Full title

Predictors, Moderators, and Mediators of Treatment Outcome Following Manualised

Cognitive-Behavioural Therapy for Eating Disorders: A Systematic Review

Short title

Predictors, Moderators and Mediators of Outcome for Eating Disorders

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Abstract

This systematic review synthesised the literature on predictors, moderators, and mediators of outcome following Fairburn's CBT for eating disorders. Sixty-five articles were included. The relationship between individual variables and outcome were synthesised separately across diagnoses and treatment format. Early change was found to be a consistent mediator of better outcomes across all eating disorders. Moderators were mostly tested in binge eating disorder, and most moderators did not affect cognitive-behavioural treatment outcome relative to other treatments. No consistent predictors emerged. Findings suggest that it is unclear how and for whom this treatment works. More research testing mediators and moderators is needed, and variables selected for analyses need to be empirically and theoretically-driven. Future recommendations include the need for authors to (a) interpret the clinical and statistical significance of findings, (b) use a consistent definition of outcome so that studies can be directly compared, and (c) report null and statistically significant findings.

Predictors, Moderators, and Mediators of Treatment Outcome Following Manualised

Cognitive-Behavioural Therapy for Eating Disorders: A Systematic Review

Manualised cognitive-behavioural therapy (CBT-BN) is the current leading evidence-based treatment for bulimia nervosa (BN) and recurrent binge eating(Hay, Bacaltchuk, Stefano, & Kashyap, 2009). CBT-BN targets the maintaining mechanisms of BN (i.e., over-evaluation of weight and shape, dietary restraint). Meta-analyses have shown CBT-BN to be superior to wait-list controls (8 trials) and alternative psychological treatments (7 trials) at producing remission from binge eating (Hay et al., 2009).

CBT-BN was reformulated not only to consider the role of additional maintaining mechanisms (i.e., core low self-esteem, clinical perfectionism, interpersonal problems, and mood intolerance), but also to apply it as a treatment for *all* eating disorders (Fairburn, Cooper, & Shafran, 2003). This enhanced treatment (CBT-E), based on a transdiagnostic theory, has been evaluated in several RCTs and has been shown to be superior to wait-list controls and alternative psychological treatments, and is associated with significant short and long-term symptom improvement in BN (Poulsen et al., 2014; Thompson-Brenner et al., 2016; Wonderlich et al., 2014), other specified feeding or eating disorder (OSFED; Fairburn et al., 2015; Fairburn et al., 2009), and anorexia nervosa (AN; Zipfel et al., 2014).

Despite these advancements, there is still substantial room for improvement. Short and long-term rates of remission from eating disorder cognitions and behaviours only range from around 37-69% across trials. Thus, improving the effectiveness of CBT-E is a research priority. One possible solution is to understand how, why, and for whom such treatments work. Studying the mechanisms of action, moderators and predictors of treatment outcome can aid in this understanding.

Mechanisms of action are the processes and events that occur within treatment that cause therapeutic change (Kazdin, 2007). Identifying mechanisms of action would improve

treatment effectiveness because the focus could shift toward enhancing the elements that effectively trigger the mechanisms of action and removing those that do not (Murphy, Cooper, Hollon, & Fairburn, 2009). Establishing a mechanism has several requirements, including (1) direct manipulation of a mechanism and observing its effect, (2) ensuring change is explained by the hypothesised mechanism only, (3) demonstrating a dose-response relationships, and (4) consistency/replication (Kazdin, 2007). Ample resources are required to establish mechanisms; thus, researchers initially examine treatment mediators.. Although not all mediators are MoA, all MoA are mediators; hence, studying mediators narrows down the search for causal mechanisms (Kraemer, Wilson, Fairburn, & Agras, 2002)

Mediators are variables intervening between the onset of treatment and the outcome of interest (Kraemer et al., 2002). Mediators cannot precede treatment nor be concomitant with the outcome. Mediators change because of treatment, and for which this change is associated with changes in the outcome. This temporal precedence is established through frequent assessment of the mediator and outcome so that their trajectory of change during treatment can be tracked (Kraemer et al., 2002).

By contrast, *baseline* variables that influence treatment outcome are either moderators or predictors (Kraemer et al., 2002). In RCTs, baseline variables that *interact with treatment type* to affect outcome are moderator variables, whereas baseline variables that affect outcome, *irrespective of treatment type*, are non-specific predictors (Kraemer et al., 2002). Unlike moderators, predictors of outcome can also be examined when comparison treatments are not implemented.(e.g., open trial). Identifying moderators provides us with knowledge about which treatment works best, for whom, and under what conditions (Kraemer, 2016), while , predictors provide important prognostic information on an individual's likely success in treatment. Together, an understanding of moderators and predictors of outcome can be

used to offer more targeted and intensive treatment so that successful outcomes are maximised (Agras et al., 2000).

Recently, predictors, moderators and mediators of eating disorder outcome have been of interest. Several reviews on predictors of eating disorder outcome have been published (Agency for Healthcare Research and Quality, 2015; Berkman, Lohr, & Bulik, 2007; Shapiro et al., 2007; Vall & Wade, 2015). However, findings from these reviews are based on studies using several distinct psychological and/or pharmacological treatments; as such, it is unclear what factors affect outcome specifically for CBT-BN or its variants (CBT-E). This review therefore aimed to synthesise the literature on mediators, moderators, and predictors of treatment outcome following CBT for eating disorders. Specifically, we intend to identify consistent mediators, moderators and predictors of eating disorder outcomes (i.e., disordered eating behaviours and cognitions and weight gain) following CBT based on the original or enhanced transdiagnostic model of eating disorders (Fairburn et al., 2003). To provide a comprehensive overview of the literature, we reviewed both non RCTs and RCTs that included any active (i.e., psychological/pharmacological) or inactive comparison. **Method**

This review was conducted according to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (Moher, Liberati, Tetzlaff, & Altman, 2009).

Search Strategy and Study Selection

The primary search strategy involved searching six databases: Medline, PsycInfo, Web of Science, CINAHL, EMBASE, and the Cochrane Database. The final search was conducted in March 2016. The following concepts were combined and searched for in the title and abstract.

1. Eating Disorder* OR disordered eat* OR binge eat OR bulimia OR anorexia OR underweight

2. Cognitive-behavioral OR CBT OR CBT-E

After duplicates were removed, the title and abstracts were screened. Full-texts of potentially relevant articles were read to determine whether full inclusion criteria were met. Reference lists of included papers and relevant reviews were also searched.

Inclusion and Exclusion Criteria

Articles had to meet the following criteria: (a) peer-reviewed papers published in English before June 2016; (b) include a sample aged over 16 years and meeting a diagnosis for threshold or subthreshold eating disorder; (c) examine at least one mediator, moderator or predictor of treatment outcome; (d) at least one treatment arm had to include manualised CBT that was entirely based on the original or enhanced cognitive-behavioural model. Only studies that administered these treatments were included because the theory on which these treatments are based makes clear predictions about which factors should impact treatment outcome . We acknowledge that there are several published cognitive-behavioural treatment manuals with empirical support. However, considering these manuals implement different treatment strategies and target different maintaining mechanisms, we limited our inclusion to studies that administered Fairburn's treatment in an effort to identify consistent theory-based mediators, moderators and predictors of outcome.

Search Results and Study Categorisation

Sixty-five articles met inclusion criteria (see Figure 1). Treatment outcomes were grouped into primary and secondary outcome categories. *Primary outcomes* included eating disorder cognitions, eating disorder behaviours, and, for studies that used underweight samples, weight gain. Eating disorder cognitions were most often (87% of studies) measured through the Eating Disorder Examination (EDE; Fairburn & Beglin, 1994). The remaining studies used the Bulimia Investigatory Test – Edinburgh (BITE; Henderson & Freeman, 1987) or the Eating Disorder Inventory (EDI; Garner, Olmstead, & Polivy, 1983). Eating disorder behaviours were assessed through objective binge eating (OBE) and/or purge

frequency, although some studies reported categorical behavioural outcomes (cessation of OBE) and others reported dimensional outcomes (changes in OBE). *Secondary outcomes* included weight loss (BED samples), psychosocial (e.g., depression, quality of life), and diagnostic status (e.g., diagnostic cross-over, relapse)[1].

Methodological Quality

All papers were coded for quality using criteria outlined by Porter and Chambless (2015) and Steketee and Chambless (1992) review of methodological issues in research on prediction of treatment outcome. There are eight indicators of the quality of a study, and each study gets a "quality score" based on the percentage of indicators that the study met. The indicators are: (a) for outcome measures, the authors controlled for baseline severity or reported change instead of raw scores; (b) authors did not use stepwise regression, as this form of regression excludes variables that account for a smaller proportion of variance, hence making it difficult to draw conclusions about the importance of omitted variables; (c) authors provided a rationale for *all* variables tested; (d) reliability ($\alpha \geq .70$) has been established for all variables examined; (e) Type I error was controlled if ≥ 20 tests were conducted; (f) both null and statistically significant findings were reported; (g) the study was sufficiently powered[2]; (h) the authors used a single, pre-specified primary outcome measure. If studies report multiple outcomes, interpretation becomes difficult when a variable affects one outcome but not the other. Quality scores were calculated for each paper, with papers receiving one point for each *applicable* criterion satisfied (e.g., criteria *d* were not applicable for studies that only tested demographic predictors). Porter and Chambless (2015) suggest that quality scores of 5 (63%) or more reflect good quality predictor/moderator analyses.

Data Analytic Method

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[1] Diagnostic status was included under secondary outcomes as only five of 56 studies reported these.
[2] Underpowered studies was defined by Porter and Chambless (2015) as those that lacked 80% power to detect a medium effect of $f^2 = .15$ at $\alpha = .05$ in a linear regression with one predictor. Power analysis showed that 55 participants or more are needed to meet this requirement

A meta-analysis was not feasible for this review because (a) there was considerable heterogeneity across studies with respect to the variables tested and outcomes reported, and (b) most studies did not provide sufficient data to calculate an effect size. Studies were instead categorised and summarised qualitatively. Mediation, moderation and prediction findings are presented and summarised for end of treatment (EOT) behavioural and cognitive outcomes for studies on BN, BED and mixed transdiagnostic samples (underweight and normal weight samples), and also for weight gain in underweight samples. Since the aim was to identify consistent predictors, findings are only presented for variables that were tested in two or more studies (supplementary material presents a list of all variables). For variables tested in two or more studies, the number of studies finding a statistically significant positive, negative or non-significant relationship to outcomes was tallied, providing an indication of the likely relationship between a particular variable and outcome. Findings pertaining to follow-up outcome were difficult to interpret as the length of follow-up varied widely. Thus, these findings are reported in supplementary materials. A few studies also reported secondary outcomes (see above). A brief synthesis is presented in supplementary materials

. **Results**

Sixty-five papers met full inclusion criteria. Twenty papers tested mediators, 12 papers tested moderators, and 34 papers tested predictors of outcome[3]. Across studies, 22 papers used a BN sample, 14 used a BED sample, two used an AN sample, and 27 papers used a mixed sample. CBT-BN was administered in most studies (26 studies), followed by CBT-E (25 studies) and guided self-help (14 studies). Tables 1, 2, and 3 present mediation, moderation, and predictor findings, respectively. Each table presents the information for BN, BED and mixed samples separately. In all cases, the rightmost column shows the average quality score and the range of scores for the studies investigating each relationship.

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[3] Four papers tested *both* mediators and predictors of outcome

Mediation findings

Twenty studies tested potential mediators of outcome. Of these, eight were RCTs. Ten studies were of BN, five were of BED, and five were of mixed samples.

Bulimia Nervosa. Four mediators were identified. Three studies tested *early symptom change* (in all cases defined as a 65-75% reduction in binge eating and/or purging by week four), as a mediator of outcome. All three studies, with good quality scores, found early symptom change to be associated with greater abstinence of binge eating and purging rates (Agras et al., 2000; Marrone, Mitchell, Crosby, Wonderlich, & Jollie-Trottier, 2009; Thompson-Brenner, Shingleton, Sauer-Zavala, Richards, & Pratt, 2015). Changes in *therapeutic alliance* ratings were tested in four studies (Constantino, Arnow, Blasey, & Agras, 2005; Loeb et al., 2005; Raykos et al., 2014; Wilson, Fairburn, Agras, Walsh, & Kraemer, 2002) and only one study found positive alliance ratings to be associated with a reduced purge frequency following CBT-BN (Constantino et al., 2005). Two studies assessed changes in *dietary restraint* and changes in *body-related concerns* (Spangler, Baldwin, & Agras, 2004; Wilson et al., 2002), and although greater changes in dietary restraint were significantly associated with better behavioural outcomes in both studies, changes in body-concerns were unrelated to outcome.

Binge Eating Disorder. *Early symptom change* was the only mediator tested in two or more BED studies.. Four studies tested early behavioural symptom change (all defined as a 65-70% reduction in binge eating by week 4), as a mediator of outcome. Three studies found early symptom change to be associated with better behavioural outcome (Grilo, White, Wilson, Gueorguieva, & Masheb, 2012b; Hilbert, Hildebrandt, Agras, Wilfley, & Wilson, 2015; Schlup, Meyer, & Munsch, 2010), while one high quality study did not replicate this (Masheb & Grilo, 2007). Three of these also assessed cognitive outcomes (EDE global

scores), and only Hilbert et al. (2015) found early behavioural symptom change to be associated with lower EDE global scores.

Mixed samples including Underweight Eating Disorders. Early *cognitive and behavioural symptom change* was the only mediator explored in mixed, underweight samples. Turner, Bryant-Waugh, and Marshall (2015) found no relationship between early change and remission from binge eating and purging. Two studies assessed whether early cognitive change was associated with outcome (Raykos, Watson, Fursland, Byrne, & Nathan, 2013; Turner et al., 2015). Both studies found early cognitive symptom change to be associated with better behavioural and cognitive outcomes. No mediators of weight gain were identified.

Mixed Samples of Normal Weight Eating Disorders. No mediator was assessed in two or more studies that used mixed samples of normal weight individuals.

Moderation findings

Eleven studies that met inclusion criteria tested moderators of treatment outcome. All of them were RCTs. Seven were BED trials, two were trials of BN, and two were trials of mixed samples of normal weight eating disorders.

Bulimia Nervosa. *EDE subscale* scores were tested as moderators in more than one RCT of BN. Both these trials compared CBT-E with a psychological comparison treatment (Accurso et al., 2016; Thompson-Brenner et al., 2016). Neither study, with reasonable quality scores, found EDE subscales to moderate behavioural and cognitive outcomes.

Binge Eating Disorder. Several variables tested as moderators of BED were identified. Client demographics, including *age, gender, ethnicity* and *education*, were commonly tested.. No study found evidence that demographics moderated outcome when CBT was compared with pharmacological (Grilo et al., 2014; Grilo, Masheb, Wilson, & Crosby, 2012a) or psychological treatment (Masheb & Grilo, 2008a; Wilson, Wilfley, Agras,

& Bryson, 2010). Two trials tested whether *age of binge eating onset* moderated treatment outcome (Grilo et al., 2012a; Masheb & Grilo, 2008a). While Masheb and Grilo (2008) found no evidence of moderation, Grilo et al. (2012) found that participants with an older age of binge eating onset had faster reductions in binge eating and EDE global scores *only* if they received CBT-BN and not fluoxetine.

Several trials assessed whether BED symptoms moderated outcome. No moderation effects were found for baseline *EDE global scores* and those classed as *pure dietary subtype* (vs. dietary-negative affect subtype) in trials using a psychological (Grilo, White, Gueorguieva, Wilson, & Masheb, 2013; Masheb & Grilo, 2008a, 2008b) or pharmacological comparison (Grilo et al., 2012a). Three trials assessed whether baseline *OBE frequency* moderated treatment outcome. Grilo et al. (2012) found that higher OBE frequency was associated with *greater* reductions in EDE global scores for those who received CBT-BN rather than fluoxetine. However, Wilson et al. (2010) found higher OBE frequency was associated with *poorer* rates of OBE abstinence for those who received CBTgsh over IPT. Moderation effects were not observed for Masheb and Grilo (2008a). Finally, two studies assessed *clinical vs. subclinical over-evaluation of weight and shape subtype* (Grilo et al., 2012a; Grilo et al., 2013). Only Grilo et al. (2012) reported that those classed as clinical (as opposed to subclinical) over-evaluation subtype had greater reductions in EDE global scores only if they received CBT-BN.

Depression scores, comorbid personality disorder, and *self-esteem* scores were also tested as moderators of BED. Across three trials, no moderation effects for these variables were found following CBT-BN (Grilo et al., 2012a) and CBTgsh (Masheb & Grilo, 2008a; Wilson et al., 2010).

Mixed Sample. No moderator variables that were tested in two or more trials for studies that used both normal and underweight mixed samples.

Predictor findings

Across 37 studies that tested baseline predictors of outcome, only six administered a comparison treatment. Three administered a CBT-based comparison treatment (e.g., guided verse pure self-help) and three administered an alternative psychological comparison. The latter three studies are included under prediction as no interaction with treatment type was analysed.

Bulimia Nervosa. Several predictors of BN were tested. Three non RCT's tested *age* and *duration of illness* as a predictor of cognitive and behavioural outcome (Agras et al., 2000; Cooper, Coker, & Fleming, 1996; Fahy & Russell, 1993). Only Fahy and Russell (1993) found evidence of prediction, where older participants and a longer duration of BN predicted poorer cognitive outcomes. This study received a low quality score. Further, client history variables, including a *history of AN* and a *history of depression* was unrelated to outcome (Agras et al., 2000; Cooper et al., 1996; Fahy & Russell, 1993).

Baseline symptoms of BN were often tested as predictors. While higher *shape concern* scores predicted poorer behavioural outcomes in one study of CBT-BN (Fahy & Russell, 1993), this effect was not replicated in two other higher quality studies (Agras et al., 2000; Cooper et al., 1996). Baseline *OBE and/or purge frequencies* were tested as predictors in eight studies, and findings were inconsistent. Three studies found higher baseline frequencies to predict poorer behavioural outcomes (Dawkins, Watson, Egan, & Kane, 2013; Fahy & Russell, 1993; Loeb, Wilson, Gilbert, & Labouvie, 2000), one found higher frequencies to predict poorer cognitive outcomes (Baell & Wertheim, 1992), one found higher frequencies to predict *better* behavioural outcomes (Leung, Waller, & Thomas, 2000) and three studies reported no relationship (Agras et al., 2000; Cooper et al., 1996; Ghaderi, 2006).

BMI and *weight suppression* were tested as predictors in three and two studies, respectively. Two non-RCT of CBT-BN found that lower BMI predicted poorer behavioural outcomes (Agras et al., 2000; Fahy & Russell, 1993). This was not replicated in a higher quality rating study of CBT-E (Dawkins et al., 2013). Out of two high quality studies, Dawkins et al. (2013) found no prognostic value for weight suppression and Butryn, Lowe, Safer, and Agras (2006) found a higher weight suppression to predict poorer abstinence rates following CBT-BN.

A few studies found evidence of prediction for comorbidity variables, although findings are inconsistent. While one low quality study of CBT-BN found higher *depression scores* to predict poorer behavioural outcomes (Fahy & Russell, 1993), three other higher quality studies did not replicate this (Agras et al., 2000; Cooper et al., 1996; Ghaderi, 2006). *Comorbid personality disorder* was observed as a predictor of poor behavioural outcomes of CBT-BN in two studies (Fahy, Eisler, & Russell, 1993; Fahy & Russell, 1993); although a higher quality study did not replicate this (Agras et al. 2000).

Mixed Samples Including Underweight Eating Disorders. Several pre-treatment predictors were explored. Two high quality studies assessed *motivation to change*. While Allen et al. (2012) found greater motivation to be associated with better cognitive outcomes following CBT-E, Ålgars et al. (2015) could not replicate this. *Age* was unrelated to treatment outcome in a study administering CBT-E (Dalle Grave, Calugi, & Marchesini, 2012). Finally, *baseline bulimic frequencies* was tested as a predictor in two studies (Dalle Grave, Calugi, & Marchesini, 2008; Dalle Grave et al., 2012), and only one study that administered CBT-E found higher bulimic frequencies to predict poorer behavioural outcomes (Dalle Grave et al., 2012).

Six variables tested as predictors of *weight gain* outcomes in underweight eating disorders were identified. *Age* was tested as a predictor of weight gain in two studies (Calugi,

Dalle Grave, Sartirana, & Fairburn, 2015; Dalle Grave et al., 2012), and only Calugi et al. (2015) found that a younger age predicted faster weight gain during CBT-E. Two studies that administered inpatient CBT-E tested *duration of illness* (Calugi, Dalle Grave, & Marchesini, 2013; Calugi et al., 2015) and *baseline EDE global scores* as predictors of weight gain (Calugi et al., 2015; Dalle Grave et al., 2008); no evidence of prediction was observed. Finally, *baseline binge eating, purge and compulsive exercise frequency* was tested as a predictor of weight gain in four studies (Calugi et al., 2015; Dalle Grave et al., 2008; Dalle Grave, Calugi, & Marchesini, 2009; Dalle Grave et al., 2012). No effects were identified.

Mixed Samples of Normal Weight Eating Disorders. Several pre-treatment predictors were explored. *Age* was unrelated to outcome in studies of CBT-BN (Castellini et al., 2012) and CBTgsh (Högdahl, Birgegård, & Björck, 2013). One study found lower *BMI* (Hogdahl et al. 2013) and lower baseline *depression scores* (Castellini et al. 2012) to predict better treatment outcome. *EDE subscale* and *global scores* were consistently unrelated to treatment outcome in a RCT (Loeb et al., 2000) and non-RCT (Högdahl et al., 2013). Six studies examined baseline *bulimic frequencies* as predictors. One RCT of CBTgsh found higher frequencies to predict poorer outcome (Loeb et al., 2000); three studies did not replicate this (Castellini et al., 2011; Castellini et al., 2012; Högdahl et al., 2013).

Binge Eating Disorder. Three variables tested as predictors were identified for BED. Participant *age*, *BMI*, and *general psychopathology* were tested in two studies that received a poor quality rating (Carter & Fairburn, 1998; Lammers, Vroling, Ouwens, Engels, & van Strien, 2015). Only one effect was observed; Lammers et al. (2015) found that higher levels of psychopathology predicted poorer cognitive outcomes.

Discussion

This review synthesised the literature on mediators, moderators or predictors treatment outcome following CBT-BN and its variants for eating disorders (Fairburn, 2008).

Sixty-two papers were included in this review, and the majority of studies were primarily designed to evaluate treatment efficacy or effectiveness. Thus, mediator, moderator and predictor findings were typically reported in post-hoc exploratory analyses. There was heterogeneity in the choice of variables tested. Studies tended to analyse variables that were not selected on the basis of theory, but were rather selected for analysis because they are routinely collected as part of clinical research (e.g., demographics). Many studies did not provide data for null findings, precluding an opportunity to conduct a meta-analysis. Little consistency emerged.

Summary of Key Findings

     Mediation findings. Few studies tested mediators of treatment outcome across the eating disorders. Two consistent findings emerged. First, early behavioural and cognitive symptom change was consistently found to lead to better behavioural and cognitive outcomes. Importantly, this effect was present across diagnoses and across different treatment modalities (e.g., self-help, therapist-led). This is consistent with two recent meta-analyses highlighting the importance of achieving early symptom change following eating disorder treatment (Linardon, Brennan, & de la Piedad Garcia, 2016; Vall & Wade, 2015). Second, reducing dietary restraint throughout the course of BN treatment was associated with better behavioural outcomes. This finding is consistent with the cognitive-behavioural model (Fairburn et al., 2003), which highlights that dietary restraint is a key mechanism maintaining binge eating. CBT attempts to eliminate dietary restraint in the early stages of treatment via the "regular eating" strategy (Fairburn, 2008), and although only two studies have explored the impact of changes in dietary restraint, the evidence thus far suggests that sufficient clinical attention should be devoted toward targeting this maintaining mechanism.

     The dearth of studies on treatment mediators suggests that the mechanisms of this treatment are still unclear. Although correlational data suggest that early change in dietary

restraint and an adherence to regular eating is associated with early behavioural symptom change (Spangler et al., 2004; Wilson et al., 2002; Zendegui, West, & Zandberg, 2014), these findings do not tell us precisely the processes and events that unfold within treatment that may *cause* this behavioural symptom change. As Kazdin (2007) notes, multiple criteria from numerous studies need to be satisfied for mechanisms to be established. Although testing mediators is often a first step for determining causal mechanisms, testing mediators requires *a priori* hypotheses that specify when and for what reasons change is likely to occur. This then determines what, when, and how often variables should be assessed throughout treatment. Thus far, studies have not been designed with the intention of testing mediators as MoA.

Moderation findings. Identifying moderations of treatment outcome is crucial for identifying which treatments work best for whom and under what conditions (Kraemer, 2016). We are yet to identify any consistent moderators of outcome across all eating disorders following CBT. Minimal research has been devoted toward testing moderators in samples other than BED. While EDE subscale scores have been the only moderator tested in multiple studies of BN, where moderation effects were not found, there have been only two studies testing moderation (using different variables) in mixed normal weight samples (Fairburn et al., 2009; Striegel-Moore et al., 2010). Moderators of BED outcome have received more attention. While several variables tested as moderators (e.g., demographics, global eating disorder psychopathology) were consistently unrelated to BED outcome, some variables (e.g., OBE frequency) produced conflicting moderator findings. Collectively, this broadly suggests that we have limited ability to match manualised CBT to certain individual profiles, and it is clearly evident that more research testing moderators of outcome is needed.

Prediction findings. A lot of research has been devoted to studying predictors of outcome following manualised CBT. The majority of studies that tested predictor variables did not employ a comparison treatment (e.g., open trials). A generally consistent finding

observed across research designs and diagnoses was that most predictors were consistently unrelated to treatment outcome (e.g., demographics, client history, psychopathology).These findings are important and may provide clarity to clinicians as they can expect their clients who receive CBT to improve to a similar degree, regardless of their age, gender, ethnicity, body weight and history. Finally, it should be noted that there was little consistency with respect to the type of variables tested. Many variables tested as predictors have only been examined in single studies (e.g., obsessionality, personality traits), limiting any opportunity for meta-analyses to pool together effects across multiple studies.

Methodological Considerations and Limitations in the Literature

Methodological limitations and differences across studies may partially explain why consistent predictors and moderators were not identified. A notable limitation was that most studies examined predictors/moderators in post-hoc, exploratory analyses using data collected for the purposes of describing the sample (e.g., demographics, client history) and/or evaluating treatment efficacy (e.g., baseline levels of the outcome). Thus, variables were not selected on the basis of theory. A focus on theoretically-grounded variables might prove useful for identifying consistent and robust predictors and moderators of outcome.

Statistical power was an issue across most included studies. Sample sizes were often small and so their power was only enough to detect large effects. Although *a priori* power analyses were typically conducted to determine sample sizes needed to detect within or between groups treatment effects (i.e., to determine efficacy), most studies did not conduct power analyses for tests of prediction, moderation and mediation.. Since the majority of studies failed to report effect sizes or provided data required to calculate effect sizes, it was not possible to assess the extent to which statistically non-significant predictors constituted cases of underpowered tests. Thus, although we recognise that null hypothesis significance

testing provides no indication of the strength of these relationships or how clinically useful they are (Cumming, 2012), this review could only rely on statistical significance for drawing conclusions.. An attempt was made, however, to extract some information regarding the quality of the evidence presented, and the lack of power would have detracted from the quality scores.

Guidelines on how to conduct, analyse, and report trials that examine treatment mediators have been provided (Kraemer et al., 2002). The lack of research on treatment mediators indicates that such guidelines have not been applied in eating disorder treatment research. Testing mediational hypotheses requires specification of the expected mechanisms and the time points at which changes are predicted to occur, in order to plan the repeated measurement of mediators and outcome within treatment. This type of research also requires large samples sizes to have adequate power for mediation tests (Fritz & Mackinnon, 2007). It is possible that researchers are deterred from testing meditational hypotheses for these reasons.

Limitations of the Current Review

Relying on p values for drawing conclusions about predictors or mediators is a limitation. Clinically significant, but not statistically significant predictors, moderators or mediators of outcome may have been overlooked in underpowered studies. Although a meta-analysis would have been the preferred choice for data analysis, it was not feasible to conduct one as effect sizes could not be calculated in many papers. .

Future Recommendations

Several recommendations are now put forth. First, researchers conducting treatment studies should plan, from the outset, to test mediators, moderators and predictors of outcome. This would allow researchers to formulate a clear set of *a priori* theory-driven hypotheses concerning the types of variables likely to impact outcome. Predictors, moderators and

mediators would then be selected on the basis of theoretical work and may be operationalised in a way that is appropriate for the specific research question. For instance, in the context of mediation researchers testing mechanisms of specific CBT components are encouraged to specify when specific therapeutic techniques are implemented, when change in the outcome is expected to occur, and how this change will come about (Kazdin, 2007). This would allow for both the mediator and outcome to be assessed simultaneously during this period. In CBT-E, for example, a reduction in weight concern is hypothesised to be mediated by a reduction in weight checking (as a result of "weekly weighing") (see Murphy et al. 2009 for a set of hypotheses concerning the likely mediators of CBT-E.). Since the weekly weighing procedure is implemented early on during CBT-E, researchers could then assess *both* weight checking and weight concern prior to, during, and after the implementation of weekly weighing. Assessing both the mediator and outcome at frequent intervals would allow one to control for prior outcome levels when predicting it subsequent change. Atheorietical" variables (e.g., demographics) should be examined only when there are clear reasons for doing so and should not compromise the testing of these theory-driven variables (e.g., decreasing power).

Second, comparing findings across studies and combining data from multiple independent studies is needed to identify robust mediators, moderators and predictors of outcome. This is currently difficult because outcomes vary (Williams et al., 2012). Ann agreed-upon definition of recovery is needed. Recently have authors highlighted the issue of disparate definitions of treatment outcome(Williams et al., 2012). Consistent with the transdiagnostic perspective, it is clear that treatment outcome should encompass disordered eating behaviours and cognitions and body weight. Indeed, Bardone-Cone et al. (2010) proposed the following definition of recovery, and found that when applied to a transdiagnostic sample, those who met all these criteria could not be distinguished from

healthy controls.. Recovery included (a) BMI ≥ 18.5, (b) abstinence from bingeing, purging and fasting for three months, (c) and achieving an EDE-Q global score within healthy population norms. It is critical that such a definition of recovery is used to advance the field.

Finally, statistical data for both null and significant findings should be reported. Small sample studies that are underpowered tend to rely on *p* values for drawing conclusions about predictors. While increasing sample size might not be feasible, we argue that this should not deter researchers from studying predictors, moderators or mediators. Instead, authors should place emphasis on reporting of effect sizes and confidence intervals, and interpret findings based on their clinical significance (Kraemer, 2016). This would also allow for the aggregation of results via meta-analyses. Currently, studies vary widely on the data they report, making it hard to synthesise findings, draw conclusions, and make clinical and policy recommendations. Independent findings of non-significant *p* values with small-moderate effect sizes that might be clinically meaningful would have been missed.

Conclusion

Studying mediators, moderators and predictors of outcome is important for improving the effectiveness of CBT for eating disorders. Limited consistent predictors and moderators of treatment outcome emerged from this review. There were also limited studies on treatment mediators.. To improve this treatments effectiveness, researchers should prioritise the study of predictors, moderators and mediators of outcome, and select variables that have strong theoretical and empirical rationales.

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[4] Note: Papers included in the systematic review are marked with an *. Some papers included in the systematic review but not referenced in text are listed in the appendices.

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Table 1

Qualitative Analysis on Mediators of Outcome for Bulimia nervosa, Mixed Samples, and Binge Eating Disorder

Sample				Study Design		EOT Behavioural Outcome			EOT Cognitive Outcome			
	Mediator Variable	Studies	RCT	Other	CBT-BN	CBT-E	CBTgsh	CBT-BN	CBT-E	CBTgsh	Mean Quality rating % (range)
BN												
	Early symptom change											
	Behavioural symptoms	3	1	2	+ +	+					86% (75-100%)
	Cognitive symptoms	0									
	Therapeutic alliance	4	3	1	+ 0 0				0		77% (50-85%)
	Dietary restraint	2	2	0	+ +			0			50% (50%)
	Body concerns	2	2	0	0 0			+			50% (50%)
BED	Early symptom change										
	Behavioural symptoms	4	3	1	+		0 + +			0 0 +	66% (33-100%)
	Cognitive symptoms	0									
Mixed											
	Early symptom change											
	Behavioural symptoms	2	0	2			+		0		66% (57-75%)
	Cognitive symptoms	2	0	2		+			+ +		80% (75-85%)

Note + = greater change in mediator is associated with better outcome; BN= Bulimia Nervosa; BED = Binge Eating Disorder; CBT-BN = cognitive-behavioural therapy for bulimia nervosa; CBT-E = enhanced cognitive-behaviour therapy; CBTgsh= cognitive-behavioural therapy guided self-help; EOT= end of treatment.

Table 2

Qualitative Analysis on Moderators of Treatment Outcome for Binge Eating Disorder and Bulimia nervosa

Sample	Moderator	RCT's	Comparison Treatment		EOT Behavioural Outcomes			EOT Cognitive Outcomes			Mean Quality rating % (range)
			Psychological	Pharmacology	CBT-BN	CBT-E	CBTgsh	CBT-BN	CBT-E	CBTgsh	
BED											
	Age	4	2	2	0		0 0 0	0		0 0	54% (43-71%)
	Gender	3	1	2	0		0 0	0		0	55% (43-71%)
	Ethnicity	2	1	1	0		0	0			57% (43-71%)
	Education	3	1	2	0		0 0	0		0	55% (43-71%)
	Older age of Illness onset	2	1	1	+		0	0		0	50% (50-50%)
	EDE global scores	2	1	1	0		0	0		0	50% (50-50%)
	Higher OBE frequencies	3	2	1	0		0 —	+		0	57% (50-71%)
	Dietary restraint subtype	2	1	1	0		0	0		0	50% (50-50%)
	Clinical over-valuation subtype	2	1	1	0		0	+		0	71% (57-85%)
	Depression scores	2	2	0			0 0			0 0	50% (50-50%)
	Comorbid personality disorder	3	2	1	0		0 0	0		0	57% (50-71%)
	Self-esteem	2	1	1	0		0	0		0	50% (50-50%)
BN											
	EDE subscale scores	2	2			0 0			0 0		60% (57-62%)

Note* 0 = no relationship identified; + = moderator was associated with better treatment outcome; — = moderator associated with poorer treatment. There were no moderators tested in more than two studies of mixed samples; BN= Bulimia Nervosa; BED = Binge Eating Disorder; CBT-BN = cognitive-behavioural therapy for bulimia nervosa; CBT-E = enhanced cognitive-behaviour therapy; CBTgsh= cognitive-behavioural therapy guided self-help; EOT= end of treatment.

Table 3

Qualitative Analysis on Predictors of Outcome for Bulimia Nervosa, Mixed Samples, and Binge Eating Disorder

Sample			Study Design		EOT Behavioural Outcome			EOT Cognitive Outcome			
	Predictor Variable	Studies	RCT	Other	CBT-BN	CBT-E	CBTgsh	CBT-BN	CBT-E	CBTgsh	Mean Quality rating % (range)
BN											
	Older age	3	0	3	0 0		0	—			57% (25-75%)
	Longer duration of illness	3	0	3	0		0	—			57% (25-75%)
	History of AN	3	0	3	0 0		—	0			57% (25-75%)
	History of depression	2	0	2	0 0			0			50% (25-75%)
	Dietary restraint	3	0	3	0 0		0	0			57% (25-75%)
	Weight concern	3	0	3	0 0		0	0			57% (25-75%)
	Higher shape concern	3	0	3	0 —		0	0			57% (25-75%)
	BITE scores	2	0	2	0 0			0			48% (25-71%)
	Lower BMI	3	0	3	— — 0			0			62% (25-85%)
	Higher weight suppression	2	0	2	—	0					93% (85-100%)
	Higher bulimic frequencies	8	2	6	0 0 — — +	—	0	— +			64% (25-100%)
	Higher depression scores	4	1	3	0 —		0	0			57% (25-75%)
	Comorbid personality disorder	3	0	3	0 — —			0			55% (25-75%)
	Lower self-esteem	3	1	2	0 0			—			67% (57-75%)
Mixed											
	Age	3	0	3	0	0			0	0	39% (25-50%)
	Motivation to change	2	0	2		+			+ 0		71% (71%)
	Dietary restraint	2	1	1	0		0				46% (43-50%)
	Higher eating concern	2	1	1	—		0				46% (43-50%)
	Higher weight concern	2	1	1	—		0				46% (43-50%)
	Higher shape concern	2	1	1	—		0				46% (43-50%)
	Lower EDE global scores	3	0	3	0				+	0	53% (25-85%)
	Higher bulimic frequencies	6	1	5	0 0 0		—		0 —	0	45% (25-85%)
	Lower BMI	5	1	4	0	0	0 0		0	+	44% (25-53%)
	Higher depression scores	3	0	3	—		0			0	44% (25-57%)

BED	Age	2	1	1	0	0	31% (25-37%)
	BMI	2	1	1	0	0	31% (25-37%)
	General psychopathology	2	1	1	0	—	31% (25-37%)

Note* 0 = no relationship identified; + = moderator was associated with better treatment outcome; — = predictor associated with poorer treatment

All studies that used an RCT implemented a CBT-based comparison group; note that there are two RCT's for BN studies; BN= Bulimia Nervosa; BED = Binge Eating Disorder; CBT-BN = cognitive-behavioural therapy for bulimia nervosa; CBT-E = enhanced cognitive-behaviour therapy; CBTgsh= cognitive-behavioural therapy guided self-help; EOT= end of treatment.