



Review article

Cognitive Behavioural Therapy for Insomnia (CBT-I) to treat depression: A systematic review

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ARTICLE INFO

Keywords:

CBT-I
Cognitive Behavioural Therapy
Depression
Group therapy
Insomnia
Telehealth

ABSTRACT

Introduction: Major depressive disorder is one of the most commonly diagnosed psychiatric illnesses, and it has a profound negative impact on an individual's ability to function. Up to 90% of individuals suffering from depression also report sleep and circadian disruptions. If these disruptions are not effectively resolved over the course of treatment, the likelihood of relapse into depression is greatly increased. Cognitive Behavioural Therapy for Insomnia (CBT-I) has shown promise in treating these sleep and circadian disturbances associated with depression, and may be effective as a stand-alone treatment for depression. This may be particularly relevant in cases where antidepressant medications are not ideal (e.g. due to contraindications, cost, or treatment resistance).

Methods: A systematic literature review was conducted of trials investigating the use of CBT-I to treat depression in adults. Therapy included in-person CBT-I, as well as telehealth and group CBT-I.

Results and conclusions: CBT-I presents a promising treatment for depression comorbid with insomnia. In-person therapy has the most supporting evidence for its efficacy, though treatment effects may not be additive with those of antidepressant medications. Insomnia improvement due to CBT-I may mediate the improvement in depressive symptoms. There is less evidence for the use of telehealth, though a stepped-care approach is indicated based on baseline depressive severity. More research on group therapy and telehealth modalities of delivering CBT-I are required before making recommendations.

1. Introduction

Major depressive disorder (MDD) has widespread negative effects on an individual's life, involving the presence of pervasive depressed mood or loss of pleasure and enjoyment, as well as distress or impairment in important areas of life including social and occupational functioning [1]. It is also one of the most frequently diagnosed psychiatric disorders [2], with a lifetime prevalence of approximately 16% [3]. First-line treatment of depression often consists of the prescription of an antidepressant medication (ADM). However, drug-resistant depression is of increasing concern [4]. Additionally, many antidepressant medications have questionable clinical efficacy compared to a placebo (e.g. [5,6]). For example, a study by Fournier et al. found that antidepressant medications had limited therapeutic effect for patients displaying only mild or moderate symptoms of depression [7]. Additionally, ADMs usually take several weeks to exert therapeutic effect on mood, which can lead to further disruption in personal, social, and occupational life, as well as increase the risk of suicide [8–10]. There is

currently a dearth of clinically accepted non-pharmacological treatments for depression, especially those which have a rapid mechanism of action and which work for patients of varying symptom severity. If such treatments were developed, it would have a positive impact on public health [6,9].

One possible avenue for future such treatments are by targeting sleep and circadian systems. Sleep and circadian disturbances frequently are reported in individuals suffering from depression [11–13], with up to 84–90% of individuals suffering from depression endorsing sleep complaints [13,14]. These sleep complaints predict worse clinical and treatment outcomes, and are associated with increased suicidal ideation and risk [11–15]. Sleep-wake disruptions often need be addressed in treatment in order to avoid relapse of depression after successfully treating the disorder [13]. If sleep or fatigue complaints are the only symptoms remaining after successful treatment, the risk of relapse in depression is greatly increased [14,16,17]. Thus, it makes sense that treatments targeting both sleep complaints and depressive symptoms often lead to larger and more sustained mood improvements

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Table 1
Characteristics of 9 studies which investigate the use of in-person Cognitive Behavioural Therapy for Insomnia (CBT-I) to treat depression.

First author, year	Subjects	n	Study design	Treatment groups/ reference	Depression outcome measure	Medication use	CBT-I dosage	Outcomes	Effect size	Retention (RT) and adverse events (AE)
Ashworth, 2015	Comorbid MDD and insomnia, treatment resistant	41	RCT	Self-help CBT-I, in-person CBT-I groups	BDI-II	6 weeks, stable ADMs; No sleep medication	4 × 50 min sessions over 8 weeks	CBT-I more effective than self-help CBT-I, $p < 0.001$ 94% of in-person group improved at least 7 points (39% of self-help group) at 3 month FU; $p < 0.001$ 78% of in-person group were in clinical remission (17% of self-help group) at FU; $p < 0.001$ 61% of in-person group was in remission (6% of self-help group) for insomnia and depression at FU; $p < 0.001$ Difference in depression between groups at FU mediated by change in insomnia severity NNT = 1.8	$d = 1.24$ (post-tx) $d = 1.65$ (FU)	RT at post-tx: In-person CBT-I = 21/21 Self-help = 20/21 RT at follow-up: CBT-I = 18/21 Self-help: 18/20
Carney, 2017	MDD, insomnia	107	RCT	ADM ^a + CBT-I, CBT-I + placebo, ADM + SH groups	HAMD-17	No hypnotics, ADMs part of trial	4 × 60 min sessions, biweekly	All groups improved over time, $p < 0.05$ No differences between groups on HAM-D-17 after treatment, $p = 1.0$ Very small between group effect size between CBT-I + ADM and CBT-I + placebo Small between group effect size between CBT-I + ADM and ADM + SH (HAM-D-17) Small between group effect size between CBT-I + placebo and ADM + SH (HAM-D-17) Rate of remission numerically higher in CBT-I group (62%) than control (33%), $p = 0.13$ Change in score over time for CBT-I group (12) larger than control (9.7) Change in score (no sleep item) over time for CBT-I group (9.7) larger than control (7.9) 43.8% in CBT-I group attained remission (36.0% in control) No significant difference in time to remission by group ($p = 0.33$) No significant difference in HDRS scores between groups after treatment ($p = 0.48$) Insomnia improvement in first 6 weeks mediated depression remission in CBT-I group ($p = 0.0004$)	$d = 0.02$ $d = 0.32$ $d = 0.32$	RT at post-tx: ADM + CBT-I = 26/36 CBT + placebo = 21/36 ADM + SH = 20/35 RT at FU: ADM + CBT = 9/11 CBT + placebo = 9/10 ADM + SH = 6/8 AE: reason for withdrawal from ADM + SH group
Manber, 2008	MDD, insomnia	30	RCT	ADM ^b + CBT-I or ADM + Quasi-Desensitization Control	HRSD ₁₇ , Depression portion of SCID	No hypnotics, ADMs part of trial	7 sessions, 5 weekly then 2 biweekly			RT at post-tx: CBT-I = 10/15 Control = 12/15
Manber, 2016	MDD, insomnia	150	RCT	ADM ^c + CBT-I or ADM + Quasi-Desensitization Control	HDRS ₁₂ , Depression portion of SCID	ADMs part of trial	7 × 45 min sessions over 16 weeks (4 weekly, 2 biweekly, final after 4 weeks)		NNT = 15	RT at post-tx: CBT = 58/75 Control = 51/75
Pigeon, 2017		27	RCT	Brief CBT-I or SH	PHQ-9				$g = -0.04$	

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