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Is cognitive behavioral therapy more effective than pharmacotherapy for binge spectrum disorders? A systematic review and meta-analysis

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Abstract

Objectives: Binge spectrum disorders are prevalent worldwide. Psychiatric and medical comorbidities are common, and societal costs are significant. Evidence-based treatment remains underutilized. Cognitive behavioral therapy is the recommended first-line treatment, but pharmacotherapy may be easier to access.

Interventions: Meta-analytic evidence directly comparing cognitive behavioral therapy with pharmacotherapy is lacking. We aimed to compare the effects of cognitive behavioral therapy interventions with any pharmacological treatment for binge spectrum disorders. We searched PubMed, Embase, CENTRAL, ClinicalTrials.gov and reference lists for randomized controlled trials comparing cognitive behavioral therapy with any pharmacotherapy for bulimia nervosa/binge eating disorder and performed pairwise meta-analytic evaluations.

Primary Outcomes: Primary outcomes are remission and frequency of binges. Secondary outcomes are frequency of purges, response, eating disorder psychopathology, weight/body mass index, depression, anxiety, quality of life and dropouts.

Results: Eleven randomized controlled trials comparing cognitive behavioral therapy with fluoxetine/imipramine/desipramine/methylphenidate/sibutramine were identified ($N = 531$). Cognitive behavioral therapy was superior to antidepressants in terms of remission, frequency of binges and eating disorder psychopathology. There were no statistically significant differences for any of the individual cognitive behavioral therapy vs drug comparisons in terms of response/depression/anxiety/weight/quality of life/dropouts. Cognitive behavioral therapy was not superior to sibutramine/methylphenidate for the primary outcomes.

Conclusions: Data are scarce, comparisons underpowered and, considering the inherent methodological limitations of psychotherapy trials, questions arise regarding the presumed superiority of cognitive behavioral therapy. Further research is needed.

Keywords

Eating disorders, psychopharmacotherapy, CBT, drug therapy, meta-analysis

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Introduction

Bulimia nervosa (BN) and binge eating disorder (BED), commonly referred to as binge spectrum disorders (BSDs) (Treasure et al., 2020), are among the most prevalent eating disorders worldwide (Erskine and Whiteford, 2018; Van Eeden et al., 2021). BN is characterized by recurrent episodes of binge eating followed by unhelpful compensatory behaviors. These are collectively referred to as purging, including abuse of laxatives, diuretics, enemas and self-induced vomiting among others, and are aimed at controlling weight (Jain and Yilanli, 2021). BED is also characterized by frequent and recurrent episodes of binge eating, but without a regular pattern of compensatory behaviors (Iqbal and Rehman, 2021). BSDs are associated with a high risk of psychiatric and medical comorbidity (Udo and Grilo, 2019), but also functional decline, as well as increased risk of suicide and parasuicidal self-harm behavior (Perkins and Brausch, 2019; Udo et al., 2019). This often has a devastating impact on the individual and their families and is also associated with significant societal costs (Tannous et al., 2021).

Cognitive behavioral therapy (CBT) interventions are considered the first-line treatment for BSDs (Agras, 2019; Treasure et al., 2020), and they are endorsed by major guidelines (Crone et al., 2023; NICE Guideline, 2017). The theoretical basis of CBT for eating disorders relies on clinical observations and social learning theory. This framework suggests that low self-esteem and persistent negative emotions lead to dissatisfaction with body weight and shape, resulting in restricted eating, loss of control over eating and binge eating with or without compensatory behaviors (Agras and Bohon, 2021).

Interpersonal issues and life stressors often trigger binge-eating episodes. Several models and forms of delivery are available (Mulkens and Waller, 2021). CBT interventions are supported by a larger body of evidence compared to any other modality of psychotherapy and are widely used in clinical practice (Peat et al., 2017; Svaldi et al., 2019). However, access to trained therapists (Kazdin et al., 2017), the willingness of patients to engage in therapy (Thompson and Park, 2016), financial and geographical access problems and long-waiting lists (Liu et al., 2022; Thompson and Park, 2016) are important barriers to psychological treatment.

Despite CBT's historical promotion as the 'gold standard', a growing body of literature is critical of this perspective. This literature argues that claims about CBT's superiority lack thorough assessment and fail to acknowledge potential harms or iatrogenic effects (Castro Batic and Hayes, 2020; Hayes and Za'ba, 2021; Lilienfeld, 2007; Parry et al., 2016). In addition, there is increasing evidence that CBT may not be the most suitable approach and can even be potentially harmful for certain marginalized communities, such as autistic individuals (Babb et al., 2022), who are overrepresented in eating disorder populations (Huke et al., 2013).

Access to pharmacotherapy is often easier and more practical. However, medication remains a second-line option for BED (Crone et al., 2023), and only fluoxetine is Food and Drug Administration (FDA)-approved for the treatment of BN, and lisdexamfetamine for BED. Fluoxetine is recommended either as initial treatment or as an augmentation to psychotherapy when the latter alone fails to deliver the desirable clinical outcomes (Crone et al., 2023). Nevertheless, the results of recent meta-analyses indicated that fluoxetine has small clinical effects compared to other treatment options and placebo (Argyrou et al., 2023; Slade et al., 2018). It is also worth mentioning that there is an ongoing and robust debate surrounding the value and risk-to-benefit ratios associated with selective serotonin reuptake inhibitors (SSRIs), including concerns about quality of life in the long term (Almohammed et al., 2022) and conditions like Post-SSRI Sexual Dysfunction (Ben-Sheetrit et al., 2023), withdrawal syndrome (Horowitz and Taylor, 2019) and movement disorders (Revet et al., 2020).

The clinical reality is that, in many healthcare systems, demand outstrips the supply of evidence-based mental health treatment, which the recent COVID-19 pandemic has exacerbated (Zima et al., 2022). Therefore, the question of whether (and if so, which) easily accessible pharmacotherapy options for the treatment of BSDs have comparable treatment effects to CBT interventions is clinically very relevant.

Several studies have examined the effects of pharmacological treatments for BN and BED. Antidepressants and other serotonin antagonists are the most widely studied drugs for the treatment of BN (Argyrou et al., 2023), while antidepressants, antiepileptics, psychostimulants, and anti-obesity drugs have been studied for the treatment of BED (Monteleone et al., 2022).

A recent systematic meta-review (Monteleone et al., 2022) summarizes the comparisons and outcomes examined in a number of meta-analyses. Some have examined the efficacy of all forms of psychotherapy and pharmacotherapy (Bacaltchuk et al., 2001; Devlin, 1996; Fairburn and Hay, 1992; Ghaderi et al., 2018; Hilbert et al., 2019; Mcelroy et al., 2015; Mitchell et al., 1993; Peat et al., 2017; Ramacciotti et al., 2013; Slade et al., 2018; Svaldi et al., 2019; Vocks et al., 2010), but no study has directly compared the efficacy of CBT-based interventions with that of individual types of pharmacotherapy. Furthermore, it is often the case that a number of smaller and/or crossover pharmacotherapy trials are excluded from meta-analyses, especially for BN, due to the strict definition of remission that has often been utilized, i.e. 100% abstinence from symptoms for at least 2 weeks or no longer meeting the diagnostic criteria (Bacaltchuk and Hay, 2003; Williams et al., 2012). Finally, important clinical outcomes, including symptoms of anxiety and quality of life, have rarely been examined.

The present meta-analysis of randomized controlled trials (RCTs) aims to compare the effects of CBT interventions with any pharmacological treatment of BSDs for a wide range of important clinical outcomes. The goal is to address the clinical question of whether, and if so which, medication options could potentially be used as an alternative to CBT in treating these patient populations.

Materials and methods

An a priori written study protocol was published in PROSPERO (number: CRD42021230473) and is presented in Supplementary Material A. The systematic review and meta-analysis were conducted according to PRISMA (Preferred Reporting Items for Systematic reviews and Meta-Analyses) guidelines (Moher et al., 2009) (see Supplementary Material B: PRISMA checklist).

Participants and interventions

We included all RCTs that compared the effect of CBT (regardless of model or delivery format, including self-help CBT-based interventions) to any pharmacological treatment in patients with BN and/or BED, without restrictions regarding age, gender or comorbidities.

Search strategy and selection criteria

A comprehensive, systematic literature search was conducted in Embase, PubMed, Cochrane Central Register of Controlled Trials (CENTRAL) and ClinicalTrials.gov up to 15 February 2022 (last update 15 February 2023). The search strategy combined the following terms: ((OR (bulimi*) OR (binge) OR (binge*) OR (eating disorder) OR (eating disorder*)) AND ((OR (random*) OR (blind) OR (blind*) OR (clinical trial)). We also manually reviewed the reference lists of all included studies and reference lists of previously published reviews and meta-analyses.

There were no limitations in terms of language, date or publication status in the study selection process. At least two review authors worked independently to screen all the studies identified following de-duplication and assess the risk of bias using the Cochrane Risk of Bias Tool for Randomized Trials (Higgins et al., 2011). Conflicts at any stage were resolved through discussion with a third author. The first and/or corresponding authors of the original studies were contacted via email to obtain any missing data.

Outcome measures and data extraction

The primary outcomes were (1) remission, defined as a 100% reduction in bulimia/binge eating–related symptoms over a minimum of a 2-week period or as no longer meeting the relevant diagnostic criteria (including cognitive elements) (Bacaltchuk and Hay, 2003; Williams et al., 2012), and (2) frequency of binges.

Additional outcomes were (1) frequency of purges (only in bulimia trials); (2) response, defined as a reduction of at least 50% in bulimic/binge-eating episodes or according to authors' definitions (Williams et al., 2012); (3) eating disorder psychopathology measured using the Eating Disorder Inventory (EDI) or any other validated scale whose psychometric properties have been documented in a peer-reviewed journal; (4) depressive symptoms, measured by validated rating scales such as the Hamilton Depression Rating Scale (HDRS) and Beck Depression Inventory (BDI); (5) anxiety symptoms, measured by validated rating scales such as the Hamilton Anxiety Rating Scale (HAM-A); (6) weight; (7) body mass index (BMI); (8) quality of life (QoL) measured by validated rating scales such as World Health Organization Quality of Life Brief Version (WHOQOL-BREF), Quality of Life Scale (QOLS), McGill Quality of Life Questionnaire (MQOL) or health-related quality of life (HRQOL); (9) total dropouts; (10) dropouts due to side effects; and (11) the total number of patients experiencing treatment-emergent adverse effects.

When authors of the original studies used imputation methods to account for missing data, these were preferred over completers' data. In the case of crossover trials, we used the first crossover phase, if possible, to avoid the problem of carryover effects. At least two review authors independently extracted data from all included studies, and any conflicts were resolved through discussion with a third author. Missing standard deviations were estimated from *p*-values or other information, or they were substituted by the mean standard deviation of the other included studies.

Statistical analysis

We performed a pairwise meta-analysis using the Review Manager 5.4 software (The Cochrane Collaboration, 2020). We employed a random-effects model (DerSimonian and Laird, 1986), as it is usually more conservative in terms of statistical significance, although, as a disadvantage, it puts added weight onto smaller studies, which can either inflate or deflate the effect size. We therefore examined whether using a fixed-effects model would markedly affect the results in a sensitivity analysis for the primary outcomes only.

All analyses were on an intention-to-treat (ITT) basis, whenever possible. Our preferred effect size for dichotomous outcomes was the risk ratio (RR). The effect size for continuous outcomes was the weighted mean difference (MD). If data were presented in different scales, then the standardized mean difference (SMD) was calculated instead. Missing standard deviations were calculated from standard errors or estimated from confidence intervals (CIs), *t*-values or *p*-values.

Heterogeneity was investigated by inspection of the forest plots. Statistical heterogeneity was tested with the chi-square test and was quantified by calculating the I^2 statistics and its 95% CI. Potential reasons for heterogeneity were explored with subgroup analyses, but these were only conducted for the primary outcomes.

We planned several subgroup analyses considering the following a priori variables: individuals with BN vs individuals with BED; adults vs adolescents; previously treated participants vs not; participants with comorbidities vs no comorbidities; sponsored trials vs not; study duration up to 8 weeks vs longer; and treatment groups according to their main therapeutic concept. At the time the protocol was designed, the following sensitivity analyses were planned for the primary outcomes: exclusion of non-double-blind studies; exclusion of studies that did not use operationalized criteria to diagnose BN/BED; exclusion of studies that presented only completer analyses; exclusion of studies with high risk of bias; fixed effects instead of random-effects model; and exclusion of studies with imputed data.

Results

Search

We identified 11 RCTs, which met our inclusion criteria. The studies were published from 1990 to 2019 and included 531 participants in total.

Description of included studies

The PRISMA flowchart is shown in Figure S1. Table 1 presents the details of all included studies. Notably, out of the 11 included studies, 10 took place in the United States (Agras et al., 1992; Devlin et al., 2005; Grilo et al., 2005, 2014; Leitenberg et al., 1994; Quilty et al., 2019; Walsh et al., 1997) and just one in Europe (Jacobi et al., 2002) (Hamburg, Germany). A total of 319 participants (60.07% of the individuals included in the analysis) were diagnosed with BN (7 studies) (Agras et al., 1992; Goldbloom et al., 1997; Jacobi et al., 2002; Leitenberg et al., 1994; Mitchell et al., 1990; Walsh et al., 1997, 2004). A total of 212 participants (39.93%) were diagnosed with BED (Devlin et al., 2005; Grilo et al., 2005, 2014; Quilty et al., 2019) (4 studies). The participants' mean age was 31.19 years. Two RCTs (Agras et al., 1992; Leitenberg et al., 1994) compared CBT (18–22 sessions) with desipramine (100–187.5 mg/day) for 20–24 weeks among patients diagnosed with BN ($N=61$). Five RCTs (Devlin et al., 2005; Goldbloom et al., 1997; Grilo et al., 2005; Jacobi et al., 2002; Walsh et al., 2004) compared CBT (6–20 sessions) with fluoxetine (40–60 mg/day) for 16–20 weeks among patients with BN or BED ($N=239$). One RCT (Grilo et al., 2014) compared a self-help CBT intervention with sibutramine (15 mg/day) for 16 weeks among patients diagnosed with BED ($N=51$). One RCT (Quilty et al., 2019) compared CBT (12 sessions) with methylphenidate (45 mg/day) for 12 weeks among patients with BED ($N=49$). In one RCT (Walsh et al., 1997), CBT (20 sessions) was compared with desipramine (188 mg/day) or fluoxetine (55 mg/day) therapy for 16 weeks. Finally, one RCT (Mitchell et al., 1990) compared CBT group therapy (12 weeks) with imipramine hydrochloride (300 mg/day) for 12 weeks among patients diagnosed with BN ($N=78$).

Risk of bias assessment

The results of the assessment of the risk of bias summary are presented in Figure 1 and the risk of bias graph in Figure S2. Strikingly, there was a high risk of bias in performance blinding among all included studies.

Remission and frequency of binge episodes (primary outcomes)

Figure 2 presents the results for the primary outcome remission. CBT was found to be superior to fluoxetine (RR = 3.24, 95% CI = [1.46, 7.18], $p=0.004$, 2 RCTs, $N=81$, $I^2=0\%$). Comparisons of CBT with other drugs did not reveal any important differences, but only one RCT was available per comparison and sample sizes were very small, ranging from 9 to 53 patients. The pooled result for all antidepressants vs CBT favored CBT (RR = 2.24, 95% CI = [1.03, 4.87], $p=0.04$, 4 RCTs, $N=143$, $I^2=29\%$, Figure S3).

As for the frequency of binge episodes (Figure S4), CBT was found to be superior to imipramine (SMD = −0.93, 95% CI = [−1.40, −0.46], $p<0.01$, 1 RCT, $N=78$), and there was a trend for superiority of CBT over desipramine (SMD = −0.49, 95% CI = [−1.07, 0.09], $p=0.10$, 1 RCT, $N=47$), and methylphenidate (SMD = −0.56, 95% CI = [−1.13, 0.02], $p=0.06$, 1 RCT, $N=49$). The pooled result for all antidepressants vs CBT favored CBT marginally (SMD = −0.35, 95% CI = [−0.69, −0.01], $p=0.04$, 8 RCTs, $N=396$, $I^2=63\%$, Figure S5).

CBT was found to be more efficacious than imipramine for the outcomes frequency of purge (Figure 3; SMD = −0.93, 95% CI = [−1.40, −0.45], $p<0.01$, 1 RCT, $N=78$), eating disorder psychopathology (Figure S6; SMD = −1.02, 95% CI = [−1.54, −0.50], $p<0.01$, 1 RCT, $N=65$), and dropouts due to any reason (Figure S7; RR = 0.35, 95% CI = [0.15, 0.82], $p=0.02$, 1 RCT, $N=88$). When examining all antidepressants as a subgroup, CBT was found to be more efficacious only for the outcome eating disorder psychopathology (Figure S8; SMD = −0.64, 95% CI = [−0.99, −0.29], $p<0.01$, 5 RCTs, $N=239$).

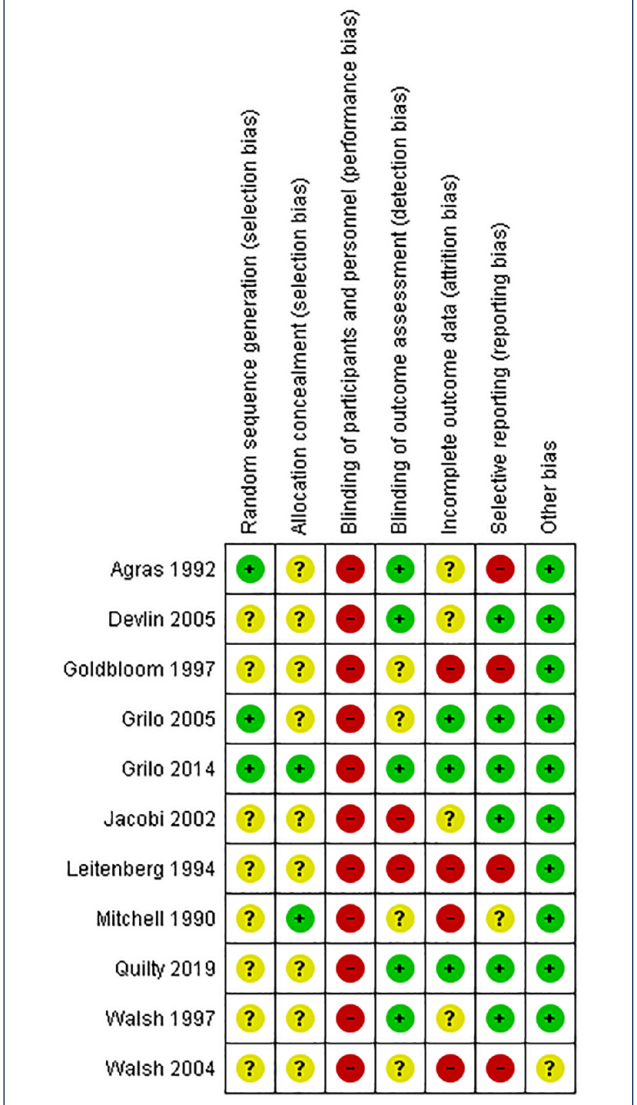
With regard to the outcome BMI, CBT had higher BMI values compared to methylphenidate (Figure S9; MD = 5.78, 95% CI = [1.37, 10.19], $p=0.01$, 1 RCT, $N=49$) and lower compared to fluoxetine (Figure S9; MD = −2.71, 95% CI = [−5.33, −0.10], $p=0.04$, 2 RCTs, $N=100$).

Table 1. Details of Included Studies.

Study ID	Study design	Treatment duration (weeks)	Mean age of participants (years)	No. randomized on CBT and number of sessions	No. randomized on pharmacotherapy, drug and mean dose per day	Main outcomes	Definition of response/remission to treatment	Eating disorder diagnostic criteria
Agras et al. (1992)	Randomized, parallel, single-blind	24	29.6	*N* = 23 18 sessions	*N* = 24 Desipramine 187.5 mg	Frequency of binge eating and purge episodes	Remission from binge eating and purge behaviors without specifying for how long	Bulimia nervosa (*DSM*-III-R)
Devlin (2005)	Randomized, parallel, double-blind[a]	20	43.0	*N* = 25 20 sessions	*N* = 32 Fluoxetine 40 mg	Frequency of binge eating episodes Weight	Remission from binge eating the previous 4 weeks	Binge eating disorder (*DSM*-IV)
Goldbloom et al. (1997)	Randomized, parallel, open	16	25.8	*N* = 14 16 sessions	*N* = 12 Fluoxetine 60 mg	Eating Disorder Examination (EDE)	Remission from binge eating and vomiting 4 weeks post treatment Response: 50% reduction of binge eating and vomiting 4 weeks post treatment	Bulimia nervosa (*DSM*-III-R)
Grilo et al. (2005)	Randomized, parallel, double-blind[a]	16	43.9	*N* = 28 16 sessions	*N* = 27 Fluoxetine 60 mg	Frequency of binge eating episodes	No binge eating episode the last 28 days	Binge eating disorder (*DSM*-IV)
Grilo et al. (2014)	Randomized, parallel, double-blind[a]	16	43.4	*N* = 25 Self-help CBT book assisted by primary care physicians	*N* = 26 Sibutramine 15 mg	Frequency of binge eating episodes Weight loss	No binge eating episode the last month based on EDE-I	Binge eating disorder (*DSM*-5)
Jacobi et al. (2002)	Randomized, parallel, open	16	26.0	*N* = 19 20 sessions	*N* = 16 Fluoxetine 40 mg	Frequency of binge eating and purge episodes EDI and Three Factor Eating Questionnaire (TFEQ) score	Remission from binge eating and purge behaviors without specifying for how long	Bulimia nervosa (*DSM*-III-R)
Leitenberg et al. (1994)	Randomized, parallel, open	20	26.7	*N* = 7 22 sessions	*N* = 7 Desipramine 100 mg	Frequency of binge eating and purge episodes	Remission from vomiting without specifying for how long	Bulimia nervosa (*DSM*-III-R)
Mitchell et al. (1990)	Randomized, parallel, double-blind[a]	12	23.5	*N* = 33 Intensive group CBT 12 weeks	*N* = 45 Imipramine Hydrochloride 300 mg	Frequency of binge eating and purge episodes (self-induced vomiting)	Remission from bulimic episodes: no episodes for the last 2 weeks	Bulimia nervosa (*DSM*-III)
Quilty et al. (2019)	Randomized, parallel, open	12	32.8	*N* = 27 12 sessions	*N* = 22 Methylphenidate 45 mg	Frequency of binge eating episodes	No binge eating episode for the previous 4 weeks of treatment	Binge eating disorder (*DSM*-5)
Walsh et al. (1997)	Randomized, parallel, double-blind[a]	16	25.0	*N* = 25 20 sessions	*N* = 28 Desipramine and then Fluoxetine (for participants that desipramine was not effective) 188 mg desipramine/55 mg fluoxetine	Frequency of binge eating and vomiting	Remission from binge eating episodes and/or purge behaviors the two last weeks	Bulimia nervosa (*DSM*-III-R)
Walsh et al. (2004)	Randomized, parallel, single-blind	16	30.6	*N* = 25 6–8 sessions (2 optional), guided self-help CBT	*N* = 20 Fluoxetine 60 mg	Frequency of binge eating and vomiting	Remission from binge eating/purging episodes for the previous 4 weeks of treatment	Bulimia nervosa (*DSM*-IV)

CBT: cognitive behavioral therapy; *DSM: Diagnostic and Statistical Manual of Mental Disorders*; EDI: Eating Disorder Inventory.
[a]Double-blind refers to drug assignment and management only.

Figure 1. Risk of bias summary.



There were no statistically significant differences for any of the individual CBT vs drug comparisons in terms of response (Figure S10; RR=1.47, 95% CI=[0.98, 2.21], p=0.06, 5 RCTs, N=170), depression (Figure S11; MD=−0.70, 95% CI=[−2.38, 0.98], p=0.41, 8 RCTs, N=388), anxiety (Figure S12; MD=−0.07, 95% CI=[−0.62, 0.48], p=0.81, 2 RCTs, N=131), weight (Figure S13; SMD=0.15, 95% CI=[−0.30, 0.60], p=0.50, 4 RCTs, N=179), quality of life (Figure S14; MD=0.57, 95% CI=[−0.16, 1.30], p=0.13, 1 RCT, N=49), and dropouts due to side effects (Figure S15; RR=0.37, 95% CI=[0.03, 5.38], p=0.47, 2 RCTs, N=113).

Publication bias

Data from fewer than 10 studies were available for most of the outcomes. When fewer than 10 studies are included in a meta-analysis, asymmetry testing for funnel plots should not be employed (Egger et al., 1997; Higgins et al., 2019). As a result, we were unable to use funnel plots for the outcomes of remission, frequency of purges, response, eating disorder psychopathology, depressive and anxiety symptoms, weight, BMI, quality of life, total dropouts and dropouts due to side effects. However, the meta-analysis included 10 studies examining the outcome of frequency of binges, and in this case, the funnel plot was symmetrical (Figure 4).

Discussion

Summary and contributions

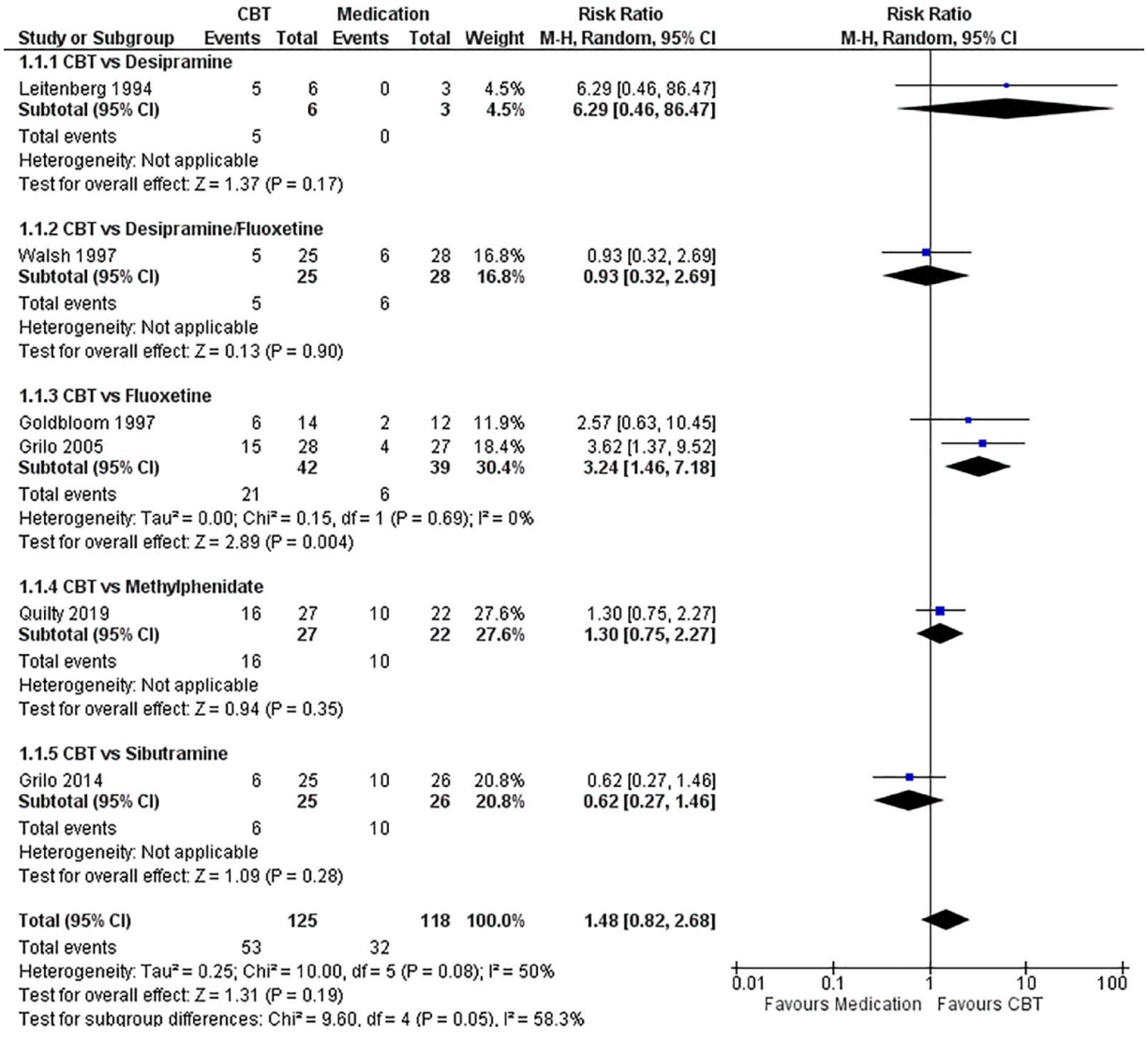
CBT is considered the first-line treatment option for BSDs (Treasure et al., 2020). Nevertheless, this treatment modality is underutilized and may be difficult to access in everyday clinical practice. Exploring other therapeutic alternatives is important. Thus, we attempted to answer the important clinical question as to whether (and if so, which) easily accessible pharmacotherapy options could potentially be equally effective to CBT for the treatment of BSDs.

Eleven RCTs published from 1990 to 2019 were identified, with a total of 531 participants. They compared CBT with five drugs, namely fluoxetine, imipramine, desipramine, methylphenidate and sibutramine. Our findings do not allow us to recommend specific drugs as an alternative to CBT, as few statistically significant differences were identified in the examined outcomes.

Remission, defined as a 100% reduction in bulimia/binge eating–related symptoms over a minimum of a 2-week period or as no longer meeting the relevant diagnostic criteria (Bacaltchuk and Hay, 2003; Williams et al., 2012), was examined in 6 out of the 11 included RCTs. CBT was superior to fluoxetine (dose range, 40–60 mg) and the pooled data for antidepressants, but few studies were included, and the sample sizes were small.

As full remission is often not achieved or maintained in the long term (Smink et al., 2013; Treasure et al., 2020), response to treatment (also referred to as partial remission) is an outcome that perhaps resonates better with the clinician and the service-user. We expected that different trials would use different definitions of response. However, it has been shown that, as long as relative measures of risk are applied, meta-analytic results do not differ significantly depending on the exact cut-off applied (Furukawa et al., 2011). We prioritized using the definition of response as a reduction of at least 50% in bulimic/binge-eating episodes (Williams et al., 2012), where data were available. When a variety of definitions were provided and this criterion was not met, we used the authors' definitions of response, selecting the strictest. Although no significant difference between CBT and antidepressants was shown, there was a trend in favor of CBT (response rates of 44% vs 29%).

Figure 2. Remission.



However, it is worth noting that only desipramine and fluoxetine were examined among antidepressants, and the availability of data was very limited.

Reduction of frequency of binges is another commonly used outcome measure. Our findings indicated a superiority for CBT over imipramine and over antidepressants in general, albeit with a relatively small effect size (SMD = −0.35, 95% CI = [−0.69, −0.01], 8 RCTs, N = 396). This finding is in line with previously published work comparing all forms of psychotherapy to pharmacotherapy (Svaldi et al., 2019; Vocks et al., 2010). The clinical significance of the frequency of binges, however, remains controversial as it does not seem to correlate with long-term treatment outcomes such as weight loss (Delinsky et al., 2006), and over-valuation of weight/shape and body dissatisfaction are perhaps better clinical indicators of the severity of illness (Grilo et al., 2015; Wilson and Sysko, 2009) and predictors of suicidality (Perkins and Brausch, 2019; Rufino et al., 2018).

Purging behavior is associated with significant medical comorbidities and healthcare utilization in the short term and long term (Mehler et al., 2015). We found that CBT was superior to imipramine and tended to be superior to desipramine. However, no significant differences were found between CBT and fluoxetine or CBT vs antidepressants overall.

Severity of psychopathology is an important indicator of illness severity and functional decline (Dahlgren et al., 2017). Therefore, this outcome is clinically relevant. We found that CBT was superior to imipramine and antidepressants overall for improving psychopathology, as measured by the Eating Disorder Examination Global Score, with a medium to large effect size (SMD = −0.64, 95% CI = [−0.99,

Figure 3. Frequency of purges.

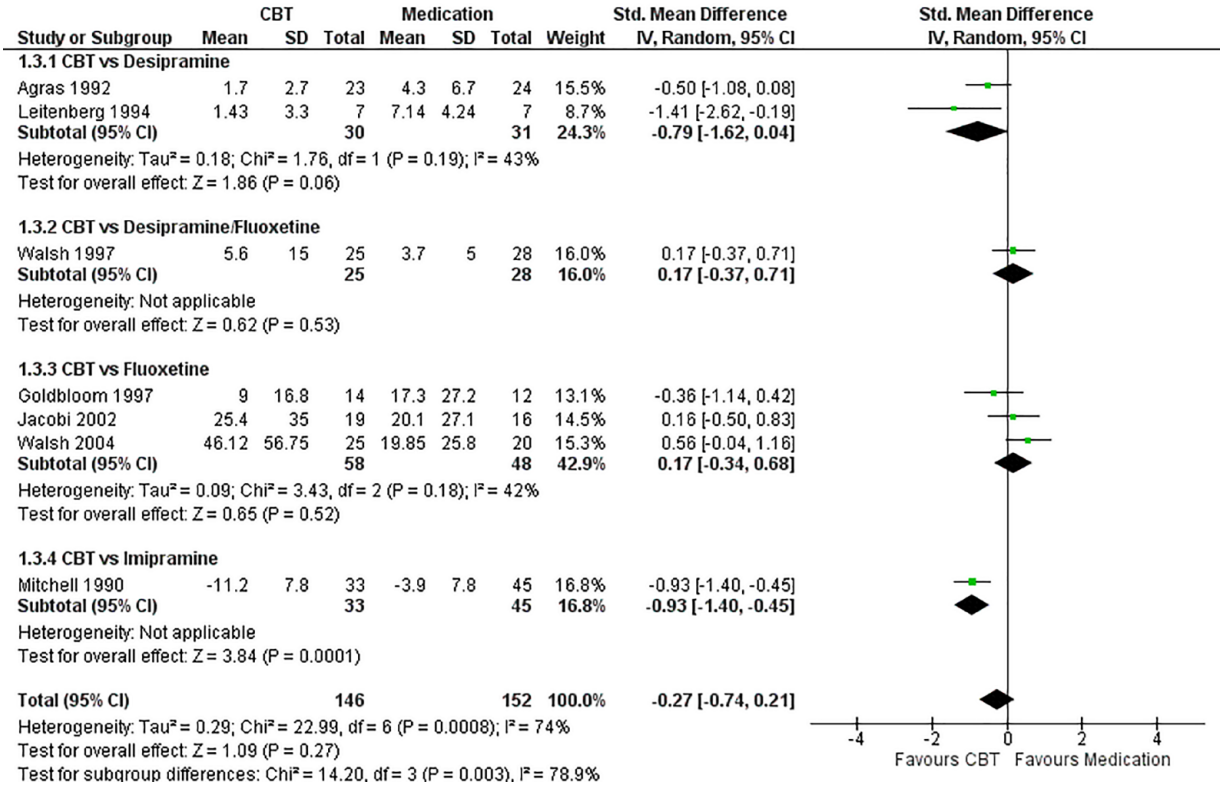
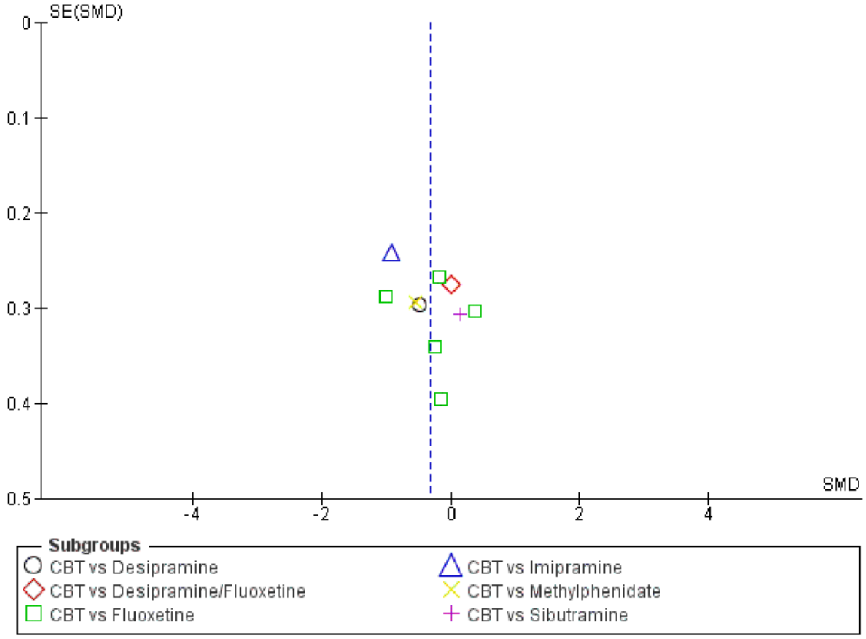


Figure 4. Frequency of binge episodes: funnel plot.



−0.29], 5 RCTs, $N=239$). This finding supports the recommendations offered by major guidelines (Crone et al., 2023; NICE Guideline, 2017). Notably, other less popular treatment options, including methylphenidate and sibutramine, were found to be equally efficacious to CBT, but usable data were only available from one RCT per comparison. Further research into these treatment options is therefore needed.

Weight loss and BMI represent clinically important outcomes consistently reported in most RCTs. BSDs often co-occur with obesity (30–45%) and its associated metabolic consequences, well-known predictors of high cardiovascular risk and overall mortality (da Luz et al., 2018; Hay et al., 2015; Kessler et al., 2013). Furthermore, body weight, body shape and attitudes toward these factors constitute major precipitating and perpetuating elements in BSDs (Treasure et al., 2020). Obesity or being overweight plays a central role in perpetuating body uneasiness (Rotella et al., 2013), although other factors, such as relentless body shaming and the prevalence of a patriarchal diet culture within Western society, can also significantly contribute to body uneasiness. Individuals with higher body weight affected by eating disorders often fall victim to systemic discrimination related to their body shape, including weight stigma (Lawrence et al., 2022). Regrettably, weight stigma is prevalent even within the medical profession, resulting in individuals with higher weight experiencing medical gaslighting and trauma, highlighting the urgent need for substantial changes in healthcare and beyond (Batterham, 2022; Talumaa et al., 2022). Collectively, these factors likely contribute to increased stress and psychological distress. Unfortunately, the evidence supporting the efficacy of CBT for weight management in overweight/obese individuals with BSDs is weak (Palavras et al., 2017), and our analyses, with only a limited number of RCTs, did not permit any meaningful interpretation of the results.

Overall, our research review holds significant importance in advancing the field of eating disorders for several compelling reasons. First, we provide a thorough and up-to-date analysis of all available evidence and incorporate a greater number of studies, enhancing the depth and relevance of our research. Second, our review expands its scope to encompass patients with BED, a relatively recently recognized diagnosis, thereby making our findings more pertinent and applicable to a broader population of individuals seeking treatment. Significantly, our study challenges the conventional perception that CBT stands as the undisputed gold standard for treating eating disorders, particularly in light of limited supporting evidence. By advocating for a wider range of treatment options, we aim to stimulate a shift in clinical practice and the development of local guidelines, potentially reducing the underutilization of pharmacotherapy in suitable cases, while also inspiring further primary clinical research in the field.

Limitations

This study has several limitations. First, and perhaps more importantly, a very small number of trials and consequently participants were included in the present meta-analysis. This did not allow us to perform the a priori planned subgroup and sensitivity analyses. Moreover, single agent results (with the possible exception of fluoxetine) are difficult to interpret adequately since many analyses included only one study.

According to Trikalinos et al., effect sizes are markedly changeable when less than 1000 participants have been included in a psychopharmacotherapy meta-analysis, but they seem to stabilize after this threshold has been reached (Trikalinos et al., 2004). In our work, none of the comparisons reached this threshold, and all sample sizes were minimal (ranging from 14 to 78 participants). This renders the derived evidence uncertain and does not allow for a definitive clinical interpretation.

In addition, the absence of several newer antidepressants and novel agents, such as lisdexamfetamine (which received FDA approval for BED in 2015), is noteworthy. Since 2005, there has been no recent trial comparing CBT to pharmacotherapy. Conducting such trials, particularly involving these novel agents, has become imperative to effectively inform clinical practice.

Moreover, blinding in psychotherapeutic interventions is always problematic, leading to performance and detection biases (Juul et al., 2020), which are strikingly present in all studies included in our meta-analysis. In addition, a significant degree of variation was evident in the delivery methods and dosages of CBT. This spectrum encompassed interventions ranging from 6 to 22 sessions and from guided self-help to group CBT and individual therapy. Addressing the placebo effect also presents challenges; factors such as time spent with the patient, empathy, and the therapeutic relationship hold substantial influence over treatment outcomes (Enck and Zipfel, 2019). In contrast, five of the identified RCTs employed a placebo-controlled double-blind design specifically for drug assignment and management. All these diverse variables increase the risk of biased treatment estimates and may, to some extent, account for the identified effects.

Another limitation is that we decided to group BN and BED together. There are distinct differences between the two disorders, highlighted across the major classification systems (Claudino et al., 2019; Smith et al., 2017). However, BED and BN do share the majority of their biological, psychological, psychosocial and behavioral etiological factors (Treasure et al., 2020), and there are compelling arguments for a spectrum model (Brooks et al., 2012; Treasure et al., 2020). Furthermore, there are many similarities in terms of psychopathology, physical findings and their effects on functioning and quality of life, and there is a significant overlap in terms of treatment goals and

approaches (Treasure et al., 2020), which we believe are reflected in the outcomes examined in this work.

Future work

Our findings only reflect short-term treatment effects and do not provide any information regarding medium- and long-term efficacy, tolerability and acceptability. Evaluating long-term efficacy necessitates extended follow-up periods, but practical challenges such as limited resources and participant attrition often hinder their implementation, explaining the scarcity of available data. This scarcity extends across various mental health conditions, where limited and sometimes contradictory evidence regarding the long-term effects of CBT exists (Cuijpers et al., 2023; Hofmann et al., 2012; Sharma et al., 2021; Van Dis et al., 2020). However, this is a significant consideration given that many mental health conditions typically follow a chronic course.

For individuals with BSDs, there is a limited body of evidence supporting the medium- and long-term efficacy of CBT in maintaining remission (Agras, 1997; Carter and Fairburn, 1998; Wilfley et al., 2002). Similarly, the maintenance of therapeutic benefits in pharmacotherapy trials has rarely been examined. A relevant published placebo-controlled trial (Stunkard et al., 1996) indicates a high risk of relapse in terms of binge episodes following the discontinuation of medication at the 4-month follow-up among patients with BED. The long-term treatment outcomes of CBT compared to pharmacotherapy for BSDs remain poorly understood, underscoring the pressing need for further research in this critical area, which represents a major clinical priority.

Conclusion

Few RCTs have investigated the efficacy and safety of CBT interventions compared to any form of pharmacotherapy for the treatment of BSDs. Only a select few antidepressants, such as fluoxetine, imipramine and desipramine, have been examined. Our results are derived from a small number of participants per individual comparison, often relying on just one RCT.

Perhaps the most important finding of this work is that, due to the underpowered comparisons and the inherent methodological limitations of psychotherapy trials, more head-to-head trials comparing CBT with monotherapy drug interventions are needed to establish the superiority of one option over the other. Further research into pharmacotherapy, especially exploring newer antidepressants and novel agents, is warranted. In addition, combination trials examining pharmacotherapy options as an augmentation to CBT are lacking and would be a valuable addition to eating disorders research and clinical practice.

Author Contributions

M.T.S. and M.C. contributed to study conception and design; N.M., A.A., E.M., D.R.B., E.T. and Z.A.P. contributed to data collection; M.T.S., N.M. and A.S.L. contributed to analysis and interpretation of results; M.T.S. and A.S.L. contributed to draft manuscript preparation; N.C., G.P. and M.C. contributed to writing review and editing. All authors reviewed the results and approved the final version of the manuscript. All authors have read and agreed to the published version of the manuscript.

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References

Agras WS (1997) Pharmacotherapy of bulimia nervosa and binge eating disorder: Long-term outcomes. *Psychopharmacology Bulletin* 33: 433–436.

Agras WS (2019) Cognitive behavior therapy for the eating disorders. *Psychiatric Clinics of North America* 42: 169–179.

Agras WS and Bohon C (2021) Cognitive behavioral therapy for the eating disorders. *Annual Review of Clinical Psychology* 17: 417–438.

Agras WS, Rossiter EM, Arnow B, et al. (1992) Pharmacologic and cognitive-behavioral treatment for bulimia nervosa: A controlled comparison. *The American Journal of Psychiatry* 149: 82–87.

Almohammed OA, Alsalem AA, Almangour AA, et al. (2022) Antidepressants and health-related quality of life (HRQoL) for patients with depression: Analysis of the medical expenditure panel survey from the United States. *PLoS ONE* 17: 1–14.

Argyrou A, Lappas AS, Bakaloudi DR, et al. (2023) Pharmacotherapy compared to placebo for people with bulimia nervosa: A systematic review and meta-analysis. *Psychiatry Research* 327: 115357.

Babb C, Brede J, Jones CRG, et al. (2022) A comparison of the eating disorder service experiences of autistic and non-autistic women in the UK. *European Eating Disorders Review* 30: 616–627.

Bacaltchuk J and Hay PP (2003) Antidepressants versus placebo for people with bulimia nervosa. *Cochrane Database of Systematic Reviews* 4: CD003391.

Bacaltchuk J, Hay P and Trefiglio R (2001) Antidepressants versus psychological treatments and their combination for bulimia nervosa. *The Cochrane Database of Systematic Reviews* 4: CD003385.

Batterham RL (2022) Weight stigma in healthcare settings is detrimental to health and must be eradicated. *Nature Reviews Endocrinology* 18: 387–388.

Ben-Sheetrit J, Hermon Y, Birkenfeld S, et al. (2023) Estimating the risk of irreversible post-SSRI sexual dysfunction (PSSD) due to serotonergic antidepressants. *Annals of General Psychiatry* 22: 15.

Brooks SJ, Rask-Andersen M, Benedict C, et al. (2012) A debate on current eating disorder diagnoses in light of neurobiological findings: Is it time for a spectrum model? *BMC Psychiatry* 12: 76.

Carter JC and Fairburn CG (1998) Cognitive-behavioral self-help for binge eating disorder: A controlled effectiveness study. *Journal of Consulting and Clinical Psychology* 66: 616–623.

Castro Batic B and Hayes D (2020) Exploring harm in psychotherapy: Perspectives of clinicians working with children and young people. *Counselling and Psychotherapy Research* 20: 647–656.

Claudino AM, Pike KM, Hay P, et al. (2019) The classification of feeding and eating disorders in the ICD-11: Results of a field study comparing proposed ICD-11 guidelines with existing ICD-10 guidelines. *BMC Medicine* 17: 93.

Crone C, Fochtmann LJ, Attia E, et al. (2023) The American Psychiatric Association practice guideline for the treatment of patients with eating disorders. *The American Journal of Psychiatry* 180: 167–171.

Cuijpers P, Miguel C, Harrer M, et al. (2023) Cognitive behavior therapy vs. control conditions, other psychotherapies, pharmacotherapies and combined treatment for depression: A comprehensive meta-analysis including 409 trials with 52,702 patients. *World Psychiatry* 22: 105–115.

da Luz FQ, Hay P, Touyz S, et al. (2018) Obesity with comorbid eating disorders: Associated health risks and treatment approaches. *Nutrients* 10: 829.

Dahlgren CL, Stedal K and Rø Ø (2017) Eating Disorder Examination Questionnaire (EDE-Q) and Clinical Impairment Assessment (CIA): Clinical norms and functional impairment in male and female adults with eating disorders. *Nordic Journal of Psychiatry* 71: 256–261.

Delinsky SS, Latner JD and Wilson GT (2006) Binge eating and weight loss in a self-help behavior modification program. *Obesity* 14: 1244–1249.

DerSimonian R and Laird N (1986) Meta-analysis in clinical trials. *Controlled Clinical Trials* 7: 177–188.

Devlin MJ (1996) Assessment and treatment of binge-eating disorder. *Eating Disorders* 19: 761–772.

Devlin MJ, Goldfein JA, Petkova E, et al. (2005) Cognitive behavioral therapy and fluoxetine as adjuncts to group behavioral therapy for binge eating disorder. *Obesity Research* 13: 1077–1088.

Egger M, Smith GD, Schneider M, et al. (1997) Bias in meta-analysis detected by a simple, graphical test. *BMJ* 315: 629–634.

Enck P and Zipfel S (2019) Placebo effects in psychotherapy: A framework. *Frontiers in Psychiatry* 10: 456.

Erskine HE and Whiteford HA (2018) Epidemiology of binge eating disorder. *Current Opinion in Psychiatry* 31: 462–470.

Fairburn CG and Hay PJ (1992) The treatment of bulimia nervosa. *Annals of Medicine* 24: 297–302.

Furukawa TA, Akechi T, Wagenpfeil S, et al. (2011) Relative indices of treatment effect may be constant across different definitions of response in schizophrenia trials. *Schizophrenia Research* 126: 212–219.

Ghaderi A, Odeberg J, Gustafsson S, et al. (2018) Psychological, pharmacological, and combined treatments for binge eating disorder: A systematic review and meta- analysis. *Peer Journal* 6: e5113.

Goldbloom DS, Olmsted M, Davis R, et al. (1997) A randomized controlled trial of fluoxetine and cognitive behavioral therapy for bulimia nervosa: Short-term outcome. *Behaviour Research and Therapy* 35: 803–811.

Grilo CM, Masheb RM and Wilson GT (2005) Efficacy of cognitive behavioral therapy and fluoxetine for the treatment of binge eating disorder: A randomized double-blind placebo-controlled comparison. *Biological Psychiatry* 57: 301–309.

Grilo CM, Masheb RM, White MA, et al. (2014) Treatment of binge eating disorder in racially and ethnically diverse obese patients in primary care: Randomized placebo-controlled clinical trial of self-help and medication. *Behaviour Research and Therapy* 58: 1–9.

Grilo CM, Ivezaj V and White MA (2015) Evaluation of the DSM-5 severity indicator for binge eating disorder in a community sample. *Behaviour Research and Therapy* 66: 72–76.

Hay P, Girosi F and Mond J (2015) Prevalence and sociodemographic correlates of DSM-5 eating disorders in the Australian population. *Journal of Eating Disorders* 3: 19.

Hayes D and Za'ba N (2021) What metrics of harm are being captured in clinical trials involving talking treatments for young people? A systematic review of registered studies on the ISRCTN. *Counselling and Psychotherapy Research* 22: 108–129.

Higgins J, Thomas J, Chandler J, et al. (2019) *Cochrane Handbook for Systematic Reviews of Interventions*, 2nd Edition. Chichester: John Wiley & Sons.

Higgins JPT, Altman DG, Gøtzsche PC, et al. (2011) The Cochrane collaboration's tool for assessing risk of bias in randomised trials. *BMJ* 343: d5928.

Hilbert A, Petroff D, Herpertz S, et al. (2019) Meta-analysis of the efficacy of psychological and medical treatments for binge-eating disorder. *Journal of Consulting and Clinical Psychology* 87: 91–105.

Hofmann SG, Asnaani A, Vonk IJJ, et al. (2012) The efficacy of cognitive behavioral therapy: A review of meta-analyses. *Cognitive Therapy and Research* 36: 427–440.

Horowitz MA and Taylor D (2019) Tapering of SSRI treatment to mitigate withdrawal symptoms. *The Lancet Psychiatry* 6: 538–546.

Huke V, Turk J, Saeidi S, et al. (2013) Autism spectrum disorders in eating disorder populations: A systematic review. *European Eating Disorders Review* 21: 345–351.

Iqbal A and Rehman A (2021) *Binge Eating Disorder*. St. Petersburg, FL: StatPearls Publishing.

Jacobi C, Dahme B and Dittmann R (2002) Cognitive-behavioural, fluoxetine and combined treatment for bulimia nervosa: Short- and long-term results. *European Eating Disorders Review* 10: 179–198.

Jain A and Yilanli M (2021) *Bulimia Nervosa*. St. Petersburg, FL: StatPearls Publishing.

Juul S, Gluud C, Simonsen S, et al. (2020) Blinding in randomised clinical trials of psychological interventions: A retrospective study of published trial reports. *BMJ Evidence-Based Medicine* 26: 109.

Kazdin AE, Fitzsimmons-Craft EE and Wilfley DE (2017) Addressing critical gaps in the treatment of eating disorders. *International Journal of Eating Disorders* 50: 170–189.

Kessler RC, Berglund PA, Chiu WT, et al. (2013) The prevalence and correlates of binge eating disorder in the World Health Organization World Mental Health Surveys. *Biological Psychiatry* 73: 904–914.

Lawrence BJ, de la Piedad Garcia X, Kite J, et al. (2022) Weight stigma in Australia: A public health call to action. *Public Health Research and Practice* 32: 3–6.

Leitenberg H, Rosen JC, Wolf J, et al. (1994) Comparison of cognitive-behavior therapy and desipramine in the treatment of bulimia nervosa. *Behaviour Research and Therapy* 32: 37–45.

Lilienfeld SO (2007) Psychological treatments that cause harm. *Perspectives on Psychological Science* 2: 53–70.

Liu L, Hay P and Conti J (2022) Perspectives on barriers to treatment engagement of people with eating disorder symptoms who have not undergone treatment: A qualitative study. *BMC Psychiatry* 22: 239.

Mcelroy SL, Guerdjikova AI, Mori N, et al. (2015) Overview of the treatment of binge eating disorder. *CNS Spectrums* 20: 546–556.

Mehler PS, Krantz MJ and Sachs KV (2015) Treatments of medical complications of anorexia nervosa and bulimia nervosa. *Journal of Eating Disorders* 3: 15.

Mitchell JE, Pyle RL, Eckert ED, et al. (1990) A comparison study of antidepressants and structured intensive group psychotherapy in the treatment of bulimia nervosa. *Archives of General Psychiatry* 47: 149–157.

Mitchell JE, Raymond N and Specker S (1993) A review of the controlled trials of pharmacotherapy and psychotherapy in the treatment

of bulimia nervosa. *International Journal of Eating Disorders* 14: 229–247.
Moher D, Liberati A, Tetzlaff J, et al. (2009) Preferred reporting items for systematic reviews and meta-analyses: The PRISMA statement. *BMJ* 339: 332–336.
Monteleone AM, Pellegrino F, Croatto G, et al. (2022) Treatment of eating disorders: A systematic meta-review of meta-analyses and network meta-analyses. *Neuroscience & Biobehavioral Reviews* 142: 104857.
Mulkens S and Waller G (2021) New developments in cognitive-behavioural therapy for eating disorders (CBT-ED). *Current Opinion in Psychiatry* 34: 576–583.
NICE Guideline (2017) *Eating Disorders: Recognition and Treatment*. London: NICE.
Palavras MA, Hay P, Filho CA, et al. (2017) The efficacy of psychological therapies in reducing weight and binge eating in people with bulimia nervosa and binge eating disorder who are overweight or obese—A critical synthesis and meta-analyses. *Nutrients* 9: 299.
Parry GD, Crawford MJ and Duggan C (2016) Iatrogenic harm from psychological therapies-time to move on. *British Journal of Psychiatry* 208: 210–212.
Peat CM, Berkman ND, Lohr KN, et al. (2017) Comparative effectiveness of treatments for binge-eating disorder: Systematic review and network meta-analysis. *European Eating Disorders Review* 25: 317–328.
Perkins NM and Brausch AM (2019) Body dissatisfaction and symptoms of bulimia nervosa prospectively predict suicide ideation in adolescents. *International Journal of Eating Disorders* 52: 941–949.
Quilty LC, Allen TA, Davis C, et al. (2019) A randomized comparison of long acting methylphenidate and cognitive behavioral therapy in the treatment of binge eating disorder. *Psychiatry Research* 273: 467–474.
Ramacciotti CE, Coli E, Marazziti D, et al. (2013) Therapeutic options for binge eating disorder. *Eating and Weight Disorders* 18: 3–9.
Revet A, Revet A, Revet A, et al. (2020) Antidepressants and movement disorders: A postmarketing study in the world pharmacovigilance database. *BMC Psychiatry* 20: 308.
Rotella F, Castellini G, Montanelli L, et al. (2013) Comparison between normal-weight and overweight bulimic patients. *Eating and Weight Disorders* 18: 389–393.
Rufino KA, Viswanath H, Wagner R, et al. (2018) Body dissatisfaction and suicidal ideation among psychiatric inpatients with eating disorders. *Comprehensive Psychiatry* 84: 22–25.
Sharma S, Hucker A, Matthews T, et al. (2021) Cognitive behavioural therapy for anxiety in children and young people on the autism spectrum: A systematic review and meta-analysis. *BMC Psychology* 9: 151.
Slade E, Keeney E, Mavranezouli I, et al. (2018) Treatments for bulimia nervosa: A network meta-analysis. *Psychological Medicine* 48: 2629–2636.
Smink FR, Van Hoeken D and Hoek HW (2013) Epidemiology, course, and outcome of eating disorders. *Current Opinion in Psychiatry* 26: 543–548.
Smith KE, Ellison JM, Crosby RD, et al. (2017) The validity of DSM-5 severity specifiers for anorexia nervosa, bulimia nervosa, and binge-eating disorder. *International Journal of Eating Disorders* 50: 1109–1113.
Stunkard AJ, Berkowitz RI, Wadden T, et al. (1996) Binge eating disorder and the night-eating syndrome. *International Journal of Obesity* 20: 1–6.
Svaldi J, Schmitz F, Baur J, et al. (2019) Efficacy of psychotherapies and pharmacotherapies for bulimia nervosa. *Psychological Medicine* 49: 898–910.
Talumaa B, Brown A, Batterham RL, et al. (2022) Effective strategies in ending weight stigma in healthcare. *Obesity Reviews* 23: e13494.
Tannous WK, Hay P, Girosi F, et al. (2021) The economic cost of bulimia nervosa and binge eating disorder: A population-based study. *Psychological Medicine* 52: 3924–3938.
The Cochrane Collaboration (2020) Review Manager (RevMan). 5.4. Available at: https://training.cochrane.org/online-learning/core-software-cochrane-reviews/revman/revman-non-cochrane-reviews (accessed 10 January 2023).
Thompson C and Park S (2016) Barriers to access and utilization of eating disorder treatment among women. *Archives of Women's Mental Health* 19: 753–760.
Treasure J, Duarte TA and Schmidt U (2020) Eating disorders. *The Lancet* 395: 899–911.
Trikalinos TA, Churchill R, Ferri M, et al. (2004) Effect sizes in cumulative meta-analyses of mental health randomized trials evolved over time. *Journal of Clinical Epidemiology* 57: 1124–1130.
Udo T and Grilo CM (2019) Psychiatric and medical correlates of DSM-5 eating disorders in a nationally representative sample of adults in the United States. *International Journal of Eating Disorders* 52: 42–50.
Udo T, Bitley S and Grilo CM (2019) Suicide attempts in US adults with lifetime DSM-5 eating disorders. *BMC Medicine* 17: 120.
Van Dis EAM, Van Veen SC, Hagenaars MA, et al. (2020) Long-term outcomes of cognitive behavioral therapy for anxiety-related disorders: A systematic review and meta-analysis. *JAMA Psychiatry* 77: 265–273.
Van Eeden AE, Van Hoeken D and Hoek HW (2021) Incidence, prevalence and mortality of anorexia nervosa and bulimia nervosa. *Current Opinion in Psychiatry* 34: 515–524.
Vocks S, Tuschen-Caffier B, Pietrowsky R, et al. (2010) Meta-analysis of the effectiveness of psychological and pharmacological treatments for binge eating disorder. *International Journal of Eating Disorders* 43: 205–217.
Walsh BT, Wilson GT, Loeb KL, et al. (1997) Medication and psychotherapy in the treatment of bulimia nervosa. *The American Journal of Psychiatry* 154: 523–531.
Walsh BT, Fairburn CG, Mickley D, et al. (2004) Treatment of bulimia nervosa in a primary care setting. *The American Journal of Psychiatry* 161: 556–561.
Wilfley DE, Welch RR, Stein RI, et al. (2002) A randomized comparison of group cognitive-behavioral therapy and group interpersonal psychotherapy for the treatment of overweight individuals with binge-eating disorder. *Archives of General Psychiatry* 59: 713–721.
Williams SE, Watts TK and Wade TD (2012) A review of the definitions of outcome used in the treatment of bulimia nervosa. *Clinical Psychology Review* 32: 292–300.
Wilson GT and Sysko R (2009) Frequency of binge eating episodes in bulimia nervosa and binge eating disorder: Diagnostic considerations. *International Journal of Eating Disorders* 42: 603–610.
Zima BT, Devaskar SU, Shah S, et al. (2022) Imperative to accelerate research aligning real-time clinical demand with mental health supply. *Pediatric Research* 92: 917–920.