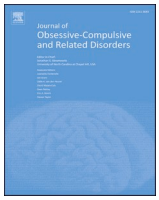
Journal of Obsessive-Compulsive and Related Disorders 42 (2024) 100891

Contents lists available at ScienceDirect

Journal of Obsessive-Compulsive and Related Disorders

journal homepage: www.elsevier.com/locate/jocrd



Effect of concentrated exposure and response prevention on symptoms of insomnia

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A B S T R A C T

Background: Insomnia symptoms are prevalent among patients with obsessive-compulsive disorder (OCD). This study involved secondary analyses from a previous randomized controlled trial testing if D-Cycloserine (DCS) augmented the effects of the Bergen 4-day treatment (B4DT) for OCD. In this study, the effects of the B4DT on sleep difficulties in a sample of difficult-to-treat OCD were explored.

Methods: The three groups received B4DT with either a placebo, 100 mg, or 250 mg of DCS. Patients ($N = 163$) had either not responded or relapsed after previous exposure-based treatment for OCD.

Results: The results showed a small, but robust treatment effect on insomnia symptoms ($d = 0.37$), maintained at 3-month follow-up ($d = 0.38$), and 12-month follow-up ($d = 0.23$). No significant differences between the groups receiving DCS or placebo were found. Insomnia did not affect OCD-treatment outcome.

Discussion: This study showed that OCD treatment is associated with some improvement in insomnia symptoms. Comorbid insomnia and DCS did not moderate treatment outcome. Patients with OCD and comorbid insomnia should be considered for specific insomnia treatment.

1. Effect of concentrated exposure and response prevention on symptoms of insomnia

Insomnia is a psychiatric diagnosis comprised of night- and day-time symptoms affecting well-being and quality of life (First et al., 2015). Night-time symptoms include early morning wakening, disturbed sleep maintenance, and delayed sleep onset. Day-time functioning is characterized by fatigue, cognitive impairment, and low mood (Riemann et al., 2022). In clinical populations (e.g., psychosis, depression, and anxiety disorders), 70–80% of patients show symptoms of insomnia in the acute phase of their mental illness (Palagini et al., 2022). As insomnia occurs across psychiatric disorders it is often regarded as a non-specific occurrence and not given sufficient clinical attention, however, it could be a causal factor and typically show a bidirectional association with other psychiatric symptoms (Freeman et al., 2020). It is important to examine insomnia in the context of other psychiatric conditions because insomnia often persists despite successful treatment and may increase the risk of relapse (Carney et al., 2011; Jansson-Fröjmark et al., 2016).

Patients with obsessive-compulsive disorder (OCD) are not exempt from comorbidity with insomnia (Cox et al., 2020). Patients with OCD show shorter sleep duration, increased awakenings, and extended sleep onset latency compared to healthy individuals (Nota et al., 2015). A Swedish population-based cohort study reported a 7-fold increase in insomnia among patients with OCD compared to the general population (Sevilla-Cermeño et al., 2020). A transdiagnostic model suggests that sleep disturbances have a negative impact on a range of neurobiological systems, which may in turn increase the development of mental illness (Harvey et al., 2011). One such effect is a decrease in cognitive control, which in turn may lead to increased obsessions (Cox et al., 2018).

Cognitive behavioral therapy (CBT) for insomnia is an evidence-based treatment (Koffel et al., 2015; Trauer et al., 2015). However, evidence for CBT on concomitant sleep disturbances is inconclusive. One review showed a medium effect size ($d = 0.53$) on sleep difficulties for treatment of anxiety disorders (Belleville et al., 2010). None of the 19 studies included in the review addressed sleep difficulties directly and insomnia outcomes were examined on a secondary basis. A more recent study showed a modest effect on global sleep quality and sleep latency

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https://doi.org/10.1016/j.jocrd.2024.100891
Received 12 March 2024; Received in revised form 13 May 2024; Accepted 13 June 2024
Available online 15 June 2024

following CBT for patients with generalized anxiety disorder and panic disorder, but no effect on other sleep indices (Ramsawh et al., 2016). The authors of these studies pointed to a lack of research on the effect of treatment on comorbid sleep difficulty despite its high prevalence.

CBT comes in different variations such as individual therapy, group sessions, and exposure-based treatments. One such concentrated treatment, the Bergen 4-day treatment (B4DT) is based on individually tailored exposure training combined with group sessions. The B4DT has mostly been tested for patients with OCD, but preliminary results suggest it can be helpful for patients with panic disorder (Iversen et al., 2022) and social anxiety disorder (Hansen et al., 2024). B4DT for OCD has proven effective in multiple studies, with remission rates up to 90% at post-treatment, 70% at 3-month follow-up, with the change maintained at long-term follow-up (Hansen et al., 2018, 2019; Kvale et al., 2018).

Some recent studies have tried to explore the B4DT's effect on concomitant insomnia. The first looked at a relatively small ($N = 36$) sample of which 70% had comorbid insomnia (Nordahl et al., 2018). The patients reported small ($d = 0.26$) reductions in insomnia at post-treatment and medium reductions ($d = 0.55$) at follow-up. A randomized control study (RCT) of B4DT for OCD showed a moderate ($d = 0.53$, $N = 48$) improvement in insomnia symptoms at 3-month follow-up (Hagen et al., 2021). Insomnia did not affect OCD treatment outcome. It is important to note that the B4DT for OCD does not directly address insomnia problems and that insomnia outcomes were included as secondary outcomes in these studies.

Related research (not B4DT) using pediatric samples or transcranial magnetic stimulation treatment have shown mixed results regarding sleep disturbances as a moderator of OCD treatment response. Two studies suggested that sleep problems could be associated with worse outcomes (Ivarsson & Skarphedinsson, 2015; Donse, Sack, Fitzgerald, & Arns, 2017), while another study found that although patients with insomnia had higher OCD severity, they still improved as much as patients without insomnia (Sevilla-Cermeño et al., 2019).

The study presented here is based on a study investigating the effects of B4DT on difficult-to-treat OCD and whether the effects were augmented by D-Cycloserine (DCS) (Kvale et al., 2020). There have been efforts to test medications which may augment and facilitate extinction learning. One such medication is DCS which works by influencing glutamatergic neurotransmission, specifically targeting N-methyl--D-aspartate (NMDA) receptors. Originally administered in larger doses as a treatment for tuberculosis, DCS has in more recent years undergone a lot of testing as a supplement medication to CBT for anxiety disorders. A meta-analysis reported small effects from pre-to post-treatment, not maintained at follow-up (Mataix-Cols et al., 2017), and another found effect sizes close to zero (Bürkner et al., 2017).

With regards to DCS' effects on insomnia, the findings are limited. The first study reported a possible effect: "such patients no longer experienced difficulty in falling asleep; they awakened well rested and with a general feeling of relaxation" (Crane, 1961, p. 54). In a RCT study looking at PTSD-treatment augmented with DCS, there was a significant improvement in sleep problems, with further improvement at follow-up (Difede et al., 2014).

In this study, we present a RCT with difficult-to-treat OCD receiving B4DT. Two groups received 100 mg and 250 mg of DCS, whilst the third group received placebo. Assessments were made at pre-treatment, post-treatment, 3-month and 12-month follow-up. We hypothesized that B4DT would improve insomnia symptoms. Secondly, the study explored whether there were differences in insomnia symptom reduction among the three groups. Lastly, it was hypothesized (based on previous B4DT studies) that insomnia would not impair the treatment of OCD nor impact the treatment's effect on depressive symptoms.

2. Methods

Fifteen specialized adult OCD clinics, established by Norwegian health authorities across the country, participated in the study, with nine of these teams' recruiting participants. The OCD clinic in Bergen organized the study and trained therapists. The study involved eight group leaders and 64 therapists. The study received approval from regional committees for medical and health research in Norway (reference number: 2013/195), and written consent was obtained from all participants.

2.1. Design and procedures

Across four consecutive days, concentrated exposure and response prevention (ERP) treatment was administered to participants in groups of three to six patients, each with an assigned therapist. The second and third day were dedicated to exposure treatment. The first day of the B4DT consisted of 2 h of psychoeducation followed by 2 h of planning individualized exposures. Both were conducted in a group setting. The second day started with repeating important elements from the psychoeducation before starting with therapist-assisted exposures. The participants met back for lunch and shared their experiences with the group. After lunch they continued with therapist-assisted exposure. In the evening, the patients carried out unassisted exposure exercises and reported on their progress by texting their therapist. The third day followed the same structure but also included a psychoeducation session for family/relatives. On the fourth day participants met with the group and planned how they could integrate treatment principles in their everyday lives, summarizing lesson learned, and planned exposure exercises they could conduct themselves in the following three weeks.

During the two exposure days, participants were given DCS, with dosages of 100 mg or 250 mg or placebo. Use of different dosage was due to the lack of clear guidelines (Rosenfield et al., 2019; Bürkner et al., 2017). The study employed a 3-group, placebo-controlled, triple--masked design, stratifying participants based on antidepressant use, due to findings of impairment on treatment response (Andersson et al., 2015). Randomization occurred within each stratum, assigning 100 mg, 250 mg, or placebo in a 2:2:1 ratio among a sample of 163 patients. Randomization, performed in blocks of five via an online tool, remained concealed from independent assessors, therapists, and patients. The distribution among groups was as follows: 67 (41.1%) in the 250 mg group, 65 (39.9%) in the 100 mg group, and 31 (19.0%) in the placebo group. Patients were assessed at pre-treatment, post-treatment, 3-month follow-up, and 12-month follow-up. The study's announcement was facilitated through the Norwegian OCD association's and OCD teams' websites, along with diverse media outlets. Recruitment spanned from January 2016 to August 2017.

Patients included in the study met criteria for OCD according to the Diagnostic and Statistical Manual of Mental Disorders. For inclusion, the Yale-Brown Obsessive Compulsive Scale (Y-BOCS; Goodman, Price, Rasmussen, Mazure, Delgado, et al., 1989) and the Mini International Neuropsychiatric Interview (MINI; Sheehan et al., 1998) were used for screening all patients. All patients who received a preliminary OCD diagnosis according to the MINI, also had a SCID-5 diagnostic interview (First et al., 2015). These interviews were conducted by trained independent assessors. Also, 20% of the taped interviews were rated by a second assessor. Diagnostic agreement was high with an obtained kappa coefficient of 0.92. Y-BOCS interviews underwent the same procedure with an intraclass correlation coefficient of 0.94.

Included patients were adult outpatients, fluent in Norwegian, who had not responded to ERP treatment earlier or had experienced a relapse after responding to ERP treatment. The definition of non-responders was less than 35% reduction in OCD symptoms and a Y-BOCS score of 16 or more. The definition of relapse was minimum 35% increase from post-treatment Y-BOCS score; a Y-BOCS score of 16 $\geq$, in addition to a Clinical Global Impression (CGI) improvement score of 6 $\geq$ (i.e. "much worse"). To be able to participate, a minimum of 4 weeks since the end of last treatment was needed.

Patients with the following were excluded: ongoing substance abuse

with or without dependence; psychosis or bipolar disorder; active suicidal ideation or plans; patients changing dosages of antidepressants; not willing to abstain from anxiety-reducing substances during the exposure; intellectual disability; or had to travel more than 1 h by train or car. Related to the DCS exclusion criteria were the following: pregnancy or breastfeeding, kidney impairment, porphyria, hypersensitivity to DCS and epilepsy.

2.2. Participants

Table 1 shows a summary of the pre-treatment characteristics of the sample. The mean age was 35 years and 72% were female. A total of 71% qualified for self-reported insomnia (scoring above cutoff on the BIS), while only 12% was diagnosed with insomnia or hypersomnia. The mean age of OCD onset was 19 years old, and the mean duration for OCD was 16 years. Following their last treatment, 62% were defined as having relapsed, while 38% were non-responders. There were no significant differences between the three groups apart from previous pre-treatment Y-BOCS scores, where the 250 mg group's mean score was lower than the two other groups. Eleven patients used sleep medication.

2.3. D-Cycloserine

On each of the two days of exposure it was administered one capsule of DCS with the respective 100 mg or 250 mg or one capsule of placebo. Both the DCS and placebo came in identical capsules produced by a research pharmacy. A letter containing information about DCS accompanied with a telephone number to address questions or to report adverse events. In some cases, when the exposure is short and does not result in a reduction in anxiety, there have been indications that DCS might potentiate negative experiences (Hofmann, 2014). To reduce the risk of this happening, the capsule was not taken before lunchtime on the second day, when the patient was familiar with the procedure.

Table 1
Pre-treatment characteristics of the sample (M(SD)/%).

Characteristics	Total	250 mg	100 mg	Placebo
Insomnia				
BIS, self-reported	70.8	63.9	75.0	75.9
Insomnia, diagnosed	8.6	5.8	6.2	19.4
Hypersomnia, diagnosed	4.3	4.5	3.1	6.5
Insomnia/ hypersomnia	12.0	9.0	9.2	25.8
Demographics				
Age	34.60 (10.87)	34.82 (11.75)	35.38 (11.42)	32.42 (7.06)
Female gender	71.8	67.2	75.4	74.2
OCD onset years	18.70 (9.92)	19.21 (10.29)	19.12 (10.70)	16.79 (7.22)
OCD duration years	16.17 (10.17)	15.89 (9.56)	16.58 (10.87)	15.93 (10.26)
OCD in family	42.1	41.3	41.9	44.4
Employment				
Working	34.4	33.3	38.5	30.0
Student	20.2	27.3	12.3	23.3
Disability	44.2	39.4	49.2	46.7
Single	47.5	48.5	43.1	55.2
Comorbid disorder	69.3	68.7	72.3	64.5
Psychotropic medication	46.6	37.3	55.4	48.4
SSRIs	31.9	32.8	33.8	25.8
Last treatment				
Y-BOCS pre	26.83 (5.00)	25.61 (4.79)	27.69 (4.99)	27.61 (5.07)
Y-BOCS post	14.14 (6.05)	14.51 (6.63)	13.52 (5.48)	14.69 (5.98)
Non-responder	38.7	43.3	35.4	35.5
Relapse	61.3	56.7	64.6	64.5
Current treatment				
Y-BOCS pre	25.16 (4.13)	24.68 (3.93)	25.52 (4.15)	25.42 (4.54)
PHQ-9 pre	11.96 (5.92)	11.40 (6.21)	12.75 (6.18)	11.43 (4.51)

Note. BIS, Bergen Insomnia Scale; OCD, Obsessive Compulsive Disorder; SSRIs, Selective Serotonin Reuptake Inhibitors; Y-BOCS, Yale-Brown Obsessive Compulsive Scale, PHQ-9, Personal Health Questionnaire-9; Pre, pre-treatment; Post, post-treatment.

2.4. Measures

The Bergen Insomnia Scale (BIS; Pallesen et al., 2008) was used to assess insomnia problems. The scale is based on the clinical criteria for insomnia outlined in the DSM-IV-TR. The first three items: difficulties with sleep onset, maintenance, and early morning wakening; correspond with criteria A (nocturnal symptoms) in DSM-IV, regarding difficulty initiating and maintaining sleep through the night. The last three items: not feeling adequately rested; experiencing daytime impairment, and feeling dissatisfied with sleep; correspond with criteria B. These two groups of items have been found to factor together in a clinical sample (Pallesen et al., 2008). The total score of BIS ranges from 0 to 42, where higher scores indicate more symptoms of insomnia. To qualify as clinically significant, one must score 3 or more on one of the items from 1 to 4 and 3 or more on item 5 or 6. The scale has shown good psychometric properties (Pallesen et al., 2008). Cronbach's alpha was 0.82.

The Yale-Brown Obsessive-Compulsive Scale (Y-BOCS; Goodman, Price, Rasmussen, Mazure, Delgado, et al., 1989; Goodman, Price, Rasmussen, Mazure, Fleischmann, et al., 1989) is a clinical interview used to assess severity of obsessive-compulsive symptoms. It consists of five items that measure obsessive symptoms on a Likert-scale from 0 (no symptoms) to 4 (severe symptoms), and likewise five items that measure compulsive symptoms from 0 to 4. The Y-BOCS total score is the sum of the ten items with a range from 0 to 40. A higher score indicates more severe symptoms of OCD. Y-BOCS has documented solid psychometric qualities (Goodman, Price, Rasmussen, Mazure, Delgado, et al., 1989; Goodman, Price, Rasmussen, Mazure, Fleischmann, et al., 1989; Goodman, Price, Rasmussen, Mazure, Delgado, et al., 1989). Cronbach's alpha was 0.73.

The Patient Health Questionnaire-9 (PHQ-9; Kroenke et al., 2001) is a self-report questionnaire consisting of nine items related to diagnosing depression in DSM-IV. PHQ-9 measures symptoms during the last two weeks on a Likert-scale from 0 (not at all) to 3 (almost every day). The total score ranges from 0 to 27, where a score from 5 to 9 is categorized as mild depressive symptoms; 10–14 as moderate; 15–19 as moderately severe and >19 as severe (Kroenke et al., 2001). Cronbach's alpha was 0.86.

2.5. Statistical analysis

To investigate the effect across time in self-reported insomnia and whether there were differences in self-reported insomnia between the treatment conditions, a repeated split plot ANOVA was conducted. BIS was the dependent variable and condition (250 mg, 100 mg, and placebo) was the grouping variable. A repeated split plot ANOVA with the dependent variables Y-BOCS and PHQ-9 was also used to examine the general effect of B4DT on symptoms of OCD and depression. Effect sizes were calculated with pooled standard deviations.

To test if insomnia moderated treatment outcome, changes in Y-BOCS, BIS, and PHQ-9 were separately compared over time (pre-treatment, post-treatment, 3-month follow-up and 12-month follow-up) for patients who according to BIS qualified for insomnia vs. patients who did not. A repeated split plot ANOVA was also used for this purpose. The same procedure was also conducted for patients with no diagnosed insomnia vs. patients with diagnosed insomnia ($n = 14$) and hypersomnia ($n = 7$), which were merged to one variable ($n = 20$, one person was diagnosed with both conditions).

Missing items were imputed using the Expectation-Maximization method; 6.4% missing values for BIS. Data was missing completely at

random, $x^2 = 257.42$, $df = 278$, $p = 0.81$. Missing values were not replaced if patients had missing values for all items of a measure ($n = 9$), hence the total sample size was 154.

3. Results

At 1-year follow-up, the total Y-BOCS score for all three conditions was significantly reduced with a large effect size of $d = 2.17$. The total PHQ-9 score for all three conditions was also significantly reduced with a small to medium effect size, $d = 0.41$. Symptoms of insomnia were also significantly reduced at 1-year follow-up with a small effect size, $d = 0.23$. There was no significant interaction effect between time and condition on self-reported insomnia ($p = 0.87$). A summary of changes in symptoms of insomnia, OCD, and depression is shown in Table 2. For patients with self-reported insomnia the effect size on the BIS (from pre-treatment to 12-month follow-up) was 0.36, while patients with no self-reported insomnia had no clear changes on the BIS ($d = 0.01$).

Before treatment there were 109 patients (70.8%) with self-reported insomnia (total $N = 154$). After one year it was reduced to 81 (52.6%) (see Fig. 1), with an effect size of $d = 0.36$.

There was no significant interaction effect between self-reported insomnia and changes in symptoms of OCD or depression (see Table 3). Similar results were obtained for patients with diagnosed insomnia/hypersomnia as there was no significant group*time interaction on Y-BOCS ($p = 0.49$) and PHQ-9 ($p = 0.95$).

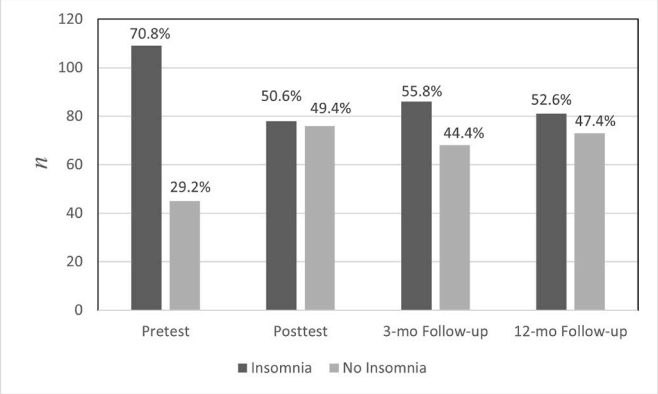

Fig. 1. Changes in Self-reported Clinical Insomnia (BIS)
Note. Clinical Insomnia measure according to BIS, Bergen Insomnia Scale; 3-mo Follow-up, 3-months follow-up; 12-mo Follow-up, 12-month follow-up; $N = 154$.

4. Discussion

This study aimed to investigate the effects of concentrated exposure and response prevention treatment on insomnia symptoms, explore the potential augmentation by DCS, and examine how clinical insomnia might moderate the treatment effect. A significant effect of B4DT on insomnia symptoms was found. No difference between the groups was discovered, showing no augmentation from DCS. Furthermore, clinically insomnia did not moderate OCD-treatment outcomes.

The first hypothesis was confirmed, adding to the small, but growing literature on the effects on concomitant insomnia for the B4DT specifically and CBT for anxiety disorders more generally. The effect size at 3-month follow-up in this study ($d = 0.38$) was slightly weaker compared to the moderate ones found ($d = 0.55$; 0.53) at 6-month and 3-month follow-up in earlier studies on the B4DT (Hagen et al., 2021; Nordahl et al., 2018). The smaller effect size could be attributed to the larger sample size in the present study and the sample comprising participants with difficult-to-treat OCD. Still, a robust effect was found and one possible mechanism behind this could be the bidirectional model concerning the relationship between OCD and insomnia (Cox et al., 2018) and the transdiagnostic model (Harvey et al., 2011). As the participants received concentrated ERP, they learned techniques to deal with their OCD (increased cognitive control), which in turn may lessen the rumination and negative activation often found in insomnia. However, many patients still reported insomnia (52% at 1-year follow-up). Despite successful OCD treatment, insomnia may persist and could increase the risk of relapse (Carney et al., 2011; Jansson-Fröjmark et al., 2016). Future research should therefore explore how integrated, parallel, or sequential treatment of insomnia could benefit patients with OCD and insomnia.

The high prevalence of self-reported insomnia (70%) at pre-treatment added to the growing consensus in the literature of a consistent link between OCD and insomnia (Cox et al., 2020). However, only 14 of the participants were diagnosed with comorbid insomnia and seven with hypersomnia. A similar difference was found in the (Hagen et al., 2021) study with 81% reporting insomnia on the BIS opposed to 19% on the clinical interview. This large discrepancy in both studies is interesting as both measures are based on the DSM diagnostic criteria (the 4th version for BIS and the 5th for SCID-5), but the BIS does not differentiate between primary insomnia and insomnia explained by coexisting mental conditions (Pallesen et al., 2008). When using DSM-5 diagnostic criteria, insomnia explained by other mental illnesses should not get coded as comorbid insomnia (DSM-5, 2022). This means that sleep difficulties presumed by the assessor to be explained by OCD,

Table 2
Changes in insomnia, symptoms of OCD, and depression.

Measure	Pre	Post	3 m FU	12 m FU			*Effect size (d)*			time * group
					F	p	Post	3 m	12 m	p
BIS										
250 mg	16.15 (8.28)	12.92 (10.60)	12.23 (8.59)	13.85 (9.69)			0.36	0.46	0.26	
100 mg	19.45 (10.62)	14.97 (10.60)	15.29 (10.43)	16.65 (10.98)			0.42	0.40	0.26	
Placebo	18.27 (9.71)	15.07 (11.92)	16.00 (9.48)	17.12 (10.78)			0.29	0.24	0.11	
Total	17.92 (9.64)	14.17 (10.84)	14.21 (9.64)	15.63 (10.48)	12.70	<0.001	0.37	0.38	0.23	0.87
Y-BOCS										
250 mg	26.60 (4.04)	11.75 (5.44)	13.23 (7.58)	14.63 (7.38)			3.10	2.20	2.01	
100 mg	27.24 (3.67)	11.78 (5.75)	13.25 (6.63)	14.19 (7.09)			3.21	2.61	2.31	
Placebo	27.20 (3.83)	14.27 (7.16)	16.10 (7.23)	14.30 (7.62)			2.25	1.92	2.14	
Total	26.98 (3.83)	12.28 (6.00)	13.83 (7.18)	14.38 (7.26)	246.16	<0.001	2.92	2.29	2.17	0.28
PHQ-9										
250 mg	11.47 (6.04)	7.12 (6.02)	6.54 (5.55)	8.61 (5.93)			0.72	0.85	0.48	
100 mg	12.77 (5.90)	8.78 (6.12)	9.17 (6.29)	10.27 (6.32)			0.50	0.59	0.41	
Placebo	11.44 (5.70)	7.60 (5.56)	8.48 (4.21)	10.00 (6.07)			0.68	0.59	0.24	
Total	12.01 (5.70)	7.91 (6.01)	7.99 (5.77)	9.56 (6.13)	33.73	<0.001	0.70	0.70	0.41	0.63

Note. BIS, Bergen Insomnia Scale; Y-BOCS, Yale-Brown Obsessive Compulsive Scale; PHQ-9, Patient Health Questionnaire-9. Pre, pre-treatment; Post, post-treatment; 3 m FU, 3-month follow-up; 12 m FU, 12-month follow-up. $N = 163$.

Table 3
Test of moderation effects of self-reported insomnia (BIS) on OCD and depression outcomes.

Measure	Pre	Post	3 m FU	12 m FU			*12m*	time * group
	M (SD)				*F*	*p*	*d*	*p*
Y-BOCS								
Insomnia	27.17 (3.20)	12.32 (5.92)	13.52 (6.94)	14.67 (6.88)			2.33	
No Insomnia	26.53 (3.20)	11.63 (5.88)	14.25 (7.57)	14.00 (6.88)			2.34	
Total	26.99 (3.75)	12.12 (5.89)	13.73 (7.11)	14.48 (7.27)	222.24	<0.001	2.16	0.61
PHQ-9								
Insomnia	13.46 (5.59)	8.74 (6.30)	9.12 (5.96)	11.01 (6.18)			0.42	
No Insomnia	8.49 (3.99)	5.46 (4.11)	5.20 (4.03)	6.07 (4.13)			0.60	
Total	12.00 (5.64)	7.78 (5.92)	7.97 (5.74)	9.56 (6.08)	30.89	<0.001	0.42	0.19

Note. BIS, Bergen Insomnia Scale; Y-BOCS, Yale-Brown Obsessive Compulsive Scale; PHQ-9, Patient Health Questionnaire-9; Pre, Pre-treatment; Post, Post-treatment; 3 m FU, 3-month follow-up; 12 m FU, 12-months follow-up. $N = 154$. The grouping variable of insomnia vs. no insomnia is based on scoring above or below the cutoff on BIS.

would not get labeled as comorbid insomnia. Another potential reason for the discrepancy is that the BIS correspond to symptoms experienced the last month, while the DSM-5 requires insomnia symptoms over a period of at least three months.

The second hypothesis explored augmentation of DCS and showed no effect in comparison with the placebo group. This is in line with a meta-analysis showing no effect of DCS (Bürkner et al., 2017). The authors pointed out that the newer, high-quality studies report smaller or no-effects of DCS on anxiety disorders. Thus, the present study, with a large sample size and robust controls, could be expected to show a smaller or non-significant effect. Additionally, the Kvale et al. (2020) study, based on the same data as this study, found no effect on OCD-symptoms between the groups. If there exists a bi-directional relationship between OCD and insomnia, no differential effect on OCD might warrant no effect of DCS on insomnia. As mentioned, there exists only one controlled study looking at the effects of DCS on sleep (Difede et al., 2014). They found a significant effect but highlighted the need for specific sleep assessment and larger samples. As this study on OCD found no effect, improvement of insomnia symptoms augmented by DCS seems less likely to be a promising route.

The third hypothesis was confirmed, showing non-significant differences in OCD outcome between patients with indications of clinical insomnia and the one without. The same was found for the group with diagnosed comorbid insomnia. This is in accordance with other studies showing no moderation effects of insomnia on OCD treatment (Hagen et al., 2021; Nordahl et al., 2018). This implicates that insomnia should not be considered an exclusion criterion for OCD-treatment, nor as impairing treatment. This contrasts with an earlier review by Cox et al. (2018) showing that sleep disturbance did limit OCD treatment response in some samples. One reason why sleep difficulties did not impair treatment could be the intensive format of the B4DT.

There are some important limitations of this study that should be considered. Firstly, the sleep assessment was based on self-reported insomnia symptoms. It can only give indications about clinical insomnia, while the full clinical interview found a different prevalence of clinical insomnia in the sample. Furthermore, objective metrics, such as actigraphy or polysomnography would provide a more complete picture of the participants' sleep quality (Nota et al., 2015). One could also consider whether the dosing procedure chosen for this study was optimal. A recent review of the DCS literature relates the decline in effect in DCS research to suboptimal dosing regimens (Rosenfield et al., 2019). They investigated most studies on DCS and showed how the studies who administer more doses (up to 9) and 60 min prior to treatment had the largest effect. While something to consider for future research, it would not be feasible in the present study due to the concentrated format.

The results of this study are promising regarding improving concomitant insomnia with OCD-treatment, while showing less promise in augmenting this treatment with DCS. Future research should focus on doing controlled studies specifically targeting insomnia with more comprehensive measures of sleep difficulty. While reduced, the symptom severity of sleep difficulties for this sample remains high. Testing specific treatment for insomnia is prudent, with CBT-I treatment recommended as first-line treatment for insomnia showing clinically meaningful effect sizes (Koffel et al., 2015; Trauer et al., 2015).

In conclusion, this randomized clinical trial found no potentiation of DCS for symptoms of insomnia following response prevention and concentrated exposure treatment for OCD. A weak improvement on insomnia symptoms following treatment were found for all groups, strongest at post-treatment. Insomnia symptoms did not moderate treatment outcome. Future research should test the effect of specific insomnia treatment for patients with OCD and comorbid insomnia.

Funding statement

This research was supported by project 243675 of the Research Council of Norway, HELSEFORSK.

CRediT authorship contribution statement

Nils Eivind Holth Landrø: Writing – review & editing, Writing – original draft, Formal analysis. **Sigurd Holmen Pryser:** Writing – review & editing, Writing – original draft, Formal analysis. **Kristen Hagen:** Writing – review & editing, Project administration, Conceptualization. **Bjarne Hansen:** Writing – review & editing, Project administration, Funding acquisition, Conceptualization. **Gerd Kvale:** Writing – review & editing, Project administration, Funding acquisition, Conceptualization. **Stian Solem:** Writing – review & editing, Supervision, Formal analysis, Data curation.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Data availability

Data will be made available on request.

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