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Table of contents

Acknowledgements	5
1. Introduction.....	6
2. Theoretical Background.....	7
2.1 Sleep mechanisms.....	7
2.1.1 Sleep cycles	8
2.1.2 Circadian rhythms	8
2.1.2 Functions of sleep	9
2.2 Insomnia disorder	10
2.2.1 Epidemiology.....	11
2.2.2 Pathophysiology	13
2.3 Insomnia treatment	15
2.3.1 Cognitive Behavioral Therapy for insomnia.....	15
2.3.2 Implementation of CBTi.....	18
3. Research objectives and hypotheses.....	22
4. Method.....	27
4.1 Setting eligibility criteria	28
4.1.1 Population	28
4.1.2 Intervention.....	28
4.1.3 Comparator	28
4.1.4 Outcome.....	29
4.1.5 Study characteristics	29
4.2 Assessment of treatment outcomes	29
4.2.1 Insomnia Severity Index	30
4.2.2 Pittsburgh Sleep Quality Index	30
4.2.3 Sleep Condition Indicator	31

4.2.4 Holland Sleep Disorder Questionnaire	31
4.2.5 Chronic Sleep Reduction Questionnaire	31
4.3 Searching for and selecting studies.....	31
4.4 Coding and data extraction	32
4.5 Categorization.....	32
4.6 Measuring study quality by risk of bias rating.....	33
4.7 Effect size calculation & statistical method.....	33
4.7.1 Specifying the effect of interest and choosing a model of analysis	34
4.7.2 Calculating the effect measures of individual studies	36
4.7.3 Checking data	39
4.7.4 Data analysis	39
5. Results	43
5.1 Identification of trials.....	43
5.2 Study characteristics	43
5.3 Hypotheses	49
5.4 Publication bias	59
5.5 Sensitivity analysis	59
5.6 Risk of bias	61
6 Discussion	61
6.1 Summary of results	61
6.2 Key findings.....	62
6.3 Additional findings	65
6.4 Limitations	65
6.5 Implications and future directions	68
References.....	70
List of figures	88

List of tables	89
List of abbreviations	90
Abstract (English)	91
Abstract (Deutsch).....	92
Appendix	93
Table A1	93
Figure A1.....	93

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1. Introduction

Insomnia is a common psychological disorder in the general population, with an estimated prevalence that varies from 6% to 19% across European epidemiological studies (Riemann et al., 2017), whilst prevalence rates among patients consulting a general practitioner seem to be even higher, with indications of a prevalence of 20% to 50% (Riemann et al., 2017). Insomnia is characterized by difficulty initiating and maintaining sleep and/or waking up too early and negatively impacts an individual's performance during waking hours. The disorder is frequently associated with adverse mental and physical health outcomes such as depression and anxiety (Roth, 2007; Sivertsen et al., 2012), cognitive impairment (Yaffe et al., 2014), poor self-rated health (Nomura et al., 2005) and cardiovascular disease (Sofi et al., 2014), as well as an impaired quality of life (Ishak et al., 2012). Missing competences in treating insomnia lead to patients suffering from insomnia for an extended period of time, with 56-74% of patients having insomnia for more than one year, and almost 50% for more than three years (Morin et al., 2009; Morin et al., 2015; Morphy et al., 2007; Pillai et al., 2015).

Cognitive behavioral therapy for insomnia (CBTi) is currently the recommended treatment for insomnia disorder, as it is highly efficient in reducing insomnia severity (Smith et al., 2002) yields better long-term effects than pharmacotherapy (Riemann & Perlis, 2009) and bypasses the side effects associated with pharmacotherapy. However, even though CBTi is commonly recognized as the superior treatment, it is rarely implemented (Spiegelhalder et al., 2020). Authors have speculated that implementational challenges might be to blame for the lack of use (Perlis & Smith, 2008), and alternative forms of applying CBTi have been developed. Digitally delivered CBTi (dCBTi) seems to be a promising treatment option, as various meta-analyses have demonstrated its effectiveness in reducing insomnia symptoms (Seyffert et al., 2016; Soh et al., 2020; Ye et al., 2016; Zachariae et al., 2016). However, it remains unclear which factors influence efficacy of dCBTi, whether dropout is comparable to face-to-face CBTi (f2f CBTi) and if results can be adopted for a diverse clinical population.

The aim of this review is to illustrate effectiveness of digital CBT for insomnia on a broad spectrum by including studies that focus on adolescents as well as older adults, studies on populations with and without clinical comorbidities, as well as recently published studies on the effectiveness of newly developed technologies. Factors possibly explaining

heterogeneity in effect size will be explored, and the odds for dropout for dCBTi and f2f CBTi examined.

The following chapters will address the mechanisms and function of sleep, as well as definition, epidemiology, and pathophysiology of insomnia disorder. Treatment options and their implications for clinical practice will be discussed and research regarding the efficacy of dCBTi presented. Research objectives and hypotheses are addressed and the individual steps in the metanalytic method described in detail. Results will be contrasted to those of previous meta-analyses and implications for clinical practice and future research discussed.

2. Theoretical Background

2.1 Sleep mechanisms

Sleep is a physiological state characterized by specific variations in the brain's electrical rhythms (Chokroverty, 2010). Due to electro-encephalography (EEG), electro-oculography and electromyography measurements, researchers were able to differentiate between distinct states of sleep, which can be grouped into non rapid eye movement (non-REM; about 75% of sleep) and rapid eye movement (REM) sleep (about 25% of sleep; Chokroverty, 2010). The EEG pattern during *REM sleep* is very similar to that during wakefulness, generating short waves of high frequency. The brain requires a lot of energy during REM phases, consuming even more oxygen than when individuals are awake and concentrated on complex problems, and the sympathetic system is activated, as heart and respiratory rate increase. During REM, the body is immobile apart from low activation of respiratory muscles and high activation of muscles controlling eye movement and muscles of the inner ear (Bear et al., 2015). During REM, eyes rapidly dart back and forth, which is predictive of when individuals experience dreaming. During *non-REM* muscle tension and movement is reduced and parasympathetic activity increases. Besides being essential for bodily rest, non-REM sleep seems to be essential for cognitive rest, as the firing rates of neurons are at their lowest during this phase. Non-REM sleep is characterized by EEG rhythms with small frequency and large amplitude, and mental processes are their lowest during this period. Non-REM sleep can be divided into four stages of distinct brain patterns (stage 1 to stage 4; Bear et al., 2015).

2.1.1 Sleep cycles

The phases of sleep recur successively in sleep cycles, which are repeated four to six times every 90 to 110 minutes (Bear et al., 2015; Chokroverty, 2010). Sleep is predominantly spent in non-REM sleep phases. When falling asleep, individuals go through the non-REM sleep stages before they reach REM sleep, after which the cycle repeats. The sleep stages successively turn deeper, with stage 1 being the lightest sleep phase with small highly frequent waves and stage 4 being the deepest stage of sleep, with large amplitudes and low frequency EEG rhythms. Stage 1 is a transitional stage between wakefulness and sleep that usually lasts for a few minutes and involves slow and rolling eye movements. The EEG alpha rhythms that are present during relaxed wakefulness turn into more irregular theta activity with frequency between 4 and 7 Hz. Stage 2 lasts for 5-15 minutes and involves almost no eye movements. This phase is characterized by the occurrence of oscillation of 8-14 Hz (sleep spindles) and K complexes, which are sharp waves of high amplitude with a frequency between 12 and 14 Hz (Halasz, 1993). Stage 3 involves slow delta activity of less than 4 Hz with large amplitudes and barely any movement of body and eyes. Stage 4 is the deepest sleep phase, with large delta waves of a frequency below 2 Hz. It lasts between 20-40 minutes, until sleep gets lighter and goes through stages 3 and 2 before a short REM-sleep phase is reached, which involves EEG beta (15-30 Hz) and gamma (30-90 Hz) waves of high frequency and rapid eye movements. As the duration of sleep progresses, there is an increase in REM sleep as non-REM sleep phases shorten. At that point, REM sleep phases last between 30 and 50 minutes, with at least 30 minutes of non-REM sleep in between REM sleep phases (Bear et al., 2015).

Among adults, the required sleep duration can vary between around five to 10 hours per night, with an average duration of 7.5 hours. Successful sleep is best measured by daytime functioning (Bear et al., 2015).

2.1.2 Circadian rhythms

The circadian rhythm is innate to human beings and regulated by a biological clock, which regulates sleep and wake patterns and is synchronized to other natural circadian rhythms such as daylight and darkness that are caused by earth rotation. Natural cues of circadian timing such as light, sounds, humidity or temperature help maintain individuals' biological clocks on a 24-hour rhythm (Bear et al., 2015). The biological clock is centered in the suprachiasmatic nuclei (SCN), has its own natural rhythm and controls certain cognitive and

physiological functions (Bear et al., 2015). The SCN coordinates the circadian rhythm of individual neurons and communicates that rhythm through neuronal activity (action potentials). In addition to the sleep-wake rhythm, the SCN controls the circadian rhythm of the autonomic nervous system, core body temperature, adrenal gland hormones and neural circuits responsible for feeding, metabolism and locomotion (Bear et al., 2015).

The circadian rhythm is complemented by the homeostatic sleep drive, and both systems help modulate the sleep-wake rhythm. After awakening, both sleep drive and neuronal activity of the SCN are low and increase during the day, until SCN activity decreases at the end of the day, resulting in sleep onset due to high homeostatic sleep drive. During sleep, the homeostatic sleep drive decreases and neuronal activity of SCN in the morning hours initiates awakening (Chokroverty, 2010).

2.1.2.1 Sleep promoting factors. The neurotransmitter adenosine promotes sleep as it inhibits diffuse modulatory neurotransmitter systems for acetylcholine, norepinephrine and serotonin that are associated with wakefulness (Bear et al., 2015). The levels of adenosine in sleep-related brain regions increase during the time awake, facilitating sleep by suppressing diffuse modulatory systems, and gradually decreases during sleep, ceasing its inhibitory effect on the modulatory systems and initiating awakening. The release of adenosine is caused by nitric oxide (NO), with NO levels rising during waking hours (Bear et al., 2015).

Melatonin is an endogenous substance that is inhibited by light and released when it gets dark, with levels rising in the evening and return to baseline in the morning, suggesting its involvement in sleep-initiation and maintenance (Bear et al., 2015).

2.1.2 Functions of sleep

Common conceptions regarding the general function of sleep can be assigned to theories of restoration, which state that sleep serves the purpose to rest for the day, and theories of adaption, suggesting that sleep helps keeping individuals safe while conserving energy during vulnerable periods (Cirelli & Tononi, 2008). However, as sleep deprivation has detrimental effects on physiological and cognitive functioning that cannot be prohibited by wakeful resting, it is evident that sleep does not solely serve a resting function, though researchers still do not understand the exact mechanisms involved (Bear et al., 2015; Cirelli & Tononi, 2008). Furthermore, reaching deep sleep phases (stage 3 and 4) as well as REM-sleep

seems to be necessary for sleep to have its restorative effect on physiological and cognitive functioning (Orzeł-Gryglewska, 2010).

Sleep is a prerequisite for essential cognitive and psychological processes and abilities (Tononi & Cirelli, 2006) such as learning, offline memory consolidation (Diekelmann & Born, 2010) and emotional processing (Gujar et al., 2011; McNamara et al., 2001; van der Helm et al., 2010). The quality of sleep has an influence on one's health and overall well-being, and sleep deprivation that results from sleep disturbances can lead to maladaptive functioning as well as numerous mental and physical illnesses (Brand & Kirov, 2011; Cirelli & Tononi, 2008). Immediate consequences of sleep deprivation are impaired perception, poor memorizing and concentration, schematic thinking, reduced attention and quality of life, emotional disturbances, more work absenteeism and increased risk for accidents (Chokroverty, 2010; Orzeł-Gryglewska, 2010). In the long term, sleep deprivation can result in a variety of somatic and mental disorders (Chokroverty, 2010).

2.2 Insomnia disorder

The fifth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5; American Psychiatric Association, 2013) offers specific and thorough diagnostic criteria for mental disorders (Hoyer & Knappe, 2020). It differentiates between 10 groups of sleep-wake disorders: insomnia disorder, hypersomnolence disorder, narcolepsy, breathing-related sleep disorders (which include obstructive sleep apnea hypopnea, central sleep apnea, and sleep-related hypoventilation), circadian rhythm sleep disorders, non-rapid eye movement (non-REM) sleep arousal disorders, nightmare disorder, rapid eye movement (REM) sleep behavior disorder, restless legs syndrome, and substance- or medication-induced sleep disorder. Non-REM sleep arousal disorder, nightmare disorder and REM sleep behavior disorder are categorized as parasomnias (American Psychiatric Association, 2013). The different disorders are characterized by patients being dissatisfied with their sleep quality, sleep timing, their amount of sleep, as well as experiencing daytime impairment and distress (Reynolds III & O'Hara, 2013).

The Diagnostic and Statistical Manual of Mental Disorders (DSM; American Psychiatric Association, 2013) the International Statistical Classification of Diseases and Related Health Problems (ICD; World Health Organization, 2019) and the International Classification system of Sleep Disorders (ICSD; American Academy of Sleep Medicine, 2014) each offer diagnostic

criteria for insomnia disorder. Although there are slight differences in insomnia criteria across these classification systems, all systems characterize insomnia disorder as having difficulties initiating sleep, problems maintaining sleep and/or waking up earlier than desired, as well as sleep being non-restorative.

According to the fifth edition of the DSM (American Psychiatric Association, 2013), insomnia disorder involves “a predominant complaint of dissatisfaction with sleep quantity or quality” (American Psychiatric Association, 2013, S. 362), and is accompanied by “difficulties in initiating sleep ..., difficulties in maintaining sleep, characterized by frequent awakenings or problems returning to sleep after awakenings ..., [or] early-morning awakening with inability to return to sleep.” (American Psychiatric Association, 2013, S. 362).

The sleep difficulty “... causes clinically significant distress or impairment in social, occupational, educational, academic, behavioral, or other important areas of functioning.” (American Psychiatric Association, 2013, S. 362). This includes symptoms such as fatigue, mood disturbances and impaired attention (American Psychiatric Association, 2013).

For a diagnosis to be made, the sleep difficulty must occur at least three nights per week, be present for at least three months and exist despite adequate opportunity for sleep (American Psychiatric Association, 2013).

The insomnia is not better explained by and does not occur exclusively during the course of another sleep-wake disorder (e.g., narcolepsy, a breathing-related sleep disorder, a circadian rhythm sleep-wake disorder, a parasomnia). The insomnia is not attributable to the physiological effects of a substance (e.g., a drug of abuse, a medication) [and] coexisting mental disorders and medical conditions do not adequately explain the predominant complaint of insomnia (American Psychiatric Association, 2013, S. 362).

The insomnia diagnosis is made in relation to a patient’s subjective experience of sleep difficulties, as well as complaints of substantial distress and daytime impairments (Morin et al., 2015). The completion of a sleep diary is helpful in the correct assessment of sleep problems, as the records are less influenced by recall biases than a retrospective assessment of a global score (Morin et al., 2015).

2.2.1 Epidemiology

Insomnia is highly common among the general population, with prevalence rates ranging between 5.7% and 19% across European epidemiological studies (Riemann et al.,

2017), whilst rates among patients that consult a general practitioner indicate even higher occurrence of 20% to 50% (Riemann et al., 2017). Insomnia symptoms (as opposed to insomnia diagnoses) seem to be present in about 30-40% of the population, as studies from around the world record similar estimates (Morin et al., 2011; Morin et al., 2015; Ohayon & Reynolds, 2009).

Insomnia frequently coincides with other physiological and mental disorders, (Budhiraja et al., 2011; Pearson et al., 2006; Roth et al., 2006; Sarsour et al., 2010) and is therefore more common among people with medical or mental disorders compared to the general population (Pearson et al., 2006). Moreover, patients suffering from sleep disturbance in addition to another primary mental condition report higher symptom and disorder severity, a lower level of functioning and less treatment benefits (Espie et al., 2012a).

An epidemiological report revealed that 40% of insomnia patients suffer from at least one additional mental disorder, with 24% being diagnosed with anxiety disorder, 23% with major depression or dysthymia, 7% with alcohol abuse and 4% with drug abuse (Ford & Kamerow, 1989). Insomnia symptoms are especially prevalent amongst depressed individuals, with 91% of patients with major depressive disorder experiencing insomnia symptoms (Seow et al., 2016).

The relationship between insomnia and other mental disorders seems to be bidirectional. Insomnia can be a consequence as well as a predisposing factor to certain psychiatric conditions such as depression, anxiety (Johnson et al., 2006; Lustberg & Reynolds, 2000; Neckelmann et al., 2007; Siversten et al., 2012) and substance abuse disorder (Roane & Taylor, 2008), and treating either condition can benefit the recovery of the other (Morin et al., 2015).

Furthermore, people with insomnia have a high risk for comorbid medical disorders (Roth, 2007). Insomnia is related to several indicators of physical health such as poor self-rated health (Nomura et al., 2005), cognitive impairment (Yaffe et al., 2014), obesity (Pearson et al., 2006), cardiovascular diseases (Sofi et al., 2014), diabetes, asthma, COPD, stomach ulcers, menstrual problems, arthritis, and migraine (Budhiraja et al., 2011).

There is a high rate of insomnia turning into a chronic disease as the sleeping disorder often perseveres even after its trigger has disappeared (Morin et al., 2015). The median duration of insomnia disorder is three years, with a persistence rate of 46% at three years

(Morin et al., 2009) and between 56% and 74% at one year (Morin et al., 2009; Morphy et al., 2007; Pillai et al., 2015). Janson et al. (2001) reported only 56% of male patients with severe insomnia symptoms going into remission over a period of 10 years (Morin et al., 2015).

Women are affected by insomnia disorder at higher rates than men (Roth, 2007). This could be explained by gonadal steroid effects, as well as the presence of certain risk factors of insomnia, such as socioeconomic factors (lower income and unemployment; Lallukka et al., 2012) and higher rates of depression and anxiety disorder (Angst et al., 2002; Asher & Aderka; 2018; Moieni et al., 2015).

Insomnia symptoms occur more frequently in older adults, which could be contributed to the decline of functionality of sleep control systems, as well as higher prevalences of medical conditions (Roth, 2007). However, insomnia is also common among adolescents, with especially high prevalence rates among older adolescents and girls between the age of 16 and 18 (Hysing et al., 2013; Johnson et al., 2006). The disorder is most often chronic and occurs in combination with other mental disorders in half of the affected adolescents (Hysing et al., 2013).

2.2.2 Pathophysiology

While the exact etiology of insomnia disorder is still unclear, psychological theorists suggest that it entails the involvement of predisposing, precipitating and perpetuating factors. They can be of genetic, behavioral, cognitive, and emotional nature and make someone vulnerable to developing the disorder, acutely cause episodes of insomnia and contribute to the perpetuation of the disease (Morin et al., 2015).

Demographic factors such as age and gender, familial history of insomnia, socioeconomic factors, physical and mental health as well as psychological traits such as sleep reactivity and higher arousal predisposition have been identified as risk factors of insomnia disorder (Drake et al., 2014; Lallukka et al., 2012; LeBlanc et al., 2009; Roth, 2007). The episodes of insomnia themselves are most often triggered by negative stressful life events that are related to family, health, work or school (Bastien et al., 2004; Morin et al., 2015).

Both predisposing and precipitating factors make patients more vulnerable to developing insomnia disorder. Perpetuating factors are however responsible for maintaining the disorder even when initial triggers are no longer present. Specific behaviors and

cognitions, as well as hyperarousal have been identified as the main factors contributing to the maintenance of insomnia disorder (Schwartz & Carney, 2012).

2.2.2.1 Behavioral influences. Sleep and wakefulness are regulated by the homeostatic system, as well as the circadian system, which is centered in the SCN (Bear et al., 2015). Specific behaviors such as daytime napping disturb the homeostatic regulation as they decrease the sleep drive, which normally builds up during the day and promotes falling asleep (Schwartz & Carney, 2012). If individuals spend a lot of time in bed, have irregular sleep and wake times, are inactive, or nap during the day to compensate for their lack of sleep, these behaviors further amplify the sleep disturbance (Morin et al., 2015; Schwartz & Carney, 2012). The circadian system is sensitive to environmental cues, such as daylight or social interactions, and regulates the sleep-wake patterns. Circadian systems can have different rhythms that influence an individuals' optimal sleep schedule. If sleep is scheduled in dissonance with the circadian system (going to sleep too early or late), sleep difficulties can be further amplified (Schwartz & Carney, 2012).

2.2.2.2 Cognitive processes. Dysfunctional thoughts such as worrying or rumination are a common occurrence in patients with insomnia disorder (Carney et al., 2010). In addition to worries about everyday issues, individuals focus their thoughts on their sleep disturbance and the consequences it might have on their everyday life and functioning (Schwartz & Carney, 2012). This increases anxiety, as patients tend to have unrealistic expectations concerning sleep duration, daytime functioning and consequences of sleep loss. They focus their attention on sleep-related stimuli, becoming overly sensitive to possible internal and external stimuli that might prevent them from falling asleep. The increased cognitive activity and arousal further prevents individuals from falling asleep (Schwartz & Carney, 2012).

2.2.2.3 Hyperarousal. The concept of hyperarousal recognizes psychological and physiological factors as perpetuating factors of insomnia and further perceives conditioned arousal as a major factor contributing to the manifestation of the sleep disturbance (Riemann et al., 2010). Research indicates that insomnia patients often experience increased physiological arousal that occurs during sleep and wakefulness and keeps individuals from falling and staying asleep (Riemann et al., 2010). The affected experience increased cognitive activity and dysfunctional thought processes such as rumination and worrying, and the associated degree of physiological arousal prevents sleep initiation (Carney et al., 2010).

Physiological hyperarousal involves an increase in hypothalamic-pituitary-adrenal axis activity, indicated by higher cortisol levels (Zhang et al., 2014), hyperarousal of the autonomic nervous system (Farina et al., 2013), increased metabolic rate (Bonnet & Arand, 1995), and body temperature (Lack et al., 2008). Due to classical conditioning, the bed and its environment gets conditioned to the physiological arousal, which then perpetuates the sleeping disorder even if the initial trigger is no longer present (Riemann et al., 2010).

2.3 Insomnia treatment

2.3.1 Cognitive Behavioral Therapy for insomnia

There is strong evidence for the effectiveness of CBTi in treating insomnia disorder in adults (Geiger-Brown et al., 2015; Trauer et al., 2015; van der Zweerde et al., 2019) and adolescents (Ma et al., 2018; Vigerland et al., 2016) with both chronic insomnia and comorbid insomnia diagnosis (Taylor & Pruiksma, 2014). CBTi is directed at factors that maintain insomnia over time, such as sleep-related dysfunctional cognitions, perpetuating behaviors, and dysregulation of the homeostatic sleep drive (Mitchell et al., 2012). CBTi usually lasts for four to eight sessions (Mitchell et al., 2012) and combines different interventions that target behavioral, cognitive, and physiological factors that are maintaining the insomnia disorder (Taylor & Pruiksma, 2014). Stimulus control, sleep restriction, relaxation and cognitive therapy have the strongest empirical evidence and are usually included in CBTi (Taylor & Pruiksma, 2014). The individual components have demonstrated significant effects in reducing insomnia severity when applied on their own as well as in combination. Psychoeducation and sleep hygiene are most often included in CBTi, although the intervention alone is considered inadequate in treating insomnia (Müller & Paterok, 2010; Taylor & Pruiksma, 2014).

2.3.1.1 Psychoeducation and sleep hygiene. Psychoeducation is an essential part of most psychological interventions. Individuals are provided with information regarding the predisposing, precipitating and perpetuating factors of insomnia disorder to gain knowledge about their disorder (Schöller & Weeß, 2020). Patients are educated about healthy sleep behaviors and environmental conditions that have a positive influence on sleep, so that their own behavioral patterns can be reflected and adapted. These sleep promoting behaviors are formulated as rules about caffeine and alcohol intake, bedroom environment and activities to avoid during the day. Psychoeducation about the sleep disorder and sleep hygiene rules form

a basis for effective treatment as they support the effectiveness of other techniques, such as sleep restriction and stimulus control (Schöller & Weeß, 2020).

2.3.1.2 Sleep restriction. Sleep restriction is a behavioral technique and restricts the time spent lying in bed so that a state of partial sleep deprivation is achieved (Schöller & Weeß, 2020). If the technique is applied long enough, the pressure for sleep should enhance and in turn lead to better sleep efficiency and sleep continuity. The time spent in bed is adjusted to the patient's subjective total sleep time. The patient cannot use all the available time in bed for sleep, which leads to the aspired sleep deficit and its effect on sleep pressure (Schöller & Weeß, 2020). The aim of this intervention is to reach a subjective sleep duration between six and eight hours. Additionally, by reducing time spent awake in bed, the association between the bed and aversive stimuli such as rumination or frustration will be extinguished (Schöller & Weeß, 2020).

2.3.1.3 Stimulus control. Stimulus control is based on behavioral and cognitive learning theories which assume that people with sleeping disorders do not associate the bed and bedroom with relaxation and sleep, but instead associate it with activities that do not conform with sleep, such as watching tv, reading, ruminating, or solving problems (Schöller & Weeß, 2020). This leads to a heightened psychophysiological activation level, enhanced activity of the waking system and therefore prevents falling asleep. Stimulus control should re-establish the association between the bedroom and relaxation, so that a reduced activation can enable patients to fall asleep. Rules of stimulus control are, that patients should only go to sleep when they feel tired, should only use the bed for sleep and sexual activities, leave the bed if they do not fall asleep within 20 minutes and only return as soon as they feel tired again (Schöller & Weeß, 2020). This procedure should be repeated as often as necessary. Patients should also get out of bed at the same time every day, regardless of how long they had slept, and should refrain from sleeping during the day (Schöller & Weeß, 2020).

Stimulus control and sleep restriction are the most important elements of CBTi, which is why building knowledge about mechanisms of sleep and sleep disorders via psychoeducation is an essential part of CBTi, so that patients are motivated to adhere to the intervention protocol (Schöller & Weeß, 2020).

2.3.1.4 Cognitive therapy.

2.3.1.4.1 Techniques targeting dysfunctional beliefs about sleep. People suffering from insomnia often develop dysfunctional beliefs about the function of their own sleep and the consequences of a lack thereof (Schöller & Weeß, 2020). These beliefs are mostly negative, unrealistic, catastrophizing, and are associated with negative emotions like anxiety, frustration and anger (Schöller & Weeß, 2020). These dysfunctional cognitions lead to increased psychophysiological arousal and can amplify the sleep disturbance. The cognitive and emotional over-engagement with one's own ability to sleep often results in monitoring behavior, which further enhances the sleep disturbance due to selective attention on sleep-disturbing stimuli (Schöller & Weeß, 2020). Cognitive therapy is important so that dysfunctional beliefs and automated thoughts can be identified, checked for adequacy, and modified accordingly. Cognitive therapy offers different techniques for modifying these dysfunctional assumptions and cognitions (Schöller & Weeß, 2020).

2.3.1.4.2 Techniques targeting rumination and worrying. Patients with insomnia disorder tend to worry and ruminate when they are in bed. These thought processes are aversive, as they focus the patient's attention on negative aspects of their life (Schöller & Weeß, 2020). Patients often postpone their worrying until they go to bed, which strengthens the association between the bedroom and these thought processes. Due to classical conditioning, the bedroom turns into a stimulus that triggers rumination and worrying. Cognitive techniques should help patients separate these aversive cognitions from the bedroom by scheduling when and where they are allowed to engage in these thought processes and offering techniques to handle aversive thoughts (Schöller & Weeß, 2020). The goal is that patients do not feel that they need to worry about their daily responsibilities when they are in bed, so that the association between the bedroom and cognitive arousal dissolves (Schöller & Weeß, 2020).

2.3.1.5 Relaxation. Using and teaching relaxation techniques is an essential part of insomnia treatment, as they enable patients to calm their own activation system to help them facilitate sleep more easily (Schöller & Weeß, 2020). These exercises often center on bodily sensations, breathing, mindfulness, and imagination. The techniques should be chosen on an individual level, as different techniques may be suited for different forms of insomnia disorder. Relaxation techniques that are often used in combination with cognitive and behavioral

elements are progressive muscle relaxation, autogenic training, meditation and imagination exercises (Schöller & Weeß, 2020).

2.3.2 Implementation of CBTi

There is strong evidence for the efficacy and effectiveness of CBTi in treating both adults and adolescents with insomnia disorder. It is recommended as first line treatment for primary insomnia since 2016 (Qaseem et al., 2016; Riemann et al., 2017), having comparable short-term efficacy to pharmacotherapy (Smith et al., 2002), and better long-term efficacy (Riemann & Perlis, 2009), whilst circumventing adverse effects of pharmacotherapy, including dependency, tolerance, and relapse when medication is discontinued (Kay-Stacey & Attarian, 2016). However, pharmacotherapy is still widely implemented in primary care despite contrary treatment guidelines (Koffel et al., 2018; Spiegelhalder et al., 2020), and benzodiazepines are frequently prescribed to older adults even though the American Geriatrics Society published a list of medications that should be avoided in patients over 65 years of age that included benzodiazepines (American Geriatrics Society, 2015; Mendelson et al., 2018).

In contrast, patients rarely receive adequate psychotherapeutic treatment, as in 2017 only 10% of adult patients with insomnia diagnosis received psychotherapeutic treatment in Germany (Spiegelhalder et al., 2020). This might be because even though CBTi is widely accepted as an effective treatment, its implementation entails various challenges. The intervention is time-consuming and practitioners themselves often are not trained to offer CBTi (Perlis & Smith, 2008). Patients would have to be referred to psychologists, psychotherapists, or sleep specialists, where access is often limited and linked to high costs. Other constricting factors such as physical impairment or the stigmatization of psychotherapy further limit access to and utilization of psychotherapy (Gum et al., 2010; Perlis & Smith, 2008). Meanwhile, the implications of the COVID-19 pandemic further demonstrate the necessity of therapy that can be accessed remotely. There is a need for adapted interventions that can be implemented more effectively within the healthcare system, and digitally delivered therapy is a promising treatment option that could enhance feasibility and thereby enable application of CBTi on a larger scale.

2.3.2.1 Digital CBTi (dCBTi). Since the advancement of web technology, new approaches on delivering CBT via the assistance of internet and digital devices have been

developed for the treatment of mental disorders (Ritterband et al., 2006). By using these technologies, many barriers that are currently keeping individuals away from receiving appropriate treatment could be dissolved and result in a wider distribution of psychotherapy. Various dCBTi interventions have been developed that differ in their implementation regarding required time involvement by a therapist, degree of automatization, costs, and scalability. Luik et al. (2017) propose that these interventions should be divided into the categories “digital CBT as support”, “guided digital CBT” and “fully automated digital CBTi”.

Digital CBTi can be used to *support therapy* that is facilitated by a therapist. Digital tools are used to deliver the therapy remotely (teletherapy; Gehrman et al., 2016), or to ease the implementation of certain aspects of the treatment, such as sleep diaries or the provision of information (Kuhn et al., 2016). Support through digital technologies can improve CBTi delivery and feasibility but does not relieve therapists from their therapeutic commitment.

Many *dCBTi* interventions use an *automated program* that digitally delivers the therapeutic content and is supported by a therapist or health professional (Kaldo et al., 2015; Thiart et al., 2013). The clinical staff usually provides feedback after each digital session via e-mail or chat and can sometimes dictate the order in which the modules are accessed. Guided dCBTi is provided by a digital program but still requires involvement of trained health professionals. The time commitment is however much lower than in conventional or digitally supported f2f CBTi.

Fully automated dCBTi programs function without any human involvement in the therapeutic process. Different applications deliver the treatment content in unique ways by using interactive elements, or by having an animated therapist deliver the intervention (Espie et al., 2012b; Ritterband et al., 2009). Sleep diaries and online questionnaires serve as sources of information, so that the treatment can automatically be adapted to fit the patient’s needs.

While digital CBTi can be useful if applied in support of conventional therapy, guided and fully automated applications allow for more access to treatment without physical presence of patients, less time involvement of and dependence on therapists and less costs compared to conventional f2f CBTi (Darden et al., 2020). These treatment options could allow for greater distribution of CBTi in general, as well as among disadvantaged groups. Besides making therapy more affordable, treatment could easily be provided in different languages as well as for people who are deaf or have hearing impairments.

However, possible disadvantages that are associated with the lack of involvement by an actual therapist should be considered as well. It is therefore essential to explore efficacy, compare effectiveness of guided CBTi and fully automated CBTi, and regard potential negative aspects of this treatment modality.

2.3.2.2 Dropout. Research on factors contributing to attrition in psychotherapeutic interventions are still inconclusive, though a combination of patient and therapist characteristics seem to be essential. Among other factors, a therapist's experience and skills, as well as the quality of the therapeutic alliance seem to be associated with patients' dropout rates (Roos & Werbart, 2013). As dCBTi functions without any direct contact to a human therapist, it is essential to consider how the lack of therapeutic alliance might impact the adherence to treatment.

Results from extensive meta-analytic reviews have identified similar dropout rates in studies assessing effectiveness of dCBTi, with an average dropout rate that ranged between 24,7% and 35,2% (Seyffert et al., 2016; Soh et al., 2020; Zachariae et al., 2016). In contrast, f2f CBTi seems to result in much higher adherence rates, with only a small fraction of participants discontinuing the intervention before completion (Alessi et al., 2016; Irwin et al., 2014; Lovato et al., 2014) though there are exceptions of studies exhibiting dropout rates similar to those of dCBTi (Ong et al., 2008).

If patients drop out of treatment prematurely, the aspired treatment effects cannot be obtained as lower treatment adherence is associated to smaller effect sizes (Zachariae et al., 2016) and resources are wasted. It is therefore essential that dropout rates between dCBTi and f2f CBTi are directly compared in systematic meta-analyses, so that results can be regarded when considering implementation of dCBTi on a larger scale.

2.3.2.3 Effectiveness of dCBTi. Since 2012 there has been an increase in studies examining the effectiveness of dCBTi, producing a large body of research that offers promising results on the efficacy of the intervention format (Luik et al., 2017). Soh and colleagues (2020) recently published an extensive meta-analysis of 33 studies, supporting results of prior reviews assessing the efficacy of dCBTi (Seyffert et al., 2016; Ye et al., 2016; Zachariae et al., 2016). The results indicate that dCBTi is an effective therapeutic option for adult patients suffering from insomnia symptoms, with a mean difference in insomnia severity index (ISI) scores of 5 points, matching previously reported effect sizes of MD = 4.3 (Seyffert et al., 2016)

and MD = 5.88 (Ye et al., 2016). Treatment effects on insomnia severity were maintained at short-term follow up (MD = -3.99) and one year after treatment (MD = -3.48). The report further suggests that dCBTi leads to improvements of insomnia symptoms that are similar even to those achieved by f2f CBTi. Even though the statistical power of this analysis was weak, the MD in ISI scores of 3.07 points was within the predefined non-inferiority interval (Soh et al., 2020). There is need for further analyses directly comparing effectiveness of dCBTi to f2f CBTi. Nevertheless, these results underline the impact dCBTi could have on the health care system by providing effective treatment to a large population that is currently not receiving adequate treatment (Spiegelhalder et al., 2020).

There is however no equivalent meta-analysis reviewing the effectiveness of dCBTi in adolescents, with the latest review including only three studies in its analysis on adolescents and young adults, where effect sizes (Cohen's *d*) ranged between of 0.17 and 1.3 (Werner-Seidler et al., 2018). Adolescents are underrepresented in dCBTi research despite similar prevalence rates of insomnia disorder (Hysing et al., 2013). Meanwhile, digital CBT seems to be an effective treatment format for adolescents across various mental and somatic conditions ($g = 1.27$ and $g = 0.49$, respectively; Vigerland et al., 2016) and its effectiveness in treating insomnia should be further examined.

Furthermore, Luik et al. (2017) highlight the importance of constantly developing and simultaneously reviewing new interventions for the deliverance of CBTi to keep up with digital advancements. As the number of studies on the effectiveness of digital CBTi is increasing, there is constant demand for papers reviewing new publications and analyzing factors contributing to effectiveness of dCBTi.

This review therefore aims at illustrating effectiveness of digital CBTi on a broad spectrum by including studies that focus on adolescents as well as older adults, studies on populations with and without clinical comorbidities, as well as recently published studies on the effectiveness of newly developed technologies. As this meta-analysis explores the effectiveness of dCBTi in patients meeting diagnostic criteria for insomnia disorder, it will add to results of the most recently published meta-analysis examining participants with insomnia symptoms (Soh et al., 2020). As there are few direct comparisons between dCBTi and f2f CBTi, this review will compare the effectiveness of these treatment modalities in a second analysis to find out whether dCBTi yields results of similar size than those achieved by f2f CBTi.

Furthermore, this meta-analysis will explore whether participants are more likely to drop out of dCBTi than f2f CBTi, as dropout is considered a serious adverse event in psychotherapeutic treatment.

3. Research objectives and hypotheses

The primary objective of this meta-analysis is to assess the effectiveness of dCBTi in adolescents and adults suffering from insomnia disorder compared to control conditions, as well as compared to traditional f2f CBTi. The second objective is the assessment of the relationship between the size of the intervention effect and the age of the population, type of intervention, control group, and study quality by examining whether these factors are associated with the intervention effect. By this, factors possibly explaining heterogeneity in intervention effects are explored. The third objective of this analysis is to explore whether patients are more likely to drop out of treatment prematurely when they are assigned to dCBTi compared to f2f CBTi.

The research questions and matching hypotheses of this systematic review with meta-analysis are as follows:

Research question 1: Is dCBTi effective in reducing insomnia severity in people with insomnia at post-assessment?

Past research has shown that dCBTi produces significant results in the reduction of insomnia severity in individuals with insomnia symptoms (Seyffert et al., 2016; Soh et al., 2020; Ye et al., 2016; Zachariae et al., 2016). Accordingly, the following hypotheses were formulated for the current meta-analysis:

H0: The overall standardized mean difference (SMD) of insomnia severity between the intervention group and the control groups at the post-assessment is non-significant: $p \geq .05$.

H1: dCBTi improves insomnia severity compared to the control conditions, resulting in SMD in favor of dCBTi at post assessment: $g > 0$ and $p < .05$.

Research question 2: Is dCBTi effective in reducing insomnia severity at follow-up-assessment?

The effects of dCBTi in reducing insomnia symptoms seem to be sustained after the treatment has ended. Ye et al. (2016) report significant improvements in sleep efficiency, total

sleep time, sleep onset latency, and wake after sleep onset at short-term follow up (2 to <6 months), and effects on insomnia severity were recorded up to one year after treatment (Soh et al., 2020).

H0: The overall SMD of insomnia severity between the intervention group and the control groups at the 1-6 months and > 6 months follow-assessment is non-significant: $p \geq .05$.

H1: dCBTi improves insomnia severity compared to the control conditions, resulting in SMD in favor of dCBTi at 1-6 months and > 6 months follow-up assessment: $g > 0$ and $p < .05$.

Research question 3: Does dCBTi reduce insomnia severity, measured via insomnia severity index, to a clinically significant degree of ≥ 6 points at post-assessment?

Previous meta-analyses identified a mean difference in ISI scores that ranged between 4.3 and 5.88 points (Seyffert et al., 2016; Soh et al., 2020; Ye et al., 2016). The authors conclude that these results indicate sufficient treatment success. However, Yang et al. (2009) recommend that the ISI value representing clinically meaningful change of insomnia severity should be set at six points. The hypotheses were therefore formulated in line with these recommendations:

H0: The overall unstandardized mean difference (MD) of ISI between intervention and control group at post-assessment is < 6 .

H1: The overall unstandardized MD of ISI between intervention and control group at post-assessment is ≥ 6 .

Research question 4: Does dCBTi reduce insomnia severity, measured via insomnia severity index, to a clinically significant degree of ≥ 6 points at follow-up-assessment?

dCBTi has demonstrated significant short-term effects in improving sleep efficiency, total sleep time, sleep onset latency, wake after sleep onset, and insomnia severity (Ye et al., 2016). Insomnia severity was also improved at the long-term follow-up assessment (Soh et al., 2020). The mean differences were however below 6 points and even slightly below the 4 points which can also be seen as indicative for treatment success (Yang et al., 2009).

H0: The overall unstandardized MD of ISI between intervention and control group at 1-6 months and > 6 months follow-up-assessment is < 6 .

H1: The overall unstandardized MD of ISI between intervention and control group at 1-6 months and > 6 months follow-up-assessment is ≥ 6 .

Research question 5: Is the overall mean effect moderated by the age of the participants when comparing dCBTi to control groups at post-assessment?

Studies assessing effectiveness of dCBTi in adolescents and young adults yielded effect sizes ranging between 0.17 and 1.3 (Werner-Seidler et al., 2018), while Zachariae et al. (2016) estimated the effect of dCBTi on insomnia severity in an adult population at 1.03. This analysis tries to assess whether the age of participants is associated with the efficacy of dCBTi in treating insomnia.

H0: The overall SMD between dCBTi and control groups is not moderated by mean age of the participants included in the studies ($p \geq .05$).

H1: The overall SMD between dCBTi and control groups is moderated by mean age of the participants included in the studies ($p < .05$).

Research question 6: Is the overall mean effect for studies using fully automatized dCBTi different from the effect for studies using guided dCBTi?

Even though guided dCBTi substantially reduces the necessary time involvement by a therapist or health professional, fully automated dCBTi functions without any human involvement (Luik et al., 2017). The analysis explores whether fully automatized interventions can produce results similar to dCBTi that is guided and supervised by a health professional.

H0: The SMD for studies using fully automatized dCBTi is not different from the SMD for studies using guided dCBTi: $\mu_1 = \mu_2$ or $p \geq .05$

H1: The SMD for studies using fully automatized dCBTi is different from the SMD for studies using guided dCBTi: $\mu_1 \neq \mu_2$ and $p < .05$.

Research question 7: Is the overall mean effect for studies using placebo control groups (ineffective digital intervention) different from the effect for studies using passive control groups (treatment as usual, wait-list)?

Studies using passive control groups are at higher risk of bias than studies using placebo control groups (Sterne et al., 2019). By comparing results of studies using different

comparisons, this analysis assesses whether participants knowledge on treatment allocation might have influenced the size of the treatment effect.

H0: The SMD for studies using placebo control groups is not different from the SMD for studies using passive controls: $\mu_1 = \mu_2$ or $p \geq .05$.

H1: The SMD for studies using placebo control groups is different from the SMD for studies using passive control groups: $\mu_1 \neq \mu_2$ and $p < .05$.

Research question 8: Is dCBTi as effective in reducing insomnia severity as f2f CBTi at post-assessment?

Soh and colleagues (2020) offered results which indicate that dCBTi leads to improvements in insomnia severity that are not inferior to those of f2f CBTi, assessing a MD of 3.07 points on the ISI score. Therefore, the following hypotheses were formulated:

H0: There is no significant difference in the rate of improvement (SMD) of insomnia severity at the post-intervention assessment between dCBTi and f2f CBTi: $g=0$ or $p \geq .05$.

H1: There is a significant difference in the rate of improvement (SMD) of insomnia severity at the post-intervention assessment between dCBTi and f2f CBTi, favoring f2f-CBTi: $g > 0$ and $p < .05$.

Research question 9: Is dCBTi as effective in reducing insomnia severity as f2f CBTi at follow-up-assessment?

Treatment effects of dCBTi on insomnia severity could be maintained at short-term follow-up assessments (Soh et al., 2020). It remains unclear, whether the long-term effects of dCBTi are comparable to those of f2f CBTi.

H0: There is no significant difference in the rate of improvement of insomnia severity at the 1-6 months and > 6 months follow-up assessment between dCBTi and f2f CBTi: $g=0$ or $p \geq .05$.

H1: There is a significant difference in the rate of improvement of insomnia severity at the 1-6 months and > 6 months follow-up-assessment between dCBTi and f2f CBTi, favoring f2f-CBTi: $g > 0$ and $p < .05$.

Research question 10: Does the mean difference of ISI between dCBTi and f2f CBTi at post-assessment lie within the non-inferiority margin of 4 points?

Soh et al. (2020) reported a MD of 3.07 between dCBTi and f2f CBTi at follow-up assessment, which lies within the predefined non-inferiority margin of four points, which is based on results by Yang et al. (2009). In this meta-analysis, the same criteria for non-inferiority were applied.

H0: The overall unstandardized MD of ISI between dCBTi and f2f CBTi at post-assessment is less than or exactly 4 points or non-significant, indicating non-inferiority of dCBTi.

H1: The overall unstandardized MD of ISI between dCBTi and f2f CBTi at post-assessment is larger than 4 points, favoring f2f CBTi.

Research question 11: Does the mean difference of ISI between dCBTi and f2f CBTi at follow-up-assessment lie within the non-inferiority margin of 4 points?

While Soh et al. (2020) found that the MD of dCBTi and f2f CBTi at post-assessment lies within the non-inferiority margin of 4 points, it remains unclear whether this effect can be maintained until follow-up assessment. The following hypotheses were therefore formulated:

H0: The overall unstandardized MD of ISI between dCBTi and f2f CBTi at 1-6 months and > 6 months follow-up-assessment is less than or exactly 4 points, indicating non-inferiority of dCBTi.

H1: The overall unstandardized MD of ISI between dCBTi and f2f CBTi at 1-6 months and > 6 months follow-up-assessment is larger than 4 points, favoring f2f CBTi.

Research question 12: Is the overall mean effect moderated by the age of the participants when comparing dCBTi and f2f CBTi at post-assessment?

Past research has oftentimes disregarded the influence of age on the size of the treatment effects. This analysis will include the factor age as a potential influence.

H0: The overall SMD between dCBTi and f2f CBTi is not moderated by mean age of the participants included in the studies ($p \geq .05$).

H1: The overall SMD between dCBTi and f2f CBTi is moderated by mean age of the participants included in the studies ($p < .05$).

Research question 13: Are participants more likely to drop out of the intervention if they are placed in dCBTi compared to f2f CBTi?

Dropout rates in studies assessing effectiveness of dCBTi tend to be much higher than those in f2f CBTi, ranging between 24,7% and 35,2% (Seyffert et al., 2016; Soh et al., 2020; Zachariae et al., 2016), while attrition seems to be very low in f2f CBTi (Alessi et al., 2016; Irwin et al., 2014; Lovato et al., 2014), even though there are exceptions where similarly high dropout rates are reported (Ong et al., 2008).

H0: Participants are not more likely to drop out of the intervention when they are placed in dCBTi compared to f2f CBTi: Odds Ratio = 0 or $p \geq .05$.

H1: Participants are more likely to drop out of the intervention when they are placed in dCBTi compared to f2f CBTi: Odds Ratio $\neq 0$ and $p < .05$.

4. Method

To gather studies for the current meta-analysis, databases were systematically searched for literature. Studies comparing dCBTi to control conditions in adolescents and adults with insomnia disorder were eligible for inclusion. Primary outcome was insomnia severity at post intervention, short-term follow-up (ranging 1-6 months) and long-term follow-up (> 6 months). Data was extracted from each study regarding study characteristics, sample characteristics, intervention characteristics, and outcome data. Risk of bias of the included studies was assessed in line with the revised Cochrane risk of bias tool for randomized trials (Sterne et al., 2019).

Mean differences of individual studies were assessed using the standardized effect measure Hedges' g and pooled to generate an overall effect size using the random effects model. Unstandardized mean difference of ISI scores was calculated for studies reporting the effect measure. Subgroup analyses were employed to explain heterogeneity in intervention effects and a moderator analysis was conducted to explore the influence of the factor age. An analogous analysis was performed to compare dCBTi to f2f CBTi. Odds ratio (OR) was calculated to compare odds for dropout between dCBTi and f2f CBTi. Statistical analyses were conducted via the system *Meta-Essentials* (Suurmond et al., 2017).

The following section will elaborate on the individual steps of the meta-analytic process.

4.1 Setting eligibility criteria

Eligibility criteria for the inclusion of studies were defined based on population, intervention, control condition and outcome of the study (PICO elements), as well as the study characteristics. These criteria were defined based on conceptual consideration and chosen with precision, so that decisions on inclusion could be made with low risk of bias.

4.1.1 Population

Participants had to be at least 10 years old and had to be either formally diagnosed with insomnia or report insomnia symptoms in line with standardized diagnostic criteria. The accepted criteria were based on the definitions of either the DSM-IV, the DSM-5 (American Psychiatric Association, 1994, 2013), the ICD-10, the ICD-11 (World Health Organization, 2016, 2019), the ICSD-2 or the ICSD-3 (American Academy of Sleep Medicine, 2005, 2014). Patients with comorbid mental and medical diagnoses were also eligible for inclusion.

4.1.2 Intervention

The intervention had to be multimodal dCBTi and include at least one cognitive strategy, one key behavioral strategy (stimulus control or sleep restriction) and consist of at least four elements in total. The intervention period had to last for a minimum of four weeks and sessions, as four sessions seem to be sufficient to produce durable treatment response (Edinger et al., 2007). The intervention had to be delivered digitally, either with guidance by trained staff or via a fully automated program. dCBTi was defined and categorized as follows: *guided dCBTi* combines an automated program with support by clinical staff. The content of the intervention is provided digitally across a digital modality and complemented by therapeutic feedback. The provider may also have influence on the order of the digitally delivered content. *Fully automated dCBTi* functions without any human assistance in the implementation of the program. Sleep diaries and questionnaires offer information to help the program alter the intervention to the patients' needs (Luik et al., 2017).

4.1.3 Comparator

Studies comparing the effectiveness of dCBTi to either controls with less effective or "placebo" components (e.g. psychoeducational websites), to passive controls (wait-list

condition, treatment as usual) or to f2f CBTi were eligible for inclusion, to allow for an analysis of the effectiveness of dCBTi in general and an analysis comparing dCBTi to the standard treatment (f2f CBTi).

4.1.4 Outcome

Studies had to report the primary outcome insomnia severity, measured via insomnia severity index (ISI) or an alternative validated diagnostic questionnaire. The study had to report results in a way that could be used for the meta-analysis. These included either the effect of interest itself (SMD), or the number of participants, post-intervention/ follow-up means of each group and either standard deviation (SD), standard error (SE), the 95% confidence interval (CI) or interquartile ranges with symmetrical distribution. The assessment points are post-intervention, short-term follow up, which is between one and six months, or long-term follow-up, which is defined as more than six months after treatment. The study had to report the necessary outcome data at any of the assessment points.

4.1.5 Study characteristics

Because of the large body of research, only studies that were randomized controlled trials (RCTs) were eligible for inclusion to produce the most valid results. Studies that were published and studies that were unpublished were eligible to reduce the risk of bias due to selective publication of significant results (publication bias). Studies from 2015 until March 2021 were eligible to include most recent results and complement the meta-analytic results of previous meta-analyses (Seyffert et al., 2016; Ye et al., 2016; Zachariae et al., 2016). There were no limitations to the country in which the study was conducted and published, however, due to limited resources for the translation of studies, only studies written in English and German language could be tested for eligibility and included in the meta-analysis.

4.2 Assessment of treatment outcomes

Data from patient reported diagnostic questionnaires were used as outcome measures for the assessment of insomnia severity. As the measure is well validated, sufficiently reliable, and commonly used in trials, the insomnia severity index (ISI) score was the preferred outcome and therefore used for the calculation of Hedges' g if studies provided the data. If studies did not report ISI scores, results from Pittsburgh Sleep Quality Index (PSQI) were chosen as the primary outcome. If a study reported neither ISI nor PSQI scores but included

alternative validated and sufficiently reliable measures, those were used for the calculation of effect estimates. If data from more than one diagnostic questionnaire were reported and the superiority of one measure was unclear to the researcher, data from the available diagnostic questionnaires were combined into one value.

4.2.1 *Insomnia Severity Index*

The Insomnia Severity Index (Morin, 1993) is a reliable and valid instrument that can detect insomnia cases in the general population and suitably registers treatment responses in a clinical sample (Cronbach $\alpha = .91$; Morin et al., 2011). ISI measures the severity of insomnia and includes symptoms affecting individuals during the day and during the night. The measure consists of seven items that investigate sleep difficulties, degree of interference with daytime functioning and degree of experienced distress over the past month (Morin et al., 2015). The questions are each rated on a 5-point scale and the overall score can range between 0 and 28 points, with more points indicating more severe insomnia. A total score of 0-7 indicates no clinical insomnia, a score from 8-14 is considered subthreshold insomnia, 15-21 points suggest insomnia of moderate severity and a result of 22-28 indicates severe insomnia (Morin et al., 2015).

There are different approaches to defining an ISI value that represents clinically meaningful improvement. Yang et al. (2009) recommend that the value should be set at a 6-point reduction, which was used as a guideline for this meta-analysis. However, a reduction of more than 4 points has been shown to already reduce the risk for negative treatment outcomes, increase vitality and decrease fatigue (Yang et al., 2009).

4.2.2 *Pittsburgh Sleep Quality Index*

The Pittsburgh Sleep Quality Index (Buysse et al., 1989) is considered a valid and reliable measure (test-retest correlation $r = .87$, Cronbach $\alpha = .84$; Backhaus et al., 2002; Sohn et al., 2011) for sleep quality and disturbances in clinical populations. The global score is made up of seven component scores that are generated by 19 questions using a 4-point scale and assess sleep quality over a one-month period. The domains are subjective sleep quality, sleep duration, sleep latency, sleep disturbances, habitual sleep efficiency, use of sleeping medication and daytime dysfunction. The global score ranges between 0 and 21, and a value of over 5 points is considered a significant sleep disturbance (Buysse et al., 1989).

4.2.3 Sleep Condition Indicator

The Sleep Condition Indicator (SCI; Espie et al., 2014) is considered an eligible screening questionnaire for assessing insomnia symptoms against DSM-5 criteria, has sufficient internal consistency (Cronbach $\alpha = .86$) and demonstrated convergent validity with the PSQI and ISI (Espie et al., 2014). The measure consists of eight items that are rated on a 5-point scale, assessing concerns about getting to sleep, remaining asleep, sleep quality, daytime performance, daytime personal functioning, nights per week having sleep problems, duration of sleep problems and extent troubled by poor sleep. The score ranges between 0 and 32 points, with higher scores representing better sleep (Espie et al., 2014).

4.2.4 Holland Sleep Disorder Questionnaire

The Holland Sleep Disorder Questionnaire (HSDQ; Kerkhof et al., 2012) is an accurate self-assessment questionnaire with sufficient reliability (Cronbach $\alpha = .90$) and is based on diagnostic criteria for sleep disorders by the ICSD-2 classification system (American Academy of Sleep Medicine, 2005). The questionnaire includes 32 items and screens for sleep disorders in six categories. The subscale for the assessment of insomnia symptoms (HSDQi) includes eight items that are rated on a 5-point scale, with a score above 3.68 indicating insomnia. The subscale has a Cronbach alpha of .88 (van Maanen et al., 2014).

4.2.5 Chronic Sleep Reduction Questionnaire

The Chronic Sleep Reduction Questionnaire (CSRQ; Dewald et al., 2012) assesses symptoms of chronic sleep reduction in the past two weeks, is validated and has sufficient reliability for adolescent populations (Cronbach alpha = .84 – .87). The questionnaire includes 20 items that are rated on a 3-point scale and consists of four subscales: shortage of sleep, loss of energy, irritation, and sleepiness. Higher scores indicate more symptom severity (Dewald et al., 2012).

4.3 Searching for and selecting studies

PubMed, Web of Science, PsycINFO, Cochrane Library, Google Scholar, as well as databases for grey literature were systematically searched for literature to find eligible RCTs to include in the meta-analysis. The following keywords were used when searching the databases: *insomnia, sleep disorder, sleep disturbance, sleep problem, internet, digital, website, web, online, computer, mobile, self-help, self-administer, self-care, self-instruct, self-*

*management, cognitive therapy, behavioral therapy, behavioural therapy, cognitive-behavioral therapy, cognitive-behavioural therapy, CBT, CBTi, CBT-I, dCBT, I-CBT, I-CBTi, I-CBT-I, dCBTi, dCBT-I, adults, elderly, adolescents, youth, old**. The keywords were combined using “boolean operations” to enable an advanced literature search.

To consider all potentially eligible studies, the terms included in the literature search covered a wide scope that resulted in numerous outcomes but had low precision. Literature cited in studies and reviews that were identified were examined (“footnote-chasing”) and databases for grey literature were searched to reduce potential bias due to the overrepresentation of studies with significant results compared to non-significant results (publication bias). After the removal of duplicates, the titles and abstracts were screened for inclusion and reports that were obviously irrelevant were excluded. If there were multiple reports of the same study, these reports were connected. The full texts of the remaining reports were reviewed regarding their compliance with the eligibility criteria. Reasons for exclusion were marked and studies that met the eligibility criteria were chosen to be included in the meta-analysis.

4.4 Coding and data extraction

Data was extracted and coded from each study regarding study characteristics, sample characteristics, intervention characteristics and outcome data. The following features were coded: *Study characteristics* - name of authors; country in which the study was conducted; language of the paper; type of publication; year of publication; control condition; dropout rate per group (in percent); attrition rate per group (in percent); study quality (measured via risk of bias rating), *sample characteristics* - sample size (overall and per group); definition of the population; classification system regarded for insomnia diagnosis; presence of comorbid disorder; percentage females; mean age per group, *intervention characteristics* - degree of automatization (guided vs. fully automated); components included in the intervention; duration of the intervention in weeks, and *outcome data* - measured construct; instrument; scale-characteristics; sample size (per group and time point); time of measurement; post-assessment and follow-up mean scores per group; standard deviation/ standard error/ 95% CI per group; SMD. The data was coded by the researcher.

4.5 Categorization

After the coding process was completed, study characteristics of each study were categorized and illustrated in tables to demonstrate similarities and differences between studies.

For subgroup analyses, control condition was categorized into active placebo control, passive control and f2f CBTi, and dCBTi was categorized into fully automatized and guided interventions. For the calculation of OR, dropout needed to be defined. This was dependent on conceptual considerations as well as data provided by the individual studies. If studies offered the according data, drop out was defined as participants completing less than four sessions. This is in line with the criteria set for minimum treatment duration. If there were no data available for that value, studies reporting drop out as less than three sessions completed were also accepted and included in the analysis.

4.6 Measuring study quality by risk of bias rating

Risk of bias of the included studies was assessed in line with the revised Cochrane risk of bias tool for randomized trials (ROB 2; Sterne et al., 2019).

The tool entails five domains that cover different forms of bias that affect the results of randomized trials according to the current state of research (Sterne et al., 2019). These are “risk of bias arising from the randomization process”, “risk of bias due to deviations from the intended interventions (effect of assignment to intervention)”, “risk of bias due to missing outcome data”, “risk of bias in measurement of the outcome” and “risk of bias in selection of the reported result” (Sterne et al., 2019). Questions concerning each domain must be answered so that the algorithm can calculate risk of bias for each domain as well as overall risk of bias. For an overall low risk-of-bias judgement to be made, the study has to be judged at low risk of bias for all domains (Sterne et al., 2019). If the study raises some concerns in at least one domain but is not of high risk for any domain, the overall judgement is “some concerns” (Sterne et al., 2019). For the study to be considered of overall high risk of bias, it must “be at high risk of bias in at least one domain ... or ... judged to have some concerns for multiple domains in a way that substantially lowers confidence in the result” (Higgins et al., 2021b: chapter 8.2.4).

4.7 Effect size calculation & statistical method

4.7.1 Specifying the effect of interest and choosing a model of analysis

4.7.1.1 Standardized mean difference. The primary effect of interest was SMD at post-intervention between the intervention group and a control group. SMD represents the effect size of each study relative to the variability of outcome measurements between participants in that study (Higgins et al., 2021a). The SMD is calculated using Equation 1 (Higgins et al., 2021a):

$$\text{SMD} = \frac{\text{difference in mean outcome between groups}}{\text{standard deviation of outcome among participants}} \quad (1)$$

This measure was used because it is suitable when all studies assess the same outcome but use different ways to measure it. All included studies measure insomnia severity via diagnostic questionnaires (among other measures), however, not all studies use the same instrument. Calculating the SMD of each study standardizes the results to a uniform scale, which enables the integration of all eligible studies in the analysis (Higgins et al., 2021a). When calculating the SMD for the individual studies, it is essential to consider the direction of the scale and adapt the effect size accordingly. If that was the case, the concerning studies were conversed by multiplying the mean value by the factor -1 (Higgins et al., 2021a).

If studies did not disclose scores to calculate the post-assessment SMD but solely reported the post-assessment SMD adjusted to baseline values, this score was included in the analysis. A problem with this approach is, that there is a possibility for authors to selectively report the adjusted scores instead of post-intervention values alone because they yield more exaggerated results (Higgins et al., 2021a). A sensitivity analysis regarding studies reporting different outcome measures was conducted to attend to this issue (Higgins et al., 2021c).

4.7.1.2 Intention-to-treat vs. per-protocol. The effect of interest was an “intention-to-treat effect” (ITT), which is that of assignment to the intervention at baseline. This means that all randomized participants are considered in the analysis, regardless of whether the intervention was received as it was intended. Conducting an ITT analysis might require researchers to calculate outcome scores for all participants’ outcomes based on modern statistical methods, so that results are of lower risk of bias. It is standard procedure for clinical trials to consider the intention-to-treat effect, as the estimated effect of assignment to intervention would be most informative for considering whether an intervention can be implemented in a particular health care system (Higgins et al., 2021b). In contrast, the “per-

protocol effect” (PP) is the effect of adhering to the intervention in line with the trial protocol. Participants that drop out of the intervention or do not offer data at post-assessment are excluded from the PP analysis. This form of analysis produces some complications, as prognostic factors may influence adherence to the assigned intervention. The effect of interest was therefore chosen according to the following order of preference, in line with the recommendations of Cochrane (Higgins et al., 2021b):

1. the result corresponding to a full ITT analysis ...
2. the result corresponding to an analysis (sometimes described as a ‘modified intention-to-treat’ (mITT) analysis) that adheres to ITT principles except that participants with missing outcome data are excluded ...
3. a result corresponding to an ‘as-treated’ or naïve ‘per-protocol’ analysis, or an analysis from which eligible trial participants were excluded. (Higgins et al., 2021b: chapter 8.2.2)

4.7.1.3 Random effects model. When conducting a meta-analysis, researchers need to consider which model their analysis should be based on. In the *random effects model*, the effect size of the population is represented by a central tendency of the distribution of effect sizes (μ) and the divergence of a study from the central tendency (τ). Confidence intervals do not necessarily overlap with one effect size of a population, but with a distribution of effect sizes (see Figure 1). This assumption stands in contrast to that of the fixed effect model, which assumes that the effect size measures the true value of the population (θ) and diverges from that score simply due to sampling error (Eisend, 2020; Higgins et al., 2021c).

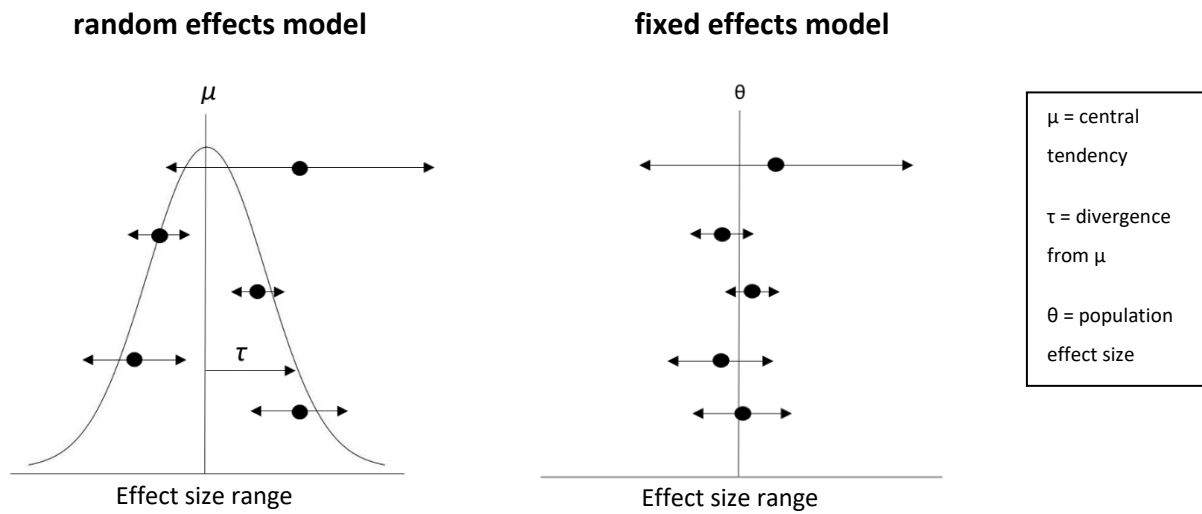
According to the random effects model, the effect size varies due to sampling error (V_i) as well as differences between studies (V_θ). When calculating all statistical values using random effects model, the Variance V_i^* is used. The Variance (V_i^*) of each study is therefore calculated as demonstrated in Equation 2 (Eisend, 2020):

$$V_i^* = V_\theta + V_i \quad (2)$$

In the random effects model, the studies included in the meta-analysis represent a random sample of true effect sizes. For most meta-analyses, the random effects model is much more realistic than the fixed effect model, which is why this model is predominantly used in meta-analytic papers (Higgins et al., 2021c). The random effects model assumes that the true effect size might vary depending on the study. In this meta-analysis, studies differ in factors such as study population or implementation of interventions, and the effect size might

Figure 1

Illustration of random effects model and fixed effects model



Note. Distribution of effects from different studies. Adapted from *Applied meta-analysis for social science research* (p. 231), by N. A. Card, 2011, The Guilford Press. Copyright 2012 by The Guilford Press.

be different among these conditions. Therefore, random effects model was chosen for this meta-analysis.

4.7.2 Calculating the effect measures of individual studies

4.7.2.1 Calculating Hedges' g. To calculate the overall standardized mean difference (SMD) at post-intervention between intervention group and control group, effect measures of individual studies need to be unified. Hedges' g was used for effect size calculation, as it is the SMD corrected for bias due to small sample sizes (Higgins et al., 2021a).

Hedge's g is calculated from Cohen's d using the correction value J (Borenstein et al., 2009) as seen in equations 3 to 7 (Borenstein et al., 2009).

$$J = 1 - \frac{3}{4df - 1} \quad (3)$$

$$df = n_1 + n_2 - 2 \quad (4)$$

$$g = J * df * d \quad (5)$$

$$SE_g^2 = J^2 * SE_d^2 \quad (6)$$

$$SE_g = \sqrt{SE_g^2} \quad (7)$$

Cohen's d is based on the standardized difference between the two group means ($\bar{Y}_1, \bar{Y}_2$) as well as the sum of squared errors (Field & Gillett, 2010) and calculated using Equations 8 and 9 (Borenstein et al., 2009; Lipsey & Wilson, 2001).

$$d = \frac{\bar{Y}_1 - \bar{Y}_2}{S_{pooled}} \quad (8)$$

$$S_{pooled} = \sqrt{\frac{(n_1 - 1)S_1^2 + (n_2 - 1)S_2^2}{n_1 + n_2 - 2}} \quad (9)$$

S_1^2, S_2^2 = variance in groups 1 and 2; n_1, n_2 = sample size in groups 1 and 2

4.7.2.2 Transforming effect measures. If the standard deviation is not stated in a study, it can be calculated using other statistical measures stated in the paper.

SD can be calculated from the standard error within a single intervention group and the sample size (see Equation 10; Higgins et al., 2021a):

$$SD = SE \sqrt{N} \quad (10)$$

SD can also be calculated from the confidence interval for a mean value within a group and the sample size. If the 95% CI is stated and the sample size is considered large (larger than 100 participants in each group; Higgins et al., 2021a), the SD is calculated with Equation 11 (Higgins et al., 2021a):

$$\frac{\sqrt{N} * (CI \text{ upper limit} - CI \text{ lower limit})}{3.92} \quad (11)$$

The denominator is replaced with 3.29 for 90% confidence intervals and with 5.15 for 99% confidence intervals. When the study consists of a small sample of less than 60 people per group, the denominator is replaced with an adapted value specific to the t distribution. This number can be found in the tables of t distribution or calculated in a Microsoft Excel spreadsheet by using the formula " $tinv=(1-0.95, N-1)$ " (Higgins et al., 2021a).

Cohen's d and therefore Hedges' g can be calculated from F values using Equation 12 (Card, 2011):

$$d = \pm \sqrt{\frac{F(n_1 + n_2)}{n_1 n_2}} \quad (12)$$

Cohen's d can be estimated using the unstandardized β (Baguley, 2011). Equations 13 and 14 transform the value and offer a rough approximation of Cohen's d (Feingold, 2015):

$$SD = \sqrt{N} * SE_{\beta} \quad (13)$$

$$d = \frac{\beta}{SD} \quad (14)$$

4.7.2.3 Combining groups. If studies report results from two subgroups separately that are summarized as one group in the meta-analysis, these subgroups can be combined to a single group. Equations 15 to 17 were used to calculate summary statistics across two subgroups that make up one group in the meta-analysis (Higgins et al., 2021a):

$$N = N_1 + N_2 \quad (15)$$

$$M = \frac{N_1 M_1 + N_2 M_2}{N_1 + N_2} \quad (16)$$

$$SD = \sqrt{\frac{(N_1 - 1)SD_1^2 + (N_2 - 1)SD_2^2 + \frac{N_1 N_2}{N_1 + N_2} (M_1^2 + M_2^2 - 2M_1 M_2)}{N_1 + N_2 - 1}} \quad (17)$$

The formulae combine numbers for the outcome measurements (sample size, mean and SD) as if the combined group had never been divided in two. This way of calculating the SMD is used instead of using the pooled SD, as the pooled SD underestimates the desired SD by providing a SD within the subgroup instead of a SD for the combined groups (Higgins et al., 2021a).

4.7.2.4 Missing data. Data that is missing at random is rather unproblematic, and an analysis of the available data might even be unbiased, although of smaller sample size (Higgins et al., 2021c). If the reason for data to be missing is related to the missing data itself, data is not missing at random. In interventions targeting populations with mental disorders, participant data might often not be missing at random, as patients experiencing more symptoms might be less likely to complete the trial (Ong et al., 2008). An analysis that is based on the available data alone is therefore typically biased (Higgins et al., 2021c). Attrition of participants within a trial often leads to data that are not missing at random, and studies that

do not use statistical methods to compensate for missing participant data accordingly might therefore introduce bias in the analysis (Higgins et al., 2021c). Sensitivity analyses were performed to assess how sensitive the results of the analysis are to the exclusion of studies that do not use statistical methods to account for missing data due to attrition or drop out.

The sensitivity analysis targets the question as to whether decisions made in the process of obtaining data influence the magnitude of the effect. If the effect and conclusions are unaffected by the changes, the results can be accepted with more certainty (Higgins et al., 2021e).

4.7.3 Checking data

Before conducting the synthesis, the data was checked to make sure that it was coded and transcribed accurately. There was however no second coder independently transferring the data.

4.7.4 Data analysis

Data was inscribed and statistical analyses performed in the spreadsheets provided by *Meta-Essentials* (Suurmond et al., 2017). The main analyses (Pooled Hedges' g at post-assessment and follow-up assessment) were conducted using the spreadsheet for differences between independent groups and continuous data, the MD of ISI score was calculated using the sheet for effect size data and OR was calculated using the spreadsheet for differences between independent groups and binary data. Pooled Hedges' g between groups and mean difference of ISI score between groups were calculated for studies comparing dCBTi to controls, as well as for studies comparing dCBTi to f2f CBTi at post and follow-up assessment. Subgroup analyses were conducted for the factors control group and degree of automatization of the intervention. A moderator analysis was calculated to see if the size of the intervention effect is influenced by the mean age of participants. Sensitivity analyses were executed to check for the robustness of effect if certain studies are removed or included in the analysis. I^2 statistics were used to test for heterogeneity (Borenstein et al., 2009; Higgins et al., 2021c). The publication bias was assessed via interpretation of a funnel plot as well as Egger's regression (Egger et al., 1997). OR was calculated via a fourfold table to assess whether participants are more likely to drop out of dCBTi than f2f CBTi.

4.7.4.1 Pooled SMD. To estimate the effect size under the random effects model, heterogeneity of the effect sizes and variability of the population is determined, random effects weights of studies effect sizes calculated and used to estimate pooled SMD and its standard deviation (Eisend, 2020).

Hedges Q measures the probability for heterogenous events (not the degree of heterogeneity), and is calculated using Equation 18 (Borenstein et al., 2009):

$$Q = \sum(w_i(ES_i - \overline{ES})^2) \quad (18)$$

The variability of the population is calculated with Equation 19 (Borenstein et al., 2009):

$$\tau^2 = \frac{Q - (k - 1)}{(\sum w_i) - \frac{(\sum w_i^2)}{(\sum w_i)}} \quad (19)$$

The random effects weights are calculated using the variability of the population (see Equation 20; Borenstein et al., 2009):

$$w_i^* = \frac{1}{\tau^2 + SE_i^2} \quad (20)$$

The weighed pooled SMD and standard deviation are calculated using Equations 21 and 22 (Borenstein et al., 2009):

$$\overline{ES} = \frac{\sum(w_i ES_i)}{\sum w_i} \quad (21)$$

$$SE_{\overline{ES}} = \sqrt{\frac{1}{\sum w_i}} \quad (22)$$

When interpreting the magnitude of effect sizes, Cohen (1988, 1992) suggests considering effect sizes above .30 and below .50 as small, effect sizes between .50 and .79 as moderate, and effect sizes of .80 or higher as large effects (Field & Gillett, 2010).

4.7.4.2 Heterogeneity: I². As clinical and methodological differences commonly occur in meta-analyses, statistical heterogeneity cannot be avoided (Higgins et al., 2021c). The diversity across studies and its influence on the meta-analysis can be quantified using statistical methods. I² is an estimate of the percentage of the variability between trials that is

due to true heterogeneity (instead of sampling error). It is calculated as seen in Equation 23 (Borenstein et al., 2009):

$$I^2 = \left(\frac{Q - df}{Q} \right) * 100\% \quad (23)$$

Note: Q = Chi² statistic; df= degrees of freedom

Cochrane gives a rough guide as to how the I^2 values can be interpreted in meta-analyses of RCTs (Higgins et al., 2021c). A I^2 value of 0-40% might be unimportant, 30-60% may be considered as moderate heterogeneity, 50-90% means substantial heterogeneity and 75-100% represent considerable heterogeneity. When interpreting these values, the strength of the evidence for the rating (e.g. p-value), as well as the magnitude and the direction of the effect should be considered by the researcher (Higgins et al., 2021c).

In this meta-analysis, heterogeneity was calculated directly in the Meta-Essentials (Suurmond et al., 2017) spread sheet. the data.

4.7.4.3 Odds ratio. Odds ratio measures the connection between two properties in a population, indicating whether the presence or absence of one property influences the presence or absence of the other (Field & Gillett, 2010). OR can be calculated to examine whether a particular condition increases the risk for a certain outcome, and to compare risk factors for the specific outcome (Field & Gillett, 2010).

OR is calculated using a fourfold table, with each chamber holding the number of participants matching a certain condition (see Figure 2).

Figure 2

Fourfold table for calculation of the odds ratio

	Y=1	Y=0
X=1	a	b
X=0	c	d

The two conditions in Figure 2 are allocation to dCBTi ($X=1$) vs. allocation to f2f CBTi ($X=0$) and dropout ($Y=1$) vs. no dropout ($Y=0$). Equation 24 can be used to calculate OR (Lipsey & Wilson, 2001):

$$OR = \frac{a * d}{b * c} \quad (24)$$

In this meta-analysis, a stands for the number of people allocated to dCBTi that dropped out, b for the number people allocated to dCBTi that did not drop out, c for the number of people allocated to f2f CBTi that dropped out, and d for the number people allocated to f2f CBTi that did not drop out.

The value of the OR can be interpreted as seen in Equation 25 (Card, 2011):

$$OR = 1: \text{no association between the two odds} \quad (25)$$

$$0 \leq OR < 1: \text{Odds } (Y=1 \mid X=1) < \text{Odds } (Y=1 \mid X=0)$$

$$1 < OR < +\infty: \text{Odds } (Y=1 \mid X=1) > \text{Odds } (Y=1 \mid X=0)$$

When interpreting OR, the CI and p-values need to be considered as well (Higgins et al., 2021a).

4.7.4.4 Publication bias. There is empirical evidence that in various scientific fields (medical, biological, physical, social sciences) studies are selectively reported depending on nature and direction of their results (Chan et al., 2014; Fanelli et al., 2017; Polanin et al., 2016). By analyzing trial registers, Schmucker et al. (2014) have revealed that merely half of registered studies were being published, and studies were more likely to be published if they revealed significant results rather than non-significant results (odds ratio 2.8; 95% CI: 2.2, 3.5; Schmucker et al., 2014). Complementary research further demonstrated that studies reporting results in favor of the experimental intervention as well as more favorable and larger results were more likely to be published (Dechartres et al., 2018; Rising et al., 2008; Scherer et al., 2018).

To counter the risk of publication bias, this meta-analysis aimed at including studies regardless of publication status. Additionally, the risk for publication bias was regarded in the statistical analysis. A funnel plot, where effect estimates are depicted in relation to their standard error (i.e., precision), was generated, forming a triangular area where 95% of studies should lie if there were no biases or heterogeneity. The studies effect sizes with similar SE

should be evenly scattered around the pooled effect size (Higgins et al., 2021d; Suurmond et al., 2017). If the statistical program registers asymmetry in the distribution, data points are imputed using the Trim-and-Fill method to adjust for studies that are potentially missing from the analysis (Suurmond et al., 2017).

Egger's regression is a weighted regression of the standardized effect sizes on their standard error (i.e., precision), and the regression slope should be zero in the absence of publication bias (Egger et al., 1997).

5. Results

5.1 Identification of trials

1552 records were identified through the search of the data bases and an additional 106 studies were identified through alternative sources (e.g. footnote chasing). After the removal of duplicates, the titles and abstracts of 955 records were screened for inclusion and reports that were obviously irrelevant excluded. If there were multiple reports of the same study, these reports were linked together. 62 full-text reports were reviewed regarding their compliance with the eligibility criteria. Reasons for exclusion are stated more specifically in Figure 3. The trial flow diagram is based on the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) checklist (Moher et al., 2009). 22 studies met the eligibility criteria and were chosen to be included in the meta-analysis.

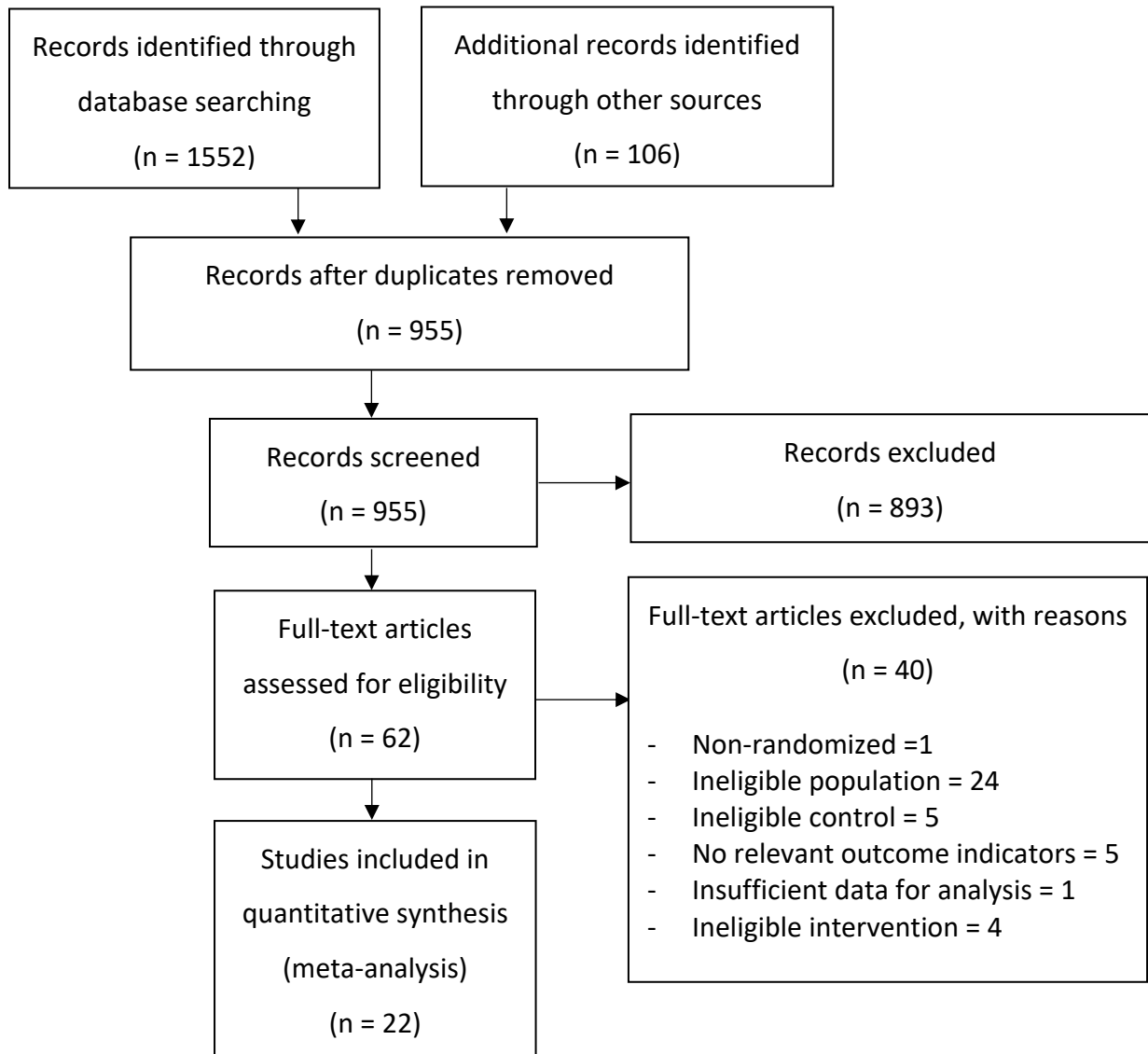
5.2 Study characteristics

Study characteristics of each study were categorized and are illustrated in table 1 and 2 to demonstrate similarities and differences between studies. Similarity of the study population and intervention across different studies included in the meta-analysis are illustrated in table 1. Within the 20 studies included in the analysis of the efficacy of dCBTi compared to control groups (passive and placebo) at post-assessment, there were 5836 participants, 76,59% female, and a mean age of 45.45 years (mean age ranged between 15.6 and 72.52). 11 studies were included in the short-term follow-up assessment of the efficacy of dCBTi, with a sample size of 4524, and three studies were included in the long-term follow-up assessment, consisting of 1600 participants. 13 studies were included in the calculation of the MD of ISI scores at post-assessment, with 2385 participants, eight studies at short-term follow up, with 2510 participants, and three studies at long-term follow up, with a sample size

of 1600. Within the four studies included in the non-inferiority analysis there were 253 participants, 81.9% of which were female and with a mean age of 45.06 (mean age ranged

Figure 3

Trial Flow Diagram



between 15.6 and 54.35 years). Three studies and 185 participants were included in the short-term follow-up non-inferiority analysis, with a mean age of 31.52 (range between 15.6 and 54.35), 74.88% of which were female.

Eight studies used placebo controls, 12 used passive controls, three used f2f CBTi and one included group f2f CBTi. The studies' attrition rates ranged between 0 and 49.4%. Some studies examined specific study populations. Two studies consisted of patients with at least subclinical depression (Batterham et al., 2017; van der Zweerde et al., 2018), one study

Table 1

Study characteristics illustrating similarity of the study population and intervention across studies

Study	Country	Population	Mean age	Intervention components										Automatization of Intervention	Intervention Period ¹
Batterham et al., 2017	Australia	Adults with insomnia and subclinical depression	$\mu=43$	S R	S C	C R	S H						R P	Fully Automated	6
Bernstein et al., 2017	USA	Adults with insomnia	$\mu=54,3$	S R	S C		S H		RE	BT	C T		M E D	Fully Automated	6
Blom et al., 2015	Sweden	Adults with insomnia	$\mu=48,01$	S R	S C	C R	S H	PE	RE					Guided	8
Blom et al., 2016	Sweden	Adults with insomnia	$\mu=48,01$	S R	S C	C R	S H	PE	RE					Guided	8
Cheng et al., 2019	USA	Adults with insomnia	$\mu=45,05$	S R	S C	C R	S H		RE			P I		Fully Automated	6
de Bruin et al., 2015	Netherlands	Adolescents with insomnia	$\mu=15,93$	S R	S C		S H	PE	RE		C T			Guided	6
de Bruin et al., 2018	Netherlands	Adolescents with insomnia	$\mu=15,6$	S R	S C		S H	PE	RE		C T			Guided	6

de Bruin et al., 2018 -Non-inferiority trial	Netherlands	Adolescents with insomnia	$\mu = 15,6$	S R	S C		S H	PE	RE	C T	Guided	6
Felder et al., 2020	USA	Pregnant women with insomnia	$\mu = 33,6$	S R	S C	C R	S H		RE	P I	Fully Automated	6
Krieger et al., 2019	Switzerland	Adults with acute or chronic insomnia	$\mu = 43,19$	S R	S C		S H	PE	RE	C T	Guided	8
Kyle et al., 2020	UK	Adults with insomnia with concentration and memory difficulties	$\mu = 52,4$	S R	S C	C R	S H		RE	P I	Fully Automated	6
Lancee et al., 2015	Netherlands	Adults with insomnia	$\mu = 48,55$	S R			S H	PE	RE	C T	Guided	6
Lancee et al., 2016	Netherlands	Adults with insomnia	$\mu = 43,15$	S R			S H	PE	RE	C T	Guided	6
Lancee et al., 2016 - Non-inferiority trial	Netherlands	Adults with insomnia	$\mu = 39,85$	S R			S H	PE	RE	C T	Guided	6

Lien et al., 2019	Norway	Adults with insomnia	$\mu= 44,8$	S R	S C	C R	S H					R P	Fully Automated	9
Luik et al., 2020	UK	Adults with insomnia	$\mu= 48$	S R	S C	C R	S H		RE		P I		Fully Automated	6
Majd et al., 2020	Iran	Adults with insomnia	$\mu= 35,75$			C R	S H	PE	RE	BT			Fully Automated	6
Ritterband et al., 2017	USA	Adults with chronic insomnia disorder	$\mu=43,28$	S R	S C	C R	S H					R P	Fully Automated	6
Sato et al., 2019	Japan	Adults with insomnia who remained symptomatic after pharmacotherapy	$\mu= 49,97$	S R	S C	C R	S H		RE	BT			Guided	6
Siebmanns et al., 2021	Sweden	Adults with insomnia and cardiovascular disease	$\mu= 72,52$	S R	S C		S H	PE	RE			M S C T	Guided	9

Taylor et al., 2017	USA	Active duty soldiers with chronic insomnia	$\mu = 32,73$	SR	SC	CR	SH		RE			Fully Automated	6
Taylor et al., 2017 - Non-inferiority trial	USA	Active duty soldiers with chronic insomnia	$\mu = 32,73$	SR	SC	CR	SH		RE			Fully Automated	6
van der Zweerde et al., 2018	Netherlands	Adults with insomnia and at least subclinical depression	$\mu = 45,99$	SR	SC	CR	SH	PE	RE	CT	RP	Guided	5
van der Zweerde et al., 2020	Netherlands	Adults with insomnia	$\mu = 50,6$	SR	SC	CR	SH	PE	RE	CT	RP	Guided	5

Note. Figure retrieved from Meta-Essentials (Suurmond et al., 2017).¹ Intervention period in weeks. SR= sleep restriction. SC= stimulus control. CR = cognitive restructuring. SH=sleep hygiene. PE = psychoeducation. RE= relaxation. BT= behavioral therapeutic intervention. CT = cognitive therapeutic intervention. PI= paradoxical intention. RP = relapse prevention. MED= meditation. MSCT= management of stress and dysfunctional thoughts.

examined patients with a cardiovascular disease (Siebmanns et al., 2021), and one focused on patients reporting difficulties with concentration and memory (Kyle et al., 2020) in addition to insomnia disorder. One study was made up of pregnant women with insomnia (Felder et al., 2020), one exclusively included patients whose insomnia disorder remained after pharmacotherapy (Sato et al., 2019) and one study only included active-duty soldiers with insomnia disorder (Taylor et al., 2017). More detailed information on the effect of interest, the outcome measure used to calculate the effect size, and the assessment points of individual studies are stated in table 2.

Two studies received a low risk of bias judgement, eight studies displayed some concerns and 14 were rated as having high risk of bias (see table 2).

5.3 Hypotheses

Hypothesis 1. dCBTi improves insomnia severity compared to the control conditions, resulting in SMD in favor of dCBTi at post assessment: $g > 0$ and $p < .05$.

20 studies were included in this analysis assessing the efficacy of dCBTi at post-intervention. The analysis yielded a large and highly significant effect, represented by Hedges' g , favoring dCBTi to the control conditions ($g = 0.98$, $SE = 0.09$, 95% CI: 0.79, 1.16, $p < .0001$). The results were highly heterogenous ($I^2 = 87.07\%$, $p < .0001$), which indicates that there is high variation between effects. The forest plot shows one outlier (Sato et al., 2019, see Figure 4) with significantly higher estimates, however, due to small sample size and study weight the study does not have an important impact on the results. Sensitivity analysis reveals that the results are robust to the exclusion of the study.

Hypothesis 2. dCBTi improves insomnia severity compared to the control conditions, resulting in SMD in favor of dCBTi at 1-6 months and > 6 months follow-up assessment: $g > 0$ and $p < .05$.

11 studies were included in the analysis to assess the efficacy of dCBTi compared to control conditions at short-term follow up. The effect was large, highly significant and in favor of dCBTi ($g = 0.84$, $SE = 0.16$, 95% CI: 0.5, 1.19, $p < .0001$). Heterogeneity was extremely high ($I^2 = 94.80\%$, $p < .000$), indicating a high variation between effects. The long-term follow-up assessment included 3 studies, had a sample size of 1600 and yielded a significant effect

Table 2*Characteristics of studies included in the meta-analysis*

Study	Effect	Control	Sample, sample size	Outcome measure	Short-Term FU	Long- Term FU	ROB judgement ¹
Batterham et al., 2017	SMD	Placebo control	ITT n= 1149	ISI	6 mo	18 mo	Low
Bernstein et al., 2017	SMD	Passive control	ITT n= 88	ISI	2 mo	/	High
Blom et al., 2015	SMD	Active control	ITT n= 48	ISI	6 mo	/	Some concerns
Blom et al., 2016	SMD	Placebo control	ITT n= 148	ISI	6 mo	36 mo	Some concerns
Cheng et al., 2019	SMD	Placebo control	PP n= 658	ISI	/	36 mo	High
de Bruin et al., 2015	SMD	Passive control	ITT n= 32	HSDQ & CSRQ	/	/	High
de Bruin et al., 2018	SMD	Passive control	ITT n= 78	HSDQ & CSRQ	2 mo	/	High

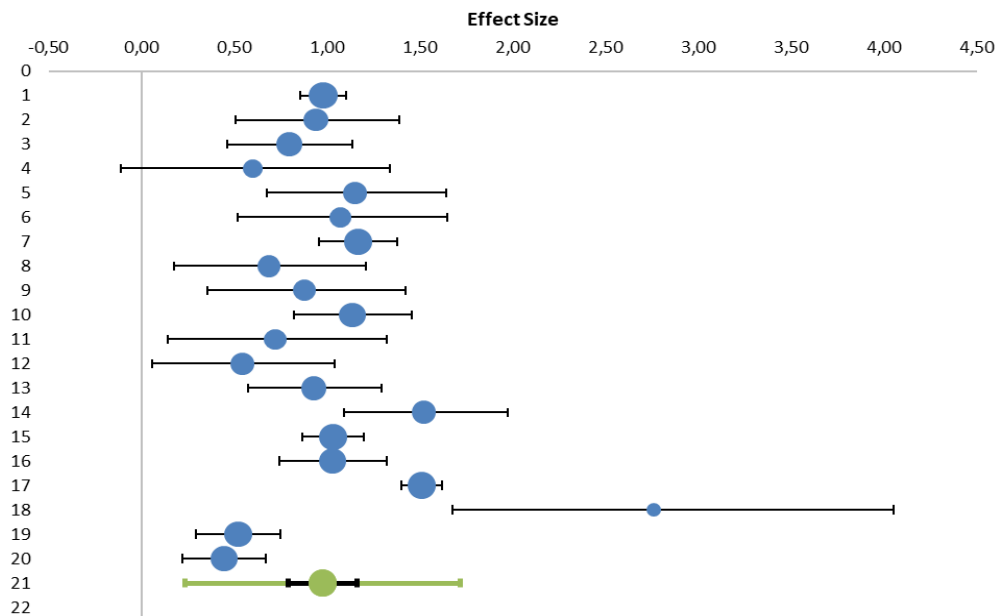
de Bruin et al., 2018 -Non-inferiority trial	SMD	Active control	ITT n= 77	HSDQ & CSRQ	2 mo	/	Some concerns
Felder et al., 2020	Adjusted SMD	Passive control	ITT n= 208	ISI	4,5 mo	/	High
Krieger et al., 2019	SMD	Passive control	ITT n= 62	ISI	6 mo	/	High
Kyle et al., 2020	SMD	Passive control	ITT n= 410	ISI	6 mo	/	High
Lancee et al., 2015	SMD	Passive control	ITT n= 63	ISI	6 mo	/	High
Lancee et al., 2016	SMD	Passive control	ITT n= 60	ISI	6 mo	/	High
Lancee et al., 2016 - Non-inferiority trial	SMD	Active control	ITT n= 60	ISI	6 mo	/	Some concerns
Lien et al., 2019	SMD	Placebo control	ITT n= 178	ISI	6 mo	18 mo	Some concerns
Luik et al., 2020	Adjusted SMD	Placebo control	ITT n= 1711	SCI	6 mo	12 mo	Some concerns

Majd et al., 2020	Adjusted SMD	Placebo control	ITT n= 312	ISI	6 mo	/	Some concerns
Ritterband et al., 2017	Adjusted SMD / FU: SMD (PP)	Placebo control	ITT n= 303 / FU: PP	ISI	6 mo	12 mo	Low
Sato et al., 2019	SMD	Passive control	ITT n= 23	PSQI	3 mo	/	High
Siebmanns et al., 2021	SMD	Placebo control	ITT n= 48	ISI	6 mo	/	Some concerns
Taylor et al., 2017	SMD	Passive control	ITT n= 67	ISI	6 mo	/	High
Taylor et al., 2017 - Non-inferiority trial	SMD	Active control	ITT n=68	ISI	6 mo	/	High
van der Zweerde et al., 2018	SMD	Passive control	ITT n= 104	ISI	6 mo	/	High
van der Zweerde et al., 2020	SMD	Passive control	ITT n= 134	ISI	6,5 mo	13 mo	High

Note. ¹ Risk of bias judgement for post-intervention effect assessment. ITT= intention-to-treat. PP= per-protocol. SMD= standardized mean difference. Adjusted SMD= SMD adjusted to baseline scores. ISI= Insomnia severity index. SCI= Sleep Condition Indicator. PSQI= Pittsburgh Sleep Quality index. FU= follow-up-assessment.

Figure 4

Forest plot of studies' effect sizes comparing dCBTi to passive and placebo controls at post-assessment



Note. Figure retrieved from Meta-Essentials (Suurmond et al., 2017).

favoring dCBTi ($g = 0.42$, $SE = 0.15$, $p < .01$). Heterogeneity in the long-term follow-up assessment was substantial ($I^2 = 79.52\%$, $p < .01$).

Hypothesis 3. The overall unstandardized MD of ISI between intervention and control group at post-assessment is ≥ 6 .

13 studies were included in the calculation of the MD of ISI scores between dCBTi and controls at post-assessment. The MD was 5.29 and highly significant in favor of dCBTi ($SE = 0.42$, 95% CI: 4.37, 6.21, $p < .0001$), which is slightly below the value defined as representing clinically meaningful change. Heterogeneity was extremely high ($I^2 = 94.80\%$, $p < .0001$), indicating a high variation between effects. Three studies produced a MD of more than 6 points (12.79% of the sample), and 11 studies had a MD of more than 4 points (95.18% of the sample).

Hypothesis 4. The overall unstandardized MD of ISI between intervention and control group at 1-6 months and > 6 months follow-up-assessment is ≥ 6 .

Eight studies were included in the calculation of the MD of ISI scores between dCBTi and controls at 1-6 months follow-up-assessment. The MD was 4.07 and highly significant in favor of dCBTi (SE = 0.54, 95% CI: 2.80, 5.34, $p < .0001$). However, the score is below the score regarded for clinically meaningful improvement. Only one study had a MD of more than 6 points (participants in this study make up merely 0.92% of the sample), and 4 studies had a MD of more than 4 points (these studies include 74.78% of participants). Assessment of variability between effects revealed extremely high heterogeneity ($I^2 = 98.43\%$, $p < .0001$). The calculation of the MD of ISI scores between dCBTi and controls at long-term follow-up assessment (> 6 months to 1 year post-intervention) included three studies that yielded a sample of 1600 participants. The MD of 2.86 in favor of dCBTi was significant (SE = 0.99, 95% CI: -1.41, 7.12, $p < .01$), and heterogeneity was very high ($I^2 = 99.27\%$). There was no study that had a MD of more than 6 at long-term follow-up assessment.

Hypothesis 5. The overall SMD between dCBTi and control groups is moderated by mean age of the participants included in the studies ($p < .05$).

The regression analysis testing the moderating effect of mean age on effect size was not significant ($p = 0.507$), indicating that the size of the effect is not dependent on the mean age of the participants included in the studies. The scatterplot reinforces these results as there seems to be no corresponding trend (see Figure 5).

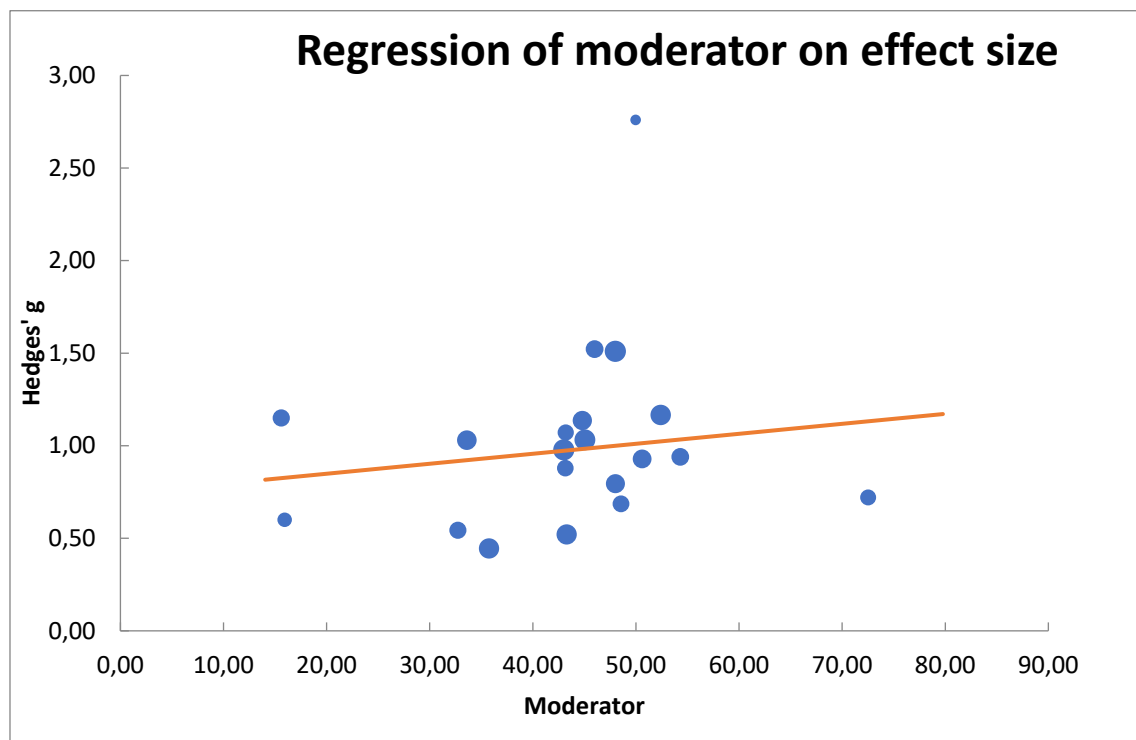
Hypothesis 6. The SMD for studies using fully automated dCBTi is different from the SMD for studies using guided dCBTi: $\mu_1 \neq \mu_2$ and $p < .05$.

A subgroup analysis was conducted to see if the SMD between dCBTi and control groups at post-assessment was different in studies using fully automated dCBTi from studies using guided dCBTi.

Of the 20 studies included in this analysis, 10 were included in the subgroups for fully automated dCBTi and guided dCBTi each (see table 3 for more details). There was no significant difference in the effect size between groups ($p = 0.69$) and the effect size within the two groups was similar. Hedges' g of the group including studies using fully automated dCBTi was $g = 0.94$, and of the group including studies using guided dCBTi Hedges' g was $g = 1.01$. Heterogeneity was high in the subgroup of fully automated dCBTi ($I^2 = 92.76\%$) but could be considered only moderately high in the subgroup of guided dCBTi interventions ($I^2 = 56.9\%$).

Figure 5

Regression of moderator mean age on effect size in main analysis at post-assessment



Note. Figure retrieved from Meta-Essentials (Suurmond et al., 2017).

Hypothesis 7. The SMD for studies using placebo control groups is different from the SMD for studies using passive control groups: $\mu_1 \neq \mu_2$ and $p < .05$.

Another subgroup analysis was conducted to see if the SMD between dCBTi and control groups at post-assessment was different in studies comparing dCBTi to placebo control groups from studies comparing dCBTi to passive controls. Of the 20 studies included in the analysis, eight were included in the subgroup for active placebo control conditions and 12 in the subgroup for passive control conditions. There was no significant difference in the effect size between groups ($p = 0.472$) and the effect size within the two groups was quite similar, though the effect size for studies using active placebo control groups was slightly smaller than for studies using passive controls ($g = 0.90$ and $g = 1.03$, respectively). The subgroup of passive controls had more homogenous results than the subgroup of active or placebo controls ($I^2 = 53.74\%$ and $I^2 = 94.3$, respectively). The results are illustrated in table 4.

Table 3

Subgroup analysis for fully automated and guided dCBTi vs. controls for post-intervention Hedges' g

Study name / Subgroup name	Hedges' g	CI Lower limit	CI Upper limit	Weight	P	I ²	PI Lower limit	PI Upper limit
Batterham et al. (2017)	0,98	0,86	1,10	11,13%				
Bernstein et al.(2017)	0,94	0,50	1,38	8,28%				
Cheng et al. (2019)	1,03	0,87	1,20	10,88%				
Felder et al. (2020)	1,03	0,74	1,32	9,82%				
Kyle et al. (2020)	1,17	0,96	1,38	10,54%				
Lien et al. (2019)	1,14	0,82	1,45	9,55%				
Luik et al. (2020)	1,51	1,40	1,62	11,20%				
Majd et al. (2020)	0,45	0,22	0,67	10,41%				
Ritterband et al. (2017)	0,52	0,29	0,75	10,37%				
Taylor et al. (2017)	0,54	0,05	1,03	7,81%				
Fully automated	0,94	0,73	1,15	64,53%	0,000	92,76%	0,09	1,79
Blom et al. (2016)	0,80	0,46	1,13	13,86%				
de Bruin et al. (2015)	0,60	-0,13	1,33	7,38%				
de Bruin et al. (2018)	1,15	0,67	1,63	10,92%				
Krieger et al. (2019)	1,07	0,51	1,64	9,51%				
Lancee et al. (2015)	0,69	0,17	1,20	10,31%				
Lancee et al. 2016	0,88	0,34	1,41	10,02%				
Sato et al. (2019)	2,76	1,57	3,95	3,75%				
Siebmans et al. (2021)	0,72	0,13	1,31	9,14%				
van der Zweerde et al. (2018)	1,52	1,08	1,96	11,73%				
van der Zweerde et al. (2020)	0,93	0,57	1,29	13,39%				
Guided	1,01	0,73	1,30	35,47%	0,013	56,90%	0,29	1,74

Note. CI= confidence interval. PI= prediction interval

Table 4*Subgroup analysis of different control conditions for post-intervention Hedges' g*

Study name / Subgroup name	Hedges' g	CI Lower limit	CI Upper limit	Weight	P	I ²	PI Lower limit	PI Upper limit
Batterham et al. (2017)	0,98	0,86	1,10	13,75%				
Blom et al. (2016)	0,80	0,46	1,13	11,87%				
Cheng et al. (2019)	1,03	0,87	1,20	13,49%				
Lien et al. (2019)	1,14	0,82	1,45	12,07%				
Luik et al. (2020)	1,51	1,40	1,62	13,83%				
Majd et al. (2020)	0,45	0,22	0,67	12,99%				
Ritterband et al. (2017)	0,52	0,29	0,75	12,95%				
Siebmans et al. (2021)	0,72	0,13	1,31	9,05%				
Active/Placebo control	0,90	0,66	1,15	43,96%	0,000	94,30%	-0,07	1,88
Bernstein et al.(2017)	0,94	0,50	1,38	8,84%				
de Bruin et al. (2015)	0,60	-0,13	1,33	5,04%				
de Bruin et al. (2018)	1,15	0,67	1,63	8,12%				
Felder et al. (2020)	1,03	0,74	1,32	12,23%				
Krieger et al. (2019)	1,07	0,51	1,64	6,84%				
Kyle et al. (2020)	1,17	0,96	1,38	14,22%				
Lancee et al. (2015)	0,69	0,17	1,20	7,56%				
Lancee et al. 2016	0,88	0,34	1,41	7,29%				
Sato et al. (2019)	2,76	1,57	3,95	2,37%				
Taylor et al. (2017)	0,54	0,05	1,03	7,99%				
van der Zweerde et al. (2018)	1,52	1,08	1,96	8,90%				
van der Zweerde et al. (2020)	0,93	0,57	1,29	10,62%				
Passive control	1,03	0,81	1,25	56,04%	0,014	53,74%	0,47	1,59

Note. CI= confidence interval. PI= prediction interval.

Hypothesis 8. There is a significant difference in the rate of improvement of insomnia severity at the post-intervention assessment between dCBTi and f2f CBTi, favoring f2f-CBTi: $g > 0$ and $p < .05$.

Four studies were included in this analysis assessing the efficacy of dCBTi compared to f2f CBTi at post-intervention-assessment. The statistical power of the analysis is therefore small. The analysis yielded no significant effect ($g = -0.40$, $SE = 0.29$, 95% CI: $-1.31, 0.51$, $p = .081$). The results were highly heterogenous ($I^2 = 80.17\%$, $p < .01$), which indicates that there is high variation between effects. Summary statistics are depicted in Table A1 in Appendix A.

Hypothesis 9. There is a significant difference in the rate of improvement of insomnia severity at the 1-6 months and > 6 months follow-up-assessment between dCBTi and f2f CBTi, favoring f2f-CBTi: $g > 0$ and $p < .05$.

Only three trials were included in the short-term follow-up-assessment of the efficacy of dCBTi compared to f2f CBTi, so the statistical power of the analysis is very small. The effect was non-significant ($g = -0.44$, $SE = 0.36$, 95% CI: $-2.00, 1.12$, $p = .110$) and the results highly heterogenous ($I^2 = 82.56\%$, $p < .01$), which indicates that there is high variation of effect sizes between studies. Due to lack of studies, no long-term follow-up assessment was conducted, and the according research hypothesis regarding efficacy of dCBTi compared to f2f CBTi at long-term follow-up could not be answered.

Hypothesis 10. The overall unstandardized mean difference of ISI between dCBTi and f2f CBTi at post-assessment is larger than 4 points, favoring f2f CBTi.

Only three studies reported the necessary values to include in the calculation of MD of ISI scores between dCBTi and f2f CBTi at post-assessment, so the statistical power of this analysis is very low. The MD was -3.20 and the result was significant ($p < .01$) in favor of f2f CBTi ($SE = 1.16$, 95% CI: $-8.17, 1.77$, $p < .01$). This score lies within the non-inferiority margin that was defined at 4 points. Heterogeneity was extremely high ($I^2 = 98.05\%$, $p < .000$), indicating a high variation between effects.

Hypothesis 11. The overall unstandardized MD of ISI between dCBTi and f2f CBTi at 1-6 months and > 6 months follow-up-assessment is larger than 4 points, favoring f2f CBTi.

Again, only three studies were included in the assessment of MD of ISI scores between dCBTi and f2f CBTi at short-term follow up, so the statistical power of the analysis is very low.

The MD was -1.92 in favor of f2f CBTi, the result was non-significant (SE = 1.51, 95% CI: -8.41, 4.56, $p = .101$). As heterogeneity was again extremely high ($I^2 = 99\%$, $p < .000$), variation of effect sizes between studies is high. Due to lack of studies, no long-term assessment was conducted, and the research hypothesis could not be answered for long-term follow-up assessment.

Hypothesis 12. The overall SMD between dCBTi and f2f CBTi is moderated by mean age of the participants included in the studies ($p < .05$).

Because there were only three studies included in the analysis, the moderator analysis has too few data points to have significant meaning. However, the scatter plot can be regarded for a rough interpretation, and there seems to be no apparent pattern in the distribution (see Appendix A, Figure A1).

Hypothesis 13. Participants are more likely to drop out of the intervention when they are placed in dCBTi compared to f2f CBTi: Odds Ratio $\neq 0$ and $p < .05$.

The four trials included in the non-inferiority analysis assessing the efficacy of dCBTi compared to f2f CBTi were incorporated in this analysis. Therefore, statistical power is low. Three studies offered data for dropout as less than four sessions completed, for one study dropout data of less than three sessions completed was available. The odds ratio was calculated for the probability of dropout when participants are assigned to dCBTi compared to f2f CBTi. OR was 2.43 and highly significant (95% CI: 1.08, 5.5, $p < .001$), indicating that participants in dCBTi are 2.43 times more likely to drop out of the intervention than participants in f2f CBTi.

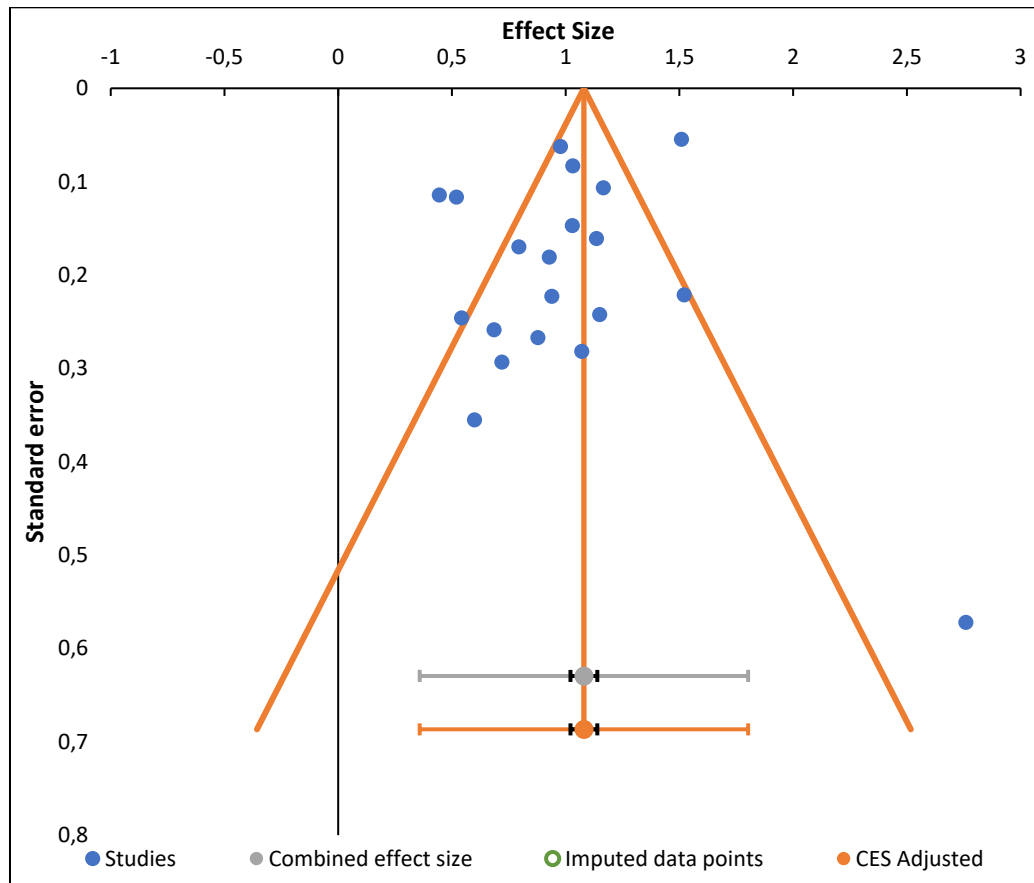
5.4 Publication bias

Publication bias was tested by interpreting results of a funnel plot (see Figure 6). Effect sizes with similar precision should be distributed symmetrically around the overall effect size. The program did not impute new data-points based on the Trim – and – Fill method, which can be interpreted as there being no substantial asymmetry. If the program had identified asymmetry, additional studies would have been imputed to adjust the pooled effect sizes for studies that might be missing due to publication bias. These results are confirmed by insignificant results from Egger's regression ($p = 0.192$).

5.5 Sensitivity analysis

Figure 6

Funnel plot of studies included in the meta-analysis comparing effects from dCBTi to control conditions



Note. Figure retrieved from Meta-Essentials (Suurmond et al., 2017).

Sensitivity analysis was conducted to check if decisions made in the research process regarding the inclusion of studies with certain features had an influence on the intervention effect. Studies were individually and categorically excluded. There was no considerable change in overall effect size when studies reporting adjusted SMD were excluded from the analysis. To see whether the results had been the same if studies with a PP sample had not been eligible, the concerned studies were excluded and the overall effect checked for changes. Results were robust to the exclusion of results from Cheng et al. (2019). When excluding results from Majd et al. (2020) from the analysis, the overall effect changed from $g = 0.98$ to $g = 1.1$. Since the danger of including studies using a PP sample lies in overestimation of effect size and results were smaller when the study was included, the study remained in the analysis.

5.6 Risk of bias

A subgroup analysis for studies categorized to different risk of bias ratings was not conducted, because studies were predominantly assigned to have high risk of bias. Most studies were rated as having high risk of bias because participants were unblinded to the treatment allocation. The outcome used to calculate the effect of interest in this analysis is derived from self-rated questionnaires. This means that participants themselves are considered the outcome assessors and their knowledge about allocation could influence their judgement on insomnia severity (Higgins et al., 2021b). For participants to be blinded, studies had to include a placebo control group or an alternative intervention. A subgroup analysis compared effect in groups using passive controls (TAU, wait-list) to groups using placebo controls (e.g. online psychoeducation programs). There was no significant difference in effect size. These results indicate that the effectiveness of the intervention was not dependent on the studies using passive or active control groups.

6 Discussion

6.1 Summary of results

dCBTi is effective in reducing insomnia severity in patients with insomnia disorder compared to passive and placebo control conditions at post-intervention-assessment, at short-term follow-up of one to six months post-intervention as well as long-term follow-up of more than six months until one-year post-intervention. Fully automated dCBTi interventions produce similar treatment effects to guided dCBTi, however, heterogeneity of effect sizes was higher in studies using fully automated dCBTi than in those with guided dCBTi. The results seem to be robust whether active placebo or passive control groups were used, indicating that the size of the treatment effect cannot be attributed to participants' expectations to experience improvements. The MD of ISI scores at post-assessment and at short-term follow-up-assessment were below the predefined score representing clinically meaningful change (6 points), but lay above a score of 4, which seems to be indicative for treatment success (Yang et al., 2009). The MD of ISI scores at long-term follow-up assessment was however less than 4 points, though significant. dCBTi seems to produce similar but slightly inferior results than f2f CBTi at post-assessment and short-term follow-up, with non-significant SMD. Furthermore, participants seem to be more likely to drop out of the digital intervention than out of f2f CBTi.

Interpretation of these results must however be done cautiously since the non-inferiority analysis was underpowered. Due to lack of studies, no long-term follow-up assessment comparing dCBTi to f2f CBTi could be conducted.

6.2 Key findings

This meta-analysis has demonstrated that dCBTi produces large treatment effects in a clinically representative population. The sample exclusively consists of patients meeting diagnostic criteria for insomnia disorder, making this the only current meta-analysis that explores efficacy of dCBTi specifically in this population. There were no constrictions in eligibility regarding comorbidities, so results should be useful for a clinical population, since insomnia disorder is especially common in patients with other mental or medical disorders (Pearson et al., 2006).

In previous meta-analytic reviews, Ye et al. (2016; $n = 14$, overlap = 2), Seyffert et al. (2016; $n = 15$, overlap = 2) and Zachariae et al. (2016; $n=11$, overlap = 0) examined studies comparing dCBTi to control conditions in people meeting criteria for insomnia diagnosis (Ye et al., 2016) or exhibiting insomnia symptoms (Seyffert et al., 2016; Zachariae et al., 2016). The included trials were conducted between 2004 and 2015 in all three meta-analyses. The current study included studies published between 2015 and 2021, complementing results from these previous meta-analyses by assessing the effect of current dCBTi interventions.

The results of the analysis can be best compared to that of the most recent meta-analysis by Soh et al. (2020) including 33 trials (overlap = 17) from 2004 to 2020. Like the current systematic review, Soh et al. (2020) conducted two separate analyses by comparing dCBTi to controls as well as to f2f CBTi. The populations however differed slightly between the two meta-analyses, as in Soh et al. (2020) studies examining patients with subclinical insomnia were eligible and studies on adolescents excluded from the analysis. Soh et al. (2020) used mean difference of ISI scores as effect measure and reported a MD of -5.00 (95% CI: -5.68, -4.33, $p < 0.0001$) between participants in dCBTi and control conditions at post-assessment. The short-term follow-up analysis yielded a significant though smaller effect size of -3.99 (95% CI -4.82 to -3.16, $p < 0.0001$, $d = 0.324$). The effect size used in the current analysis was Hedges' g , and overall effects were large for post-intervention ($g = 0.98$, $SE=0.09$, 95% CI: 0.79, 1.16, $p < .0001$) and short-term follow up assessment ($g = 0.84$, $SE = 0.16$, 95% CI: 0.5, 1.19, $p < .0001$) when comparing dCBTi to control conditions, and small at long-term

follow-up assessment ($g = 0.42$, $SE = 0.15$, $p < .01$). However, the long-term assessment included only three studies, so the statistical power of the results is low. The MD analysis produced results similar to those of Soh and colleagues (2020). MD at post-assessment was 5.29 (95% CI: 4.37, 6.21, $p < .0001$), 4.07 at short-term follow-up assessment (95% CI: 2.80, 5.34, $p < .0001$), and 2.86 at long-term follow-up assessment (95% CI: -1.41, 7.12, $p < .01$). Only three studies (12.79% of the sample) at post-assessment and merely one study (less than 1% of the sample) at the short-term follow-up assessment had a MD of more than 6. However, 11 studies (96.18% of the sample) at post-assessment and 4 studies (74.78% of the sample) at short-term follow-up assessment produced a MD of more than 4 points. In this analysis, a 6-points decrease of the ISI score was considered a clinically meaningful reduction. The achieved improvements were slightly beneath the set score. However, Soh et al. (2020) argue that their results provide strong support for the efficacy of dCBTi. Since an improvement of more than 4 ISI points already reduces the participant's risk for negative treatment outcomes, increases their vitality and decreases fatigue (Yang et al., 2009), the results of the current analysis still point to the effectiveness of the dCBTi at post-treatment and short-term follow-up.

Soh et al. (2020) included four studies in their non-inferiority analysis assessing efficacy of dCBTi compared to f2f CBTi, two of which are also included in the current meta-analysis. The analysis demonstrated that f2f CBTi produced significantly better results than dCBTi (MD = 3.07, 95% CI 1.18–4.95, $p = 0.001$). In the current analysis, the effect size was represented by Hedges' g and resulted in no significant effect ($g = -0.4$, $SE = 0.29$, 95% CI: -1.31, 0.51, $p = .081$). However, the results still indicate that f2f CBTi produces superior results to dCBTi, as statistical power is low and might be responsible for producing insignificant results. The MD of ISI scores between dCBTi and f2f CBTi at post-assessment was -3.20 and the result was significant ($p < .01$) in favor of f2f CBTi ($SE = 1.16$, 95% CI: -8.17, 1.77, $p < .01$), and the MD of ISI scores at short-term follow-up was -1.92 ($SE = 1.51$, 95% CI: -8.41, 4.56). However, only three studies provided data to calculate MD both at post-assessment as well as at short-term follow-up, so the values hold little power. There is a demand for studies directly comparing dCBTi to f2f therapy, so that conclusions about efficacy can be drawn with more confidence.

In the current meta-analysis, dropout from an intervention is regarded a serious adverse event and therefore considered in the statistical analysis. The odds ratio was calculated for the probability of participants dropping out of dCBTi compared to f2f CBTi, revealing an OR of 2.43, indicating that participants in dCBTi are much less likely to complete

the intervention than participants in f2f CBTi (95% CI: 1.08, 5.5, $p < .001$). An equal analysis could not be identified in any of the previously conducted meta-analyses.

There was high heterogeneity across all analyses conducted ($I^2 = 87.07\%$, $p < .0001$ at the analysis of dCBTi vs. control conditions at post-assessment), which might be partially contributed to the heterogenous sample. Soh et al. (2020) also reported high heterogeneity, though it was slightly smaller than in the current study ($I^2 = 76\%$ for the analysis of dCBTi vs. controls at post-assessment). In this study, subgroup analyses did not reveal factors responsible for causing heterogenous results. Neither comparator group (active vs. passive control) nor the degree of automatization of the dCBTi intervention (fully automated vs. guided) could explain the large distribution of effect sizes. Likewise, Soh et al. (2020) could not identify any significant connection between the subgroup variables (specific comorbidities, therapist guidance, number of sessions) and the MD of ISI scores at post-intervention. The results of both meta-analyses indicate that the intervention effect does not depend on the intervention being delivered with or without guidance. However, the current analysis revealed that heterogeneity of effect sizes in studies using fully automated dCBTi was significantly higher than in studies using guided dCBTi, indicating that the effectiveness of fully automated dCBTi might depend on specific moderating factors, such as characteristics of the population receiving the intervention.

A moderator analysis was conducted to explore the connection between the mean age of participants and the intervention effect. The distribution of effect sizes did not reveal a pattern that would indicate an association between mean age and effect size. Soh et al. (2020) did not consider age as a possible moderating factor, so the factor age was disregarded in their analysis.

In the current meta-analysis, efficacy was demonstrated across a wide age spectrum, which might be of great value when considering whether dCBTi should be implemented in clinical practice. Vigerland et al. (2016) demonstrated that digital CBT might be an effective treatment option for adolescents with various psychiatric conditions. Results from Werner-Seidler and colleagues (2018) further indicated that dCBTi produces good results in treating insomnia symptoms in adolescents and young adults, reporting effect sizes between 0.17 and 1.30 in their systematic review of three studies. Past research regarding efficacy of dCBTi has often excluded adolescents even though the intervention seems to be similarly effective in

this patient group. By including adolescents, results of the current analysis can be regarded when considering the implementation of dCBTi in an adolescent population.

6.3 Additional findings

Besides demonstrating efficacy in reducing insomnia severity, most studies identified improvements in symptoms of comorbid disorders in their individual reports, which was in line with results from previous studies (see Morin et al., 2015). dCBTi was effective in reducing symptoms of depression (Batterham et al., 2017; Blom et al., 2015; Cheng et al., 2019; Felder et al., 2020; Lancee et al., 2016; Majd et al., 2020; van der Zweerde et al., 2018, 2020) as well as anxiety disorder (Batterham et al., 2017; Felder et al., 2020; Majd et al., 2020), and effects on depression severity were comparable to those of digital and f2f interventions for depression (Batterham et al., 2017). In adolescents, dCBTi had improved cognitive functioning, specifically visuospatial processing, selective attention, and phonological working memory (de Bruin et al., 2015), and led to a reduction in symptoms of affective disorders, anxiety disorders, somatic disorders, oppositional defiant disorder, and attention deficit hyperactivity disorder (de Bruin et al., 2018).

Participants that had received dCBTi used less sleep medication after treatment (Blom et al., 2016; Kyle et al., 2020), had better functional health, psychological well-being, and sleep-related quality of life (Luik et al., 2020). They reported experiencing less cognitive impairment (Kyle et al., 2020) and improvements in perceived stress (Bernstein et al., 2017). dCBTi was effective in reducing sleep safety behaviors and dysfunctional beliefs, which mediate the treatment effect on insomnia severity (Lancee et al., 2015).

Cheng et al. (2019) found that age (among other demographic variables) was no significant moderator of treatment effects. However, other studies did not consider age as a moderating variable. Adherence to the treatment led to larger improvements of insomnia severity (Siebmanns et al., 2021). Furthermore, Sato et al. (2019) demonstrated that dCBTi is effective in reducing insomnia severity in patients that had remained symptomatic after receiving pharmacotherapy.

6.4 Limitations

There are substantial limitations to the current meta-analysis and the assumptions that can be drawn from it.

Most of the studies included in the analysis received a high risk of bias rating, and the influence of a studies' risk of bias could not be tested directly in a subgroup analysis due to the uneven share of studies assigned to the three categories. Nevertheless, the effect size did not decrease when studies with low risk of bias rating were excluded from the analysis, and a subgroup analysis comparing studies with passive controls to placebo controls showed no difference in effect between those groups. This suggests that the effect is not dependent on participants being blinded or not. However, the reliability of the risk of bias rating is unclear since there was no second rater.

The current meta-analysis, including the literature search, eligibility screening, coding, categorization of studies and data analysis was conducted by one person. All steps were taken and decisions made with regard to recommendations by the Cochrane Collaboration (Higgins et al., 2021). The procedure was discussed and the statistical analysis done with assistance from a colleague. However, none of the steps were independently conducted by a second person, so reliability of the results might be considerably reduced.

Furthermore, the statistical power is limited in the non-inferiority analyses comparing dCBTi to f2f CBTi, which substantially limits the value of the meta-analytic results. There is a need for studies comparing these two interventions if dCBTi should be considered an alternative to f2f CBTi in clinical practice. Additionally, only very few studies offered values to assess long-term treatment effects. Meta-analytic effect measures comparing dCBTi to controls at long-term assessment are therefore of low statistical power, and no long-term follow-up effect comparing dCBTi to f2f CBTi could be calculated.

A moderator analysis was conducted to explore whether the size of the intervention effect was dependent on the age of the participants. However, there were few studies and therefore few datapoints included for the regression analysis, so results are not very informative. The scatter plot had to be interpreted as to whether age seemed to be connected to the effect size. Additionally, age is difficult to incorporate as a moderating factor in a meta-analysis, as using the mean age of a studies' participants introduces aggregation bias (Higgins et al., 2021c; Morgenstern, 1982). If the mean ages of studies are comparable, it might be possible that no connection will be identified, even though age has an effect in the individual trials. Mean age of the studies included in this analysis however range between $\mu = 15.6$ and $\mu = 72.52$, and the distribution of effect sizes does not indicate that a studies' higher or lower

mean age determines the size of the treatment effect. Nevertheless, no definite answer on whether age influences the size of the intervention effect can be obtained from the current meta-analysis.

The effect of interest was defined as SMD of outcomes assessing insomnia severity between the intervention and a control group at post-intervention or follow-up assessment. Summary data was collected and Hedges' g calculated for individual studies before calculating a pooled effect size, so that the estimates were consistent. However, not all studies included in the current meta-analysis reported the same summary data, and values had to be transformed to reach an approximation of Hedges' g . Also, some studies did not provide any summary data at all, so effect sizes were adopted directly. Studies using the SMD adjusted to baseline values were also included in the analysis, since the effect measure should merely differ in precision of capturing the true effect, rather than being qualitatively different. Since authors might systematically report data that result in higher effect estimates, the results from this analysis might be an overestimation of the true intervention effect.

Furthermore, the effect of interest was that of assignment to the intervention at baseline, so studies conducting an ITT analysis were favored. However, studies that based their analysis on PP data were also included in the analysis, even if researchers did not calculate outcome scores for all participants. Patients that did not adhere to the trial protocol might differ systematically in a way that is related to the treatment outcome, so when data from these patients is excluded in a study, the risk of bias for that study increases. A sensitivity analysis was conducted to assess whether the effect size was sensitive to the exclusion of studies using a PP sample and demonstrated that results were robust. However, by including studies using a PP sample when the effect of interest is that of assignment to the intervention, the risk of bias might still be increased for the current analysis.

The current meta-analysis did not set eligibility criteria for publication status of the trials. Nevertheless, only published trials are represented in the analysis. Studies that produce significant, favorable, and larger results are more likely to be published (Dechartres et al., 2018; Rising et al., 2008; Scherer et al., 2018). By only including published studies, the overall effect size might be an overestimation of the true effect size, since studies reporting smaller or insignificant results that haven't been published could be missing from the analysis. Signs of publication bias were checked via Meta-Essentials (Suurmond et al., 2017). However, the

results of the generated funnel plot and of Eggers' regression indicated that the publication bias is insubstantial and could be disregarded when interpreting results.

6.5 Implications and future directions

This meta-analysis produced large effect sizes when comparing dCBTi to control conditions, demonstrating that dCBTi is effective in treating patients with insomnia disorder. Digitally delivered CBTi seems to be a viable treatment option that could make insomnia treatment more accessible to patients that currently lack proper care. The intervention format comes with many advantages. dCBTi requires far less involvement by a therapist and should therefore be a more affordable alternative to f2f CBTi (Darden et al., 2020). Moreover, the intervention can be accessed remotely, which could reduce obstacles for patients with physiological or psychological impairments, and can be more easily provided in different languages, making treatment more accessible for people who would otherwise face language barriers. Results from the current meta-analysis further indicated that fully automated dCBTi and guided dCBTi yield similar treatment effects. These findings are promising, as fully automated dCBTi demands for less resources than guided dCBTi. This implies that the therapeutic involvement might be of less importance than previously assumed. If the benefit of guidance by a therapist is insignificant, fully automated interventions could be applied on a larger scale.

However, there is demand for studies directly comparing dCBTi to f2f therapy, so that conclusions about efficacy can be drawn with more confidence. If more research were conducted comparing dCBTi to f2f CBTi, extensive meta-analyses could explore factors contributing to the effectiveness of either intervention and serve as an empirical basis for future decision making on treatment recommendations and health policies.

The results of the current meta-analysis demonstrate that participants are more likely to drop out of dCBTi than out of f2f CBTi. However, it remains unclear which factors contribute to the increased attrition. More research should be done exploring the factors contributing to treatment adherence in dCBTi, as adherence is related to greater improvements of insomnia severity (Siebmanns et al., 2020) and essential for successful treatment.

Though this systematic review aimed at including studies that assessed efficacy of dCBTi across a wide age spectrum, very few studies considered age as a moderator on treatment effect or examined the intervention effect on an exclusively young or older sample.

It is essential that these populations are specifically considered in future research, so that future health policy decisions for these groups can be based on a strong empirical basis.

There is a constant flow of new studies assessing the efficacy of dCBTi. Analyzing existing studies and exploring factors potentially contributing to the efficacy of the dCBTi might yield important insights for the development and implementation of the intervention format. This is essential if dCBTi were to be considered an eligible alternative to conventional f2f CBTi and implemented as standard treatment in the healthcare system.

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List of figures

Figure 1 <i>Illustration of random effects model and fixed effects model</i>	35
Figure 2 <i>Fourfold table for calculation of the odds ratio</i>	41
Figure 3 <i>Trial Flow Diagram</i>	44
Figure 4 <i>Forest plot of studies' effect sizes comparing dCBTi to passive and placebo controls at post-assessment</i>	50
Figure 5 <i>Regression of moderator mean age on effect size in main analysis at post-assessment</i>	55
Figure 6 <i>Funnel plot of studies included in the meta-analysis comparing effects from dCBTi to control conditions</i>	60

List of tables

Table 1 <i>Study characteristics illustrating similarity of the study population and intervention across studies.....</i>	45
Table 2 <i>Characteristics of studies included in the meta-analysis.....</i>	50
Table 3 <i>Characteristics of studies included in the meta-analysis.....</i>	56
Table 4 <i>Characteristics of studies included in the meta-analysis.....</i>	57

List of abbreviations

CBTi	Cognitive behavioral therapy for insomnia
CI	Confidence interval
CSRQ	Chronic Sleep Reduction Questionnaire
dCBTi	Digital cognitive behavioral therapy for insomnia
DSM	The Diagnostic and Statistical Manual of Disorders
EEG	Electroencephalography
f2f CBTi	Face-to-face cognitive behavioral therapy for insomnia
HSDQ	Holland Sleep Disorder Questionnaire
ICSD	International Classification system of Sleep Disorders
ISI	Insomnia Severity Index
ITT	Intention to treat
MD	Mean difference
OR	Odds ratio
PP	Per protocol
PSQI	Pittsburgh Sleep Quality Index
REM	Rapid eye movement
SCN	The suprachiasmatic nuclei
SD	Standard deviation
SE	Standard error
SMD	Standardized mean difference

Abstract (English)

Insomnia disorder is highly prevalent in the general population, yet few patients receive adequate treatment. Digitally delivered cognitive behavioral therapy for insomnia (dCBTi) could increase the appliance of the treatment by making it more accessible. Meta-analyses have already demonstrated the effectiveness of dCBTi in an adult population while research on adolescents is still scarce. Furthermore, there is a lack of meta-analyses comparing efficacy of dCBTi to face-to-face (f2f) CBTi. In this meta-analysis, the efficacy of dCBTi in reducing insomnia severity in adolescents and adults with insomnia disorder was assessed by comparing dCBTi to controls and to f2f CBTi. Furthermore, factors contributing to effectiveness of the intervention were explored. 20 studies were included in the main analysis ($N= 5836$) and four studies in the non-inferiority analysis ($N= 253$). Random effects model was applied and standardized mean difference between groups used as effect measure. dCBTi was effective in reducing insomnia severity at post-assessment ($g= 0.98$) and short-term follow-up ($g= 0.84$). Subgroup analysis revealed that fully automated and guided dCBTi produced similar effects. Results were not influenced by the type of control group and did not seem to be moderated by mean age of participants. dCBTi produced slightly inferior results than f2f CBTi, though the difference was insignificant. Calculation of the odds ratio indicated that participants are more likely to drop out of the intervention if assigned to dCBTi compared to f2f CBTi. This meta-analysis demonstrates that dCBTi is an effective treatment modality that could make insomnia treatment more accessible to patients worldwide.

Keywords: dCBT, CBTi, cognitive behavioral therapy; digital; internet; web; insomnia disorder; adolescents; meta-analysis

Abstract (Deutsch)

Schlafstörungen sind in der allgemeinen Bevölkerung weit verbreitet, Betroffene verbleiben jedoch häufig ohne angemessene Behandlung. Durch den Einsatz digital vermittelter kognitiver Verhaltenstherapie für Insomnie (dKVTi) könnten Barrieren konventioneller Therapie reduziert und der Zugang zur Behandlung vereinfacht werden. Bisher publizierte Meta-Analysen verweisen auf die Wirksamkeit dieser Interventionsform bei Erwachsenen, Wirksamkeitsnachweise zur Behandlung von Jugendlichen sind jedoch rar. Weiters fehlt es an direkten Wirksamkeitsvergleichen zwischen dKVTi und konventioneller KVTi (f2f KVTi). Diese Meta-Analyse erfasst die Wirksamkeit von dKVTi in der Behandlung von Insomnie bei Erwachsenen und Jugendlichen im Vergleich zu Kontrollbedingungen sowie verglichen mit f2f KVTi. Weiters werden potenzielle Einflussfaktoren hinsichtlich ihrer Bedeutung für die Wirksamkeit der Intervention untersucht. Die Hauptanalyse umfasst 20 Studien (N= 5836), die Nebenanalyse (dKVTi vs. f2f KVTi) beinhaltet vier Studien (N=253). Unter Anwendung des Random Effects Modells wurden standardisierte Mittelwertsunterschiede zwischen den Interventionsgruppen als Effektmaß herangezogen. dKVTi führte zu einer Reduktion der Schwere der Insomnie, sowohl zum Zeitpunkt direkt nach der Intervention ($g = 0.98$) als auch zur Follow-up-Erhebung (bis zu 6 Monaten; $g = 0.84$). Anhand einer Subgruppenanalyse wurde gezeigt, dass voll automatisierte und angeleitete dKVTi ähnliche Effekte erzielten. Weiters blieb die Stärke der Ergebnisse unabhängig von der Art der Kontrollgruppe und schienen nicht vom mittleren Alter der Teilnehmer*innen beeinflusst zu werden. dKVTi und f2f KVTi führten zu keinen signifikanten Unterschieden im Behandlungseffekt. Eine Odds Ratio zeigte jedoch, dass Teilnehmer*innen der dKVTi wahrscheinlicher die Behandlung abbrechen als Teilnehmer*innen der f2f KVTi. Dennoch zeigt diese Meta-Analyse, dass dKVTi eine effektive Intervention ist, welche zukünftig vielen Betroffenen die Behandlung von Schlafstörungen ermöglichen könnte.

Schlüsselwörter: KVTi; Kognitive Verhaltenstherapie; digital; Internet; Insomnie; Schlafstörung; Jugendliche; Meta-Analyse

Appendix

Table A1

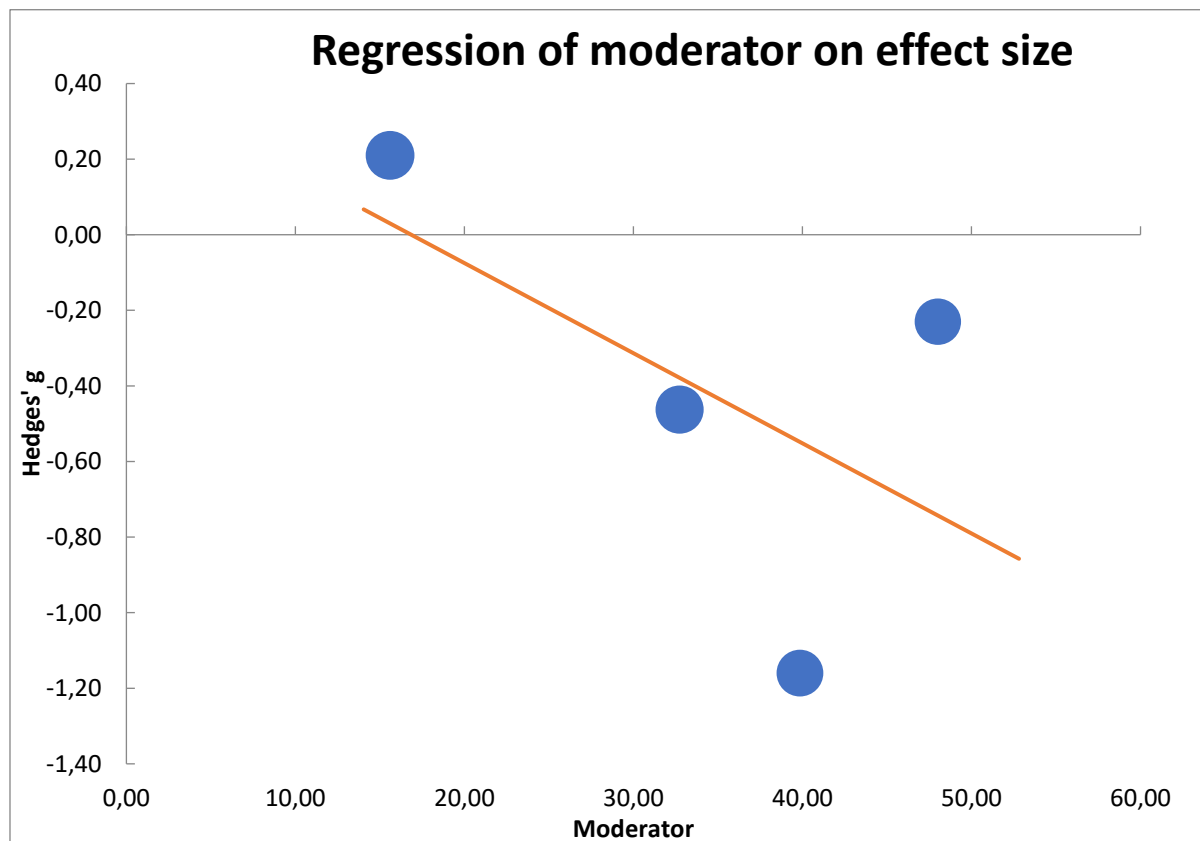
Summary statistics of non-inferiority analysis between dCBTi and f2f CBTi at post-assessment

Study name	Hedges' g	CI Lower limit	CI Upper limit
Blom et al. (2015)	-0,23	-0,81	0,34
de Bruin et al. (2018)	0,21	-0,24	0,66
Lancee et al. (2016)	-1,16	-1,73	-0,62
Taylor et al. (2017)	-0,46	-0,95	0,02
Combined	-0.4	-1.31	0.51

Note. Figure retrieved from Meta-Essentials (Suurmond et al., 2017).

Figure A1

Regression of moderator mean age on effect size in non-inferiority analysis at post-assessment



Note. Figure retrieved from Meta-Essentials (Suurmond et al., 2017).