target. Likewise, examining HADS-A alongside HADS-D would have been informative because insomnia is a transdiagnostic risk factor for both anxiety and depression, and the two are highly comorbid in clinical samples (Bjelland et al., 2002; Meaklim et al., 2023); the present design cannot say whether PSA and DBAS preferentially mediate associations with depressive symptoms specifically or with general affective distress. Future work should re-run the present mediation models at the subscale level to clarify these distinctions.

Second, the sample limits generalisability. Participants in the website group actively volunteered because they recognised sleep complaints in themselves, while the convenience-recruited group is unlikely to be representative of the general population. As a result, it is unclear whether the observed indirect effects would replicate either in a strictly clinical sample of patients meeting diagnostic criteria for insomnia disorder and major depression, or in a fully unselected community sample with mostly subclinical symptoms. Future studies should test the present model in both directions, ideally using diagnostic interviews rather than self-report cut-offs.

Third, the study relied entirely on self-report measures collected at a single time point in a fixed (non-randomised) order, which introduces several related limitations: shared method variance can inflate associations between constructs measured with the same response format; participants' answers may be coloured by mood on the day of testing; and a fixed item order leaves open the possibility of order effects on later questionnaires. Future research should add objective indicators — for example, polysomnography or actigraphy to quantify sleep, and EEG-based markers of nocturnal arousal — alongside self-report, randomise the order of the questionnaires within the survey platform, and ideally collect repeated measurements over time to assess within-person stability of the mediating processes.

Finally, the design is cross-sectional and therefore correlational. Although the analytic framework is one of mediation, the data cannot establish that insomnia causes depression via PSA or DBAS, nor that intervening on PSA or DBAS would weaken the insomnia–depression link in a clinical setting. Longitudinal designs that track change in insomnia, the candidate mediators, and depressive symptoms across multiple waves — and ideally randomised intervention studies that experimentally manipulate PSA or DBAS — are needed to evaluate the directional and causal claims that the present mediation models can only suggest.

Clinical Implications and Conclusion

With these caveats in mind, the results support the clinical relevance of explicitly addressing pre-sleep arousal and dysfunctional sleep-related beliefs when treating patients who present with comorbid insomnia and depression. Both mediators are already targeted by CBT-I (Parsons et al., 2021; Faaland et al., 2023), and the present findings strengthen the rationale for integrating these components into combined treatments rather than treating insomnia and depression as separate problems. The direct comparison of PSA and DBAS as parallel mediators, the broad range of insomnia severity in the sample, and the bootstrapped estimation of indirect effects together place these conclusions on firmer footing than studies that examine either mediator in isolation. Continued investment in longitudinal and intervention research is needed to confirm the directionality of these relationships and to identify which patients benefit most from arousal-focused versus cognition-focused components of treatment.

Conflict of Interests

There is no conflict of interests.

Acknowledgements

The author thanks the participants who took part in the study, the staff of www.insomnie.nl for their assistance with recruitment, and the reviewers who provided valuable feedback on earlier drafts of this manuscript.

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