SHORT REPORT

Mind, Body and Machine: Preliminary Study to Explore Predictors of Treatment Response After a Sleep Robot Intervention for Adults with Insomnia

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Introduction: The study aimed to explore characteristics of responders to a sleep robot intervention for adults with insomnia, and the likelihood that participants responded to the intervention.

Methods: Data from the intervention and the control group in a randomized waitlist-controlled trial (n = 44) were pooled together after both had undergone the intervention. A repeated measures ANOVA and Friedman tests were used to explore changes over time. Differences in baseline characteristics between responders (n = 13), defined as a reduction of −5 on the Insomnia Severity Index from pre- to post-intervention, and non-responders (n = 31) were analyzed with *t*-tests and chi-square tests. Finally, logistic regression models were estimated.

Results: Baseline anxiety was the only statistically significant difference between responders and non-responders (*p* = 0.03). A logistic regression model with anxiety and sleep quality as predictors was statistically significant, correctly classifying 83.3% of cases.

Discussion: The results imply that people with lower anxiety and higher sleep quality at baseline are more likely to report clinically significant improvements in insomnia from the sleep robot intervention.

Keywords: anxiety, depression, insomnia, sleep, sleep diary, sleep robot, treatment response

Introduction

Insomnia, or sleeplessness, is the most common sleep disorder in adults,[1] and the second most common mental disorder of all.[2] The definition of insomnia in the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) involves a self-experienced difficulty with falling asleep or maintaining sleep throughout the night and/or in the early morning, causing significant distress or impairment in, for instance, social or occupational functioning.[3] The first-line treatment of insomnia is Cognitive-Behavioral Therapy (CBT-I).[4] At least partly due to a shortage of CBT-therapists,[5] the most common insomnia treatment is still hypnotic medicine.[6] Research on potential treatment options is warranted. Moreover, average treatment effects in studies may mask unrecognized insomnia subtypes, and further, affect successful replication and synthetization of results from individual studies in meta-analyses.[7] Hence, it is important to consider the significance of participant characteristics for the effects, or lack thereof, in intervention studies, for more personalized insomnia treatments.

Subtypes of insomnia have been proposed, for instance, initial, middle, late, and mixed insomnia, based on people's most salient sleep complaint.[7] Moreover, it has been suggested that non-sleep characteristics may constitute more important subtypes, for instance demographics, mood, affect, personality, and life history.[7,8] According to the diagnostic criteria, insomnia does not necessarily entail short sleep duration. Insomnia combined with objectively defined short sleep duration has been found to be a more severe type of insomnia, compared with that of insomnia and adequate sleep duration.[9] People with insomnia are often diagnosed with other psychiatric disorders as well, for example anxiety and depression.[10,11] Given the nature of the diagnostic criteria, the number of people who meet the criteria, combined with objectively defined short sleep duration versus adequate sleep hours, and numerous possible comorbid conditions, people with insomnia should be viewed as a heterogeneous group of people, likely with different treatment needs (eg, full CBT-I program versus certain components, or evidence-based treatment options). Regarding clinical heterogeneity, or differences in participant characteristics, sex (female), age (older), education (low), and marital status (separated/divorced or widowed) are established predictors of insomnia.[12–14] However, these factors have not been shown to be significant predictors of treatment success when it comes to CBT-I.[15–17] Higher levels of anxiety symptoms have, on the other hand, been found to predict poorer psychological treatment response in adults with insomnia and chronic hypnotic use.[18] Insomnia is by definition more of a sleep quality than a sleep quantity problem. Sleep quality has been found to be an important outcome when it comes to defining treatment response.[19,20] It is therefore important to address sleep quality in insomnia intervention studies in general – and specifically when analyzing treatment response.

Consumer sleep technology has been defined as "Non-prescription devices directly marketed to consumers that may make an assertion to perform sleep monitoring, tracking, or sleep-related interventions" by the American Academy of Sleep Medicine.[21] Most sleep devices, and research on such devices, concern monitoring and tracking. One line of sleep-related interventions are sleep robots. In a systematic review and network meta-analysis of robots' effects on nighttime sleep, no statistically significant differences were found between robot interventions, plush toys, or treatment as usual on nighttime sleep in older adults.[22] The robot used in all four included studies was a companion robot, ie, not a robot first and foremost meant to be able to treat troubled sleep. An example of a sleep intervention device is the Somnox sleep robot, a sort of pillow with integrated Intelligent Smart Breathing Technology.[23] The robot is meant to be held close to the body at bedtime with the aim of synchronized optimal breathing in order to foster relaxation and improve sleep. Relaxation is widely recognized to have positive impacts on both physical health and mental well-being.[24,25] In the context of CBT-I, relaxation can take various forms, including progressive muscle relaxation, guided imagery, and breathing techniques.[26,27] Hyperarousal is a common feature in the majority of insomnia models, encompassing heightened levels of somatic arousal such as increased heart rate and respiration, cognitive arousal with excessive worry and rumination, and cortical arousal in terms of increased brain activity. The hyperarousal or the neurocognitive model of insomnia – an extension of the 3P behavioral model where the three Ps stand for predisposing, precipitation and perpetuating factors – highlights the importance of all three forms of hyperarousal in insomnia.[28,29] It is commonly believed that relaxation can be helpful in reducing somatic arousal and enhance sleep. However, it may also work by calming pre-sleep cognitive activity, ie, by simultaneously reducing worries and increasing positive thoughts about one's sleep.[30]

The sleep robot intervention has previously been described in the original randomized waitlist-controlled trial and in a feasibility study.[31,32] The aforementioned studies are the only independent studies on the Somnox sleep robot. The conclusion from the feasibility study was that there were mixed results regarding individual effects of the sleep robot intervention, but that the intervention was found both acceptable in terms of adherence, and safe in terms of emotional distress.[32] The randomized waitlist-controlled trial focused on somatic arousal as this factor was believed to be the sleep robot's target, to reduce physiological arousal through breathworks. Group means did not show statistically significant effects of the intervention on any of the outcome measures (insomnia severity, somatic arousal, symptoms of anxiety and depression, sleep variables measured subjectively and objectively). However, looking at the raw scores on an individual level, several participants reported clinically relevant reductions regarding insomnia severity post-intervention that are important to explore. The aims of the current study were to assess the proportions of responders and non-responders in the sleep robot intervention, find out if responders and non-responders differed from each other in terms of baseline demographics and clinical characteristics, and examine the likelihood that participants responded to the intervention based on identified characteristics. The research questions were: (1) How do responders and non-responders differ from each other at baseline; and (2) What is the likelihood that participants are responders? The study is the first to explore possible predictors of treatment response after an intervention with the Somnox sleep robot.

Methods

Study Design

In the original study,[31] a pre-registered randomized waitlist-controlled trial (n = 44) was conducted to evaluate the effects of an at-home intervention with a sleep robot (Somnox) for three weeks (n = 22) compared with the waitlist control group (n = 22). The study complied with the Declaration of Helsinki. The Swedish Ethical Review Board approved the study project (DNR 2020–069759), which was conducted in Karlstad, Sweden in 2021. Informed consent was obtained from participants prior to the start of the study. The trial was registered in the ISRCTN registry (ISRCTN35134834). In the current study, data were pooled from both the intervention and the waitlist control group when the control group had also received the robot intervention. Differences between responders and non-responders regarding baseline demographics and clinical characteristics were analyzed, as these variables constitute potential predictors of treatment response.

Participants and Procedures

Participants were screened for eligibility in two stages: firstly with the Insomnia Severity Index (ISI),[33] and the Pre-Sleep Arousal Scale (PSAS).[34] In the next stage, we administered the Duke Structured Interview for Sleeping Disorders (DSISD),[35] and the Mini International Neuropsychiatric Interview (M.I.N.I.).[36] The screening was conducted by phone by first author SJS. Inclusion criteria were that participants had to speak Swedish fluently, be over 18 years of age, and meet the DSM-5 diagnostic criteria of insomnia. Exclusion criteria were to meet the diagnostic criteria of another current psychiatric disorder, or untreated sleep disorder (hypersomnia, narcolepsy, sleep apnea, circadian rhythm sleep disorder, parasomnia, restless legs). Of the 54 people that were screened, 44 fulfilled the requirements.[31]

Intervention and Control Conditions

The intervention consisted of at-home usage of the Somnox sleep robot for three consecutive weeks. The robot had different settings but was set on the 30-minute program called "sleeping" for all participants, with the possibility to speed up or slow down the breathing manually to some degree. Meanwhile, the control group remained on a waitlist – that is, no intervention during the experimental stage of the study.[31] The control group received the intervention after the waiting period, and completed an additional post-intervention measurement, enabling the pooling of data from both groups to assess, with a larger sample size, predictors of treatment response. See Figure 1 for the study's flow chart.

Measures

Information about participants was collected in terms of gender identity, marital status, children, level of education, employment, and country of birth (in or outside Sweden). Effects of the intervention were assessed in multiple ways. Questionnaires were administered pre-intervention, mid-intervention (main outcome measure only), post-intervention, and at 1-month follow-up. Sleep variables from the Consensus Sleep Diary and wrist actigraphy were assessed the week before the intervention and the last intervention week (weekly means).[37,38] Questionnaires and sleep diaries were completed on the Iterapi online platform.[39] Additionally, participants completed three daily questions on treatment adherence throughout the intervention.

The primary outcome measure was the Insomnia Severity Index (ISI: 7 items, score range 0–28, cut-off 11). The criteria for a positive treatment response was −5 points on the ISI, as −4.7 is widely considered a slight clinical improvement.[40] Arousal was considered the probable mechanism in case of a beneficial treatment response. Arousal was measured with the Pre-Sleep Arousal Scale (PSAS), which consists of two scales (cognitive and somatic), of which only the somatic scale was used (8 items, score range 8–40). Higher scores indicate hyperarousal. Ten was used as a cut-off of hyperarousal, in line with previous studies.[41] Symptoms of anxiety and depression (emotional distress) were measured with the Hospital Anxiety and Depression Scale (HADS),[42] which consists of 14 items (7 items in each, score range 0–21). Higher scores indicate clinical levels of anxiety and depression (cut-off 8). For the anxiety scale, a change of 1.3 represents a slight improvement, whereas a change of 1.4 represents a slight improvement for the depression scale.[43]

Sleep onset latency (SOL), wake after sleep onset (WASO), total sleep time (TST), and sleep efficiency (SE) were measured both subjectively with the Consensus Diary, and objectively with wrist actigraphy. The sleep diary included
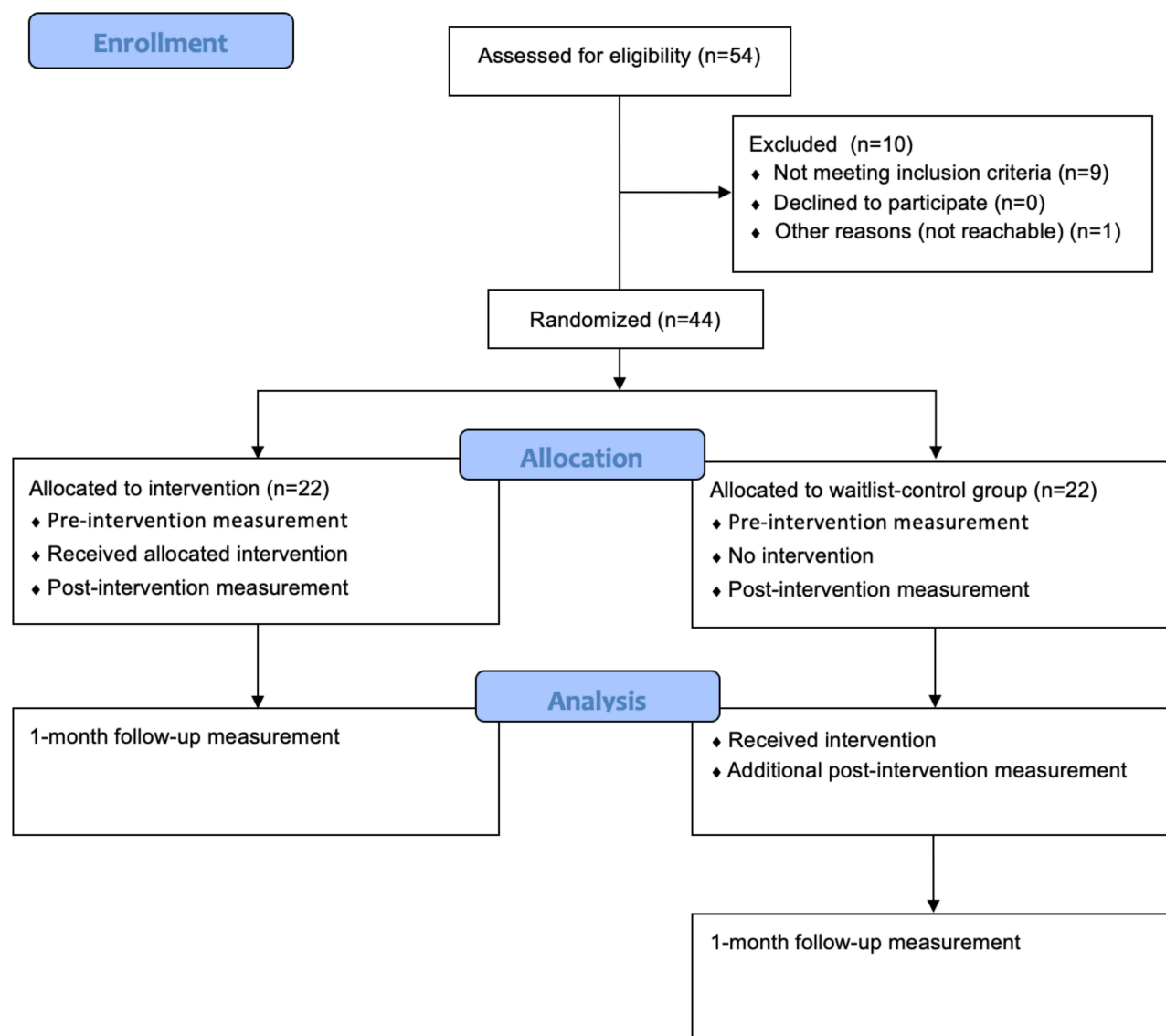
Figure 1 Study's flow chart.

several other variables that were not analyzed in the original study, for instance sleep quality (SQ). The sleep quality question was framed as *How did you sleep?* With five possible answers: (1) very poorly, (2) pretty poorly, (3) neither good nor bad, (4) pretty good, and (5) very good. The current study focused on diary-defined SOL, WASO, and TST (not SE as its calculation is based on TST), in addition to SQ.

Statistical Approach

We used the pooled data from both the intervention group and the control group when the latter group had also received the intervention (with an additional post-intervention assessment consisting of the ISI, the PSAS and the HADS), that is, after the randomized phase of the study. This enabled analyses of the 1-month follow-up data, in line with the published study protocol.[44] The power analysis, described in the study protocol, was calculated on the main outcome measure only, why the results in the current study should be interpreted with caution.

The assumptions of normality and sphericity were met for the Insomnia Severity Index (ISI), but not for the Pre-Sleep Arousal Scale (PSAS) and the Hospital Anxiety and Depression Scale (HADS). A one-way repeated measures analysis of variance (ANOVA) was conducted on the ISI, and Friedman tests on the PSAS and the HADS, to assess the short- and long-term time effects of the sleep robot intervention. Regarding missing data, cases were excluded listwise (n = 7, 15.9%).

After defining responders as those with a −5-point reduction on the ISI from pre- to post-intervention, *t*-tests and chi-square tests were conducted to compare the baseline demographics and clinical characteristics of responders versus non-responders. This was done in order to form hypotheses about possible subgroups that might benefit from the sleep robot intervention. Predictors of treatment response were investigated with logistic regression models, with a categorical responder/non-responder outcome variable. The following predictor variables were initially added as predictors in Model A: symptoms of insomnia, arousal, anxiety and depression, measured with the Insomnia Severity Index, the Pre-Sleep Arousal Scale, and the Hospital Anxiety and Depression Scale, respectively; diary defined sleep onset latency, wake after sleep onset, total sleep time, and sleep quality. These were selected based on previous findings in the literature.[45] In Model B, we only added anxiety and sleep quality as predictors. This second part of the study was not described in the study protocol, meaning that these are exploratory analyses.

Results
Insomnia Severity
For the Insomnia Severity Index, Shapiro–Wilk tests were not statistically significant for pre-intervention ($p = 0.26$), post-intervention ($p = 0.19$), or 1-month follow-up ($p = 0.47$), suggesting that the assumption of normality was not violated. Thus, a one-way repeated measures ANOVA was conducted, which resulted in a statistically significant beneficial time effect, $F (2, 35) = 7.22$, $p = 0.00$, $\eta2 = 0.29$. The Least Significance Difference post hoc test (default in SPSS) revealed statistically significant differences between pre- and post-intervention ($p = 0.01$) and between pre-intervention and 1-month follow-up ($p < 0.001$), but not between post-intervention and 1-month follow-up ($p = 0.20$). See Table 1 for means and standard deviations for each time point.

Somatic Arousal
For the Pre-Sleep Arousal Scale, Shapiro–Wilk tests were statistically significant for pre-intervention ($p = 0.01$) and post-intervention ($p = 0.00$), but not for the 1-month follow-up ($p = 0.38$), indicating that the assumption of normality was violated on the first two time points. Therefore, the Friedman test was conducted, which rendered a statistically

Table 1 Means and Standard Deviations for Each Outcome Measure at Each Time Point, and Pairwise Comparisons of Time Points

Outcome	Pre	Post	Follow-Up	Mean Diff / Z	*p*
	Mean (SD)	**Mean (SD)**	**Mean (SD)**		
ISI	16.73 (3.82)	14.92 (5.89)	14.03 (5.78)		
Pre-Post				1.81	0.01*
Pre-Follow-up				2.70	0.00*
Post-Follow-up				0.89	0.20
PSAS	16.49 (5.66)	15.11 (5.64)	15.24 (5.87)		
Pre-Post				−2.77	0.00*
Pre-Follow-up				−1.52	0.13
Post-Follow-up				−0.71	0.48
HADS-anxiety	7.22 (4.10)	6.76 (3.93)	7.32 (4.23)		
Pre-Post				−1.97	0.05
Pre-Follow-up				−0.19	0.85
Post-Follow-up				−1.55	0.12
HADS-depression	4.89 (3.24)	4.84 (3.08)	4.46 (3.17)		
Pre-Post				−0.40	0.69
Pre-Follow-up				−1.14	0.26
Post-Follow-up				−1.29	0.20

Note: *$p < 0.05$.
Abbreviations: ISI, The Insomnia Severity Index; PSAS, The Pre-Sleep Arousal Scale; HADS-a/d, The Hospital Anxiety and Depression Scale – anxiety/depression scale; SD, Standard Deviation; Mean Diff, Mean Difference.

significant beneficial time effect, χ^2 (2, N = 37) = 11.82, p = 0.00. The Wilcoxon Signed Ranks post-hoc test (default in SPSS) showed a statistically significant difference between pre- and post-intervention (p = 0.01), but no statistically significant differences between pre-intervention and 1-month follow-up (p = 0.13) or between post-intervention and 1-month follow up (p = 0.48).

Emotional Distress

Regarding the anxiety part of the Hospital Anxiety and Depression Scale, Shapiro–Wilk tests were not statistically significant for pre-intervention (p = 0.07) or 1-month follow-up (p = 0.25) but was statistically significant for post-intervention (p = 0.02). The Friedman test was not statistically significant, χ^2 (2, N = 37) = 4.27, p = 0.12, that is, there were no statistically significant short- or long-term time effects regarding symptoms of anxiety. Regarding symptoms of depression, Shapiro–Wilk tests suggested that the depression part of the Hospital Anxiety and Depression Scale was normally distributed at pre-intervention (p = 0.07), but not normally distributed post-intervention (p = 0.02) nor at the follow-up (p = 0.02), which is why the Friedman test was conducted here as well, with a non-statistically significant result, χ^2 (2, N = 37) = 3.77, p = 0.15.

Baseline Characteristics

Responders were defined as those with a −5 change on the ISI from pre- to post-intervention. Of the 44 participants, 13 met this criterion and 31 did not. Baseline characteristics of responders versus non-responders are depicted in Table 2. The only statistically significant difference between responders and non-responders on the 0.05 level was baseline *anxiety* symptoms according to the Hospital Anxiety and Depression Scale, t(42) = −2.21, p = 0.03. Sleep quality was marginally significant on the less stringent 0.10 (p = 0.09).

Table 2 Baseline Demographic and Clinical Characteristics of Responders (n = 13) versus Non-Responders (n = 31). Statistical Significance Tested with *t*-Tests and Chi-Square Tests

Variables	Responders (n=13)	Non-Responders (n=31)	t / χ^2	p
	Mean (SD) or % (n)	Mean (SD) or % (n)		
Age, in years	48.46 (17.39)	48.77 (12.02)	0.07	0.95
Gender			2.38	0.24
Female	92.31% (12)	71.00% (22)		
Male	7.69% (1)	29.00% (9)		
Marital status			3.66	0.30
Single	30.77% (4)	22.58% (7)		
Partner	0.00% (0)	3.23% (1)		
Married/cohabiting	46.15% (6)	67.74% (21)		
Divorced/separated	23.08% (3)	6.45% (2)		
Children in household			2.73	0.11
% Yes	30.77% (4)	58.10% (18)		
% No	69.23% (9)	41.90% (13)		
Born in Sweden			0.02	1.0
% Yes	92.31% (12)	93.50% (29)		
% No	7.69% (1)	6.50% (2)		
Level of education			3.03	0.39
Middle school	7.69% (1)	3.23% (1)		
High school	38.46% (5)	25.81% (8)		
Vocational	0.00% (0)	9.68% (3)		
University/college	53.85% (7)	61.29% (19)		

(*Continued*)

Table 2 (Continued).

Variables	Responders (n=13)	Non-Responders (n=31)	t / χ²	p
	Mean (SD) or % (n)	Mean (SD) or % (n)		
Employment			5.40	0.37
Permanent	53.85% (7)	70.96% (22)		
Temporary	7.69% (1)	9.68% (3)		
Self-employed	0.00% (0)	0.00% (0)		
Student	15.38% (2)	3.23% (1)		
Retired	15.38% (2)	12.90% (4)		
Sickness Compensation	0.00% (0)	3.23% (1)		
Other	7.69% (1)	0.00% (0)		
Insomnia symptoms, in years	11.38 (10.57)	13.50 (12.63)	0.53	0.60
Sleep medications			1.32	0.46
% Yes	15.38% (2)	32.30% (10)		
% No	84.62% (11)	67.70% (21)		
ISI	16.62 (4.07)	16.45 (3.78)	−0.13	0.90
PSAS	15.08 (4.23)	16.45 (6.17)	0.73	0.47
HADS - Anxiety	5.15 (3.51)	7.94 (3.93)	2.21	0.03*
HADS - Depression	3.62 (3.40)	5.19 (3.04)	1.51	0.14
SOL-d, by sleep diary, min	41.69 (28.77)	49.54 (56.75)	0.47	0.64
WASO-d, by sleep diary, min	53.33 (29.87)	47.57 (34.82)	−0.50	0.62
TST-d, by sleep diary, min	359.85 (55.10)	370.25 (59.88)	0.53	0.60
SE-d, by sleep diary, %	72.63 (10.94)	71.41 (9.14)	−0.38	0.71
SQ-d, by sleep diary	3.17 (0.58)	2.86 (0.49)	−1.75	0.09
SOL-A, by actigraphy, min	4.70 (15.72)	0.34 (0.80)	−1.57	0.13
WASO-A, by actigraphy, min	38.95 (9.64)	43.51 (18.33)	0.85	0.40
TST-A, by actigraphy, min	309.82 (66.92)	324.70 (71.96)	0.64	0.53
SE-A, by actigraphy, %	88.77 (2.07)	88.07 (4.23)	−0.57	0.57
Adherence, days	14.77 (4.69)	15.62 (5.41)	0.49	0.63
Adherence, min	41.53 (11.82)	57.32 (70.73)	0.73	0.47

Note: *$p > 0.05$.
Abbreviations: ISI, The Insomnia Severity Index; PSAS, The Pre-Sleep Arousal Scale; HADS-A/d, The Hospital Anxiety and Depression Scale – anxiety/depression scale; SOL-d/a, Sleep Onset Latency – diary/actigraphy; WASO-d/a, Wake After Sleep Onset – diary/actigraphy; TST-d/a, Total Sleep Time – diary/actigraphy; SE-d/a, Sleep Efficiency diary/actigraphy; SQ-d, Sleep Quality.

Likelihood of Treatment Response

To assess the likelihood that participants would respond to the intervention, binary logistic regression analyses were performed, firstly with the following baseline variables as predictors: the ISI, the PSAS, the HADS anxiety scale, the HADS depression scale, in addition to diary defined SOL, WASO, TST, and SQ. The result was statistically significant, χ^2 (8, N = 39) = 24.28, $p = 0.00$, R^2_N = 0.65, indicating that the model was able to distinguish between responders and non-responders. Moreover, the overall percentage of correctly classified cases was 89.7%. Only HADS-anxiety ($p = 0.03$) and sleep quality ($p = 0.02$) made unique statistically significant contributions to the model, which is why a new logistic regression analysis was conducted with these two variables as the only predictors, see Table 3. The second model was statistically significant, χ^2 (2, N = 42) = 10.21, $p = 0.01$, R^2_N = 0.30, as were the unique contributions of anxiety ($p = 0.02$) and sleep quality ($p = 0.04$). The latter model correctly classified 83.3% of cases.

Adherence

Responders did not use the sleep robot for more days than non-responders did; 14.77 days (SD 4.69) for responders compared with 15.62 days (SD 5.41) for non-responders, t (40) = −0.49, $p = 0.63$. Responders used the robot for an

Table 3 Binary Logistic Regression Models Predicting Likelihood of Treatment Response, Defined as a −5 reduction on the Insomnia Severity Index from Pre- to Post-Intervention

Predictors	Model A: OR (95% CI)	Model B: OR (95% CI)
ISI	1.51 (0.96–2.38)	
PSAS	1.09 (0.84–1.42)	
HADS-anxiety	0.48 (0.26–0.92)*	0.75 (0.58–0.96)*
HADS-depression	0.61 (0.37–1.02)	
SOL	1.00 (0.98–1.03)	
WASO	1.02 (0.99–1.06)	
TST	0.98 (0.95–1.01)	
SQ	480.83 (3.55–65,191.79)*	4.88 (1.06–22.59)*

Note: *$p > 0.05$.
Abbreviations: ISI, The Insomnia Severity Index; PSAS, The Pre-Sleep Arousal Scale; HADS-a, The Hospital Anxiety and Depression Scale – anxiety; HADS-d, The Hospital Anxiety and Depression Scale – depression; WASO, Wake After Sleep Onset; TST, Total Sleep Time; SQ, Sleep Quality; OR, Odds Ratio; CI, Confidence Interval.

average of 41.53 minutes (SD 11.82), whereas non-responders used the robot for an average of 57.32 minutes (SD 70.73) each night, $t(38) = 0.47$, $p = 0.47$.

Discussion

The study demonstrated a statistically significant time effect on the Insomnia Severity Index, for all three measurement points (pre-intervention, post-intervention, and 1-month follow-up, albeit no difference between the latter two), indicating that the sleep robot might lead to persistent changes regarding level of insomnia symptoms weeks after a person stops using it. It should be noted that the average effect was small and that post-treatment insomnia severity did not end up under the cut-off threshold for insomnia. For the Pre-Sleep Arousal Scale, the time effect was only statistically significant in the short term from pre- to post-intervention, suggesting that the sleep robot only affects physiological arousal while it is actively used. Finally, the time effects on anxiety and depression symptoms as measured with the Hospital Anxiety and Depression Scale were not statistically significant.

Both anxiety and sleep quality predicted the likelihood that participants would respond to the sleep robot intervention for insomnia. Responders (n = 13) had lower baseline anxiety compared with non-responders (n = 31) (ie, non-responders had higher baseline anxiety), and the difference was statistically significant. This is in line with previous research showing that people with higher levels of anxiety respond less well to insomnia treatments,[18] even when the symptoms do not meet the diagnostic criteria of an anxiety disorder. As Tramonti et al (p. 309) point out,[46]

> anxiety and depressive symptoms are still interpreted in terms of comorbidity, and the criteria for identifying insomnia and psychopathological conditions still rely on threshold measures, whose limitations and risks of neglecting the clinical relevance of subthreshold presentations have been underscored.

The higher level of anxiety symptoms in non-responders may also be more associated with cognitive arousal, unfortunately not captured in the current study as we only included the somatic scale from the Pre-Sleep Arousal Scale. Perhaps those with elevated levels of anxiety would have benefited more from a cognitive-behavioral intervention rather than a sleep robot whose main target can be said to be somatic arousal.

Responders also had higher baseline sleep quality than non-responders (who had lower baseline sleep quality), albeit not a statistically significant difference on the 0.05 level. Sleep quality is not easily defined. In the current study, a single-item rating scale was used from the sleep diary. In contrast, the widely used and well-validated Pittsburgh Sleep Quality Index (PSQI) contains 19 items (including questions about sleep onset latency and sleep duration).[47] The scale of the single item was 1–5, where 3 in the middle represents neither good nor bad. Qualitative research has found that people both with and without insomnia use several criteria when they judge their own sleep quality, and that people with

insomnia have more criteria compared with people without insomnia,[48] which is why the single item in the current study might be flawed. Additionally, the question *How did you sleep?* is vague and open for interpretation (eg, sleep satisfaction or depth of sleep), a common problem in sleep quality studies.[48] Lastly, the results were not due to a dose-response effect of the sleep robot, as adherence data showed that responders did not use the sleep robot more than non-responders did.

Limitations

The study has limitations and the first is that we analyzed data after the experimental stage of the study, that is, when the randomization was broken. These secondary analyses were only partly described in the study protocol and are therefore partly exploratory (ie, higher risk of bias). Furthermore, we only calculated a power analysis for the main outcome measure in the original study, and no power analyses for the current study. Adding to the limitations, only 13 participants were classified as responders, meaning that only large differences between responders and non-responders turn out statistically significant (ie, risk of ignoring smaller but meaningful differences between responders and non-responders). Yet another limitation was that tests of normality showed that three out of four outcome measures for the time effects analyses were not normally distributed, which is why the less powerful non-parametric Friedman test was conducted instead of repeated measures ANOVAs on these variables (the PSAS, the HADS – anxiety scale, the HADS – depression scale). The first logistic regression model included more predictors than what was sensible with a sample of 44 participants, and the results of the second logistic regression model with anxiety and sleep quality as predictors must be interpreted with caution as well. A final limitation is that we only included somatic arousal from the Pre-Sleep Arousal Scale, and that a cognitive scale might have yielded important information considering the higher anxiety levels and lower sleep-quality ratings for non-responders.

Concluding Remarks

This study implies that people with less anxiety and higher sleep quality are more likely to benefit from the sleep robot intervention, compared to more anxious people, and/or people reporting poorer sleep quality prior to treatment. This is important information that was concealed when we only focused on group effects in the original study.[31] A strength with our study is the thorough screening of participants including a wide number of potentially relevant demographics and clinical characteristics. Commercial products are rarely investigated independently from the producers, why another strength is that the study goes in depth and offers impartial guidance regarding for whom the Somnox sleep robot might be associated with a clinically meaningful improvement of insomnia symptoms. These results must be confirmed in more studies before clinicians recommend the Somnox sleep robot as a stand-alone treatment of insomnia. In the larger picture, the study adds to the conversation about different types of insomnia and individually tailored treatments.

Data Sharing Statement

Data are available from the corresponding author upon reasonable request.

Acknowledgments

Disclosure

The authors have no conflicts of interest to declare.

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