The cognitive-behavioural theory and treatment for eating disorders and disordered eating: A direct evaluation

Submitted by

JAKE LINARDON, BA (HONS)

A thesis submitted in total fulfilment of the requirements of the degree of

Doctor of Philosophy

School of Psychology

Faculty of Health Sciences

Australian Catholic University

Graduate Research Office

PO Box 968, North Sydney NSW 2059

AUSTRALIAN CATHOLIC UNIVERSITY

22nd September, 2017

Statement of Authorship and Source

This thesis contains no material published elsewhere or extracted in whole or in part from a thesis by which I have qualified for or been awarded another degree or diploma. No parts of this thesis have been submitted towards the award of any other degree or diploma in any other tertiary institution. No other person's work has been used without due acknowledgement in the main text of the thesis. All research procedures reported in the thesis received the approval of the relevant Ethics/Safety Committees (where required).

In all published research studies, I was the Principal Investigator, contributed 50% or more, and planned and prepared the work for publication. The four studies reported in this thesis were planned and conducted in collaboration with my primary supervisor, A/Prof Leah Brennan, and my associate supervisor, Dr Xochitl de la Piedad Garcia. Prof Tracey Wade[1] also contributed to the completion of Study 1 (chapter 5).

Signed: 

Date: 22nd September 2017.

[1] School of Psychology, Flinders University, Adelaide, South Australia.

Acknowledgements

First, I would like to thank my Principle Supervisor, Leah Brennan, for your continued guidance and words of encouragement throughout my candidature. I have most appreciated your consistent support and enthusiasm in helping me succeed as an early career researcher. My small achievements as a researcher thus far have been a result of your encouragement, guidance, and teachings. I look forward to working with you for many more years.

Second, I would like to thank my Secondary Supervisor, Xochitl de la Piedad Garcia. I am incredibly grateful for your unreserved motivation, support, and passion in helping me develop and complete my thesis. Your constant willingness to guide me throughout this process and throughout my earlier years at university has made this journey much easier for me. I also look forward to a future in working with you.

Third, I thank my amazing partner, Rachel. Your affection, support, and willingness to help with anything has made this experience much easier for me.

Fourth, I thank my mum, dad, brother, sister, and friends for the love and care they have provided me over these years. I particularly thank my Dad — if it wasn't for your words of wisdom, I would never have attended university. I'm glad I did. I can't forget my dog, Misha — thanks for keeping me company on the couch while I was writing this thesis.

Finally, thank you to my fellow PhD peers. We have all made this experience more enjoyable for ourselves.

Table of Contents

Abstract

In a series of four studies, the aim of the current research project was to evaluate the cognitive-behavioral theory and treatment (CBT) of eating disorders. The first study (Chapter 5) was a meta-analysis (Linardon, Wade, De la Piedad Garcia, & Brennan, in press) of randomized controlled trials (RCT) testing the efficacy of CBT for eating disorders. Pooling data from 79 RCTs, results showed that therapist-led and guided self-help CBT were efficacious for individuals with bulimia nervosa (BN) and binge eating disorder (BED). There was no evidence to suggest that CBT was more efficacious than other psychological interventions in anorexia nervosa (AN).

Having found evidence supporting the efficacy of CBT for certain eating disorder presentations, the second study (Chapter 6), which was a systematic review, focused on identifying the reliable factors that mediate, moderate, or predict outcome during CBT (Linardon, de la Piedad Garcia, & Brennan, 2016b). This review found that mediators and moderators of change have been largely unexplored, and that no reliable predictors of outcome emerged. Therefore, based on existing evidence, it was concluded that it remains unclear how, for whom, and under what conditions, CBT for eating disorders works.

To understand the mechanisms through which CBT for eating disorders may work, a cross-sectional evaluation of the cognitive-behavioral model in a large non-clinical sample was employed for the third study (Chapter 8; under review). This study validated the conceptual pathways hypothesized by the cognitive model; it also identified two additional variables that might be important mechanisms of change during CBT, body checking and dichotomous thinking. The inclusion of body checking and dichotomous thinking within the cognitive-behavioral model explained nearly three times the amount of variance in disordered eating symptoms than the model without these variables. The third study offered the necessary statistical support for the cognitive-behavioral model and its hypotheses.

The final study (Chapter 10) utilized a single case experimental design (*n*=8) to test the hypothesized cognitive-behavioral mechanisms of change during a CBT guided self-help program. The potential mechanisms of change examined were shape and weight concerns, dietary restraint, and adherence to regular eating strategies. Preliminary evidence showed that an adherence to regular eating in the second week of CBT was associated with a concurrent decrease in dietary restraint and binge eating. From this study, there was no evidence that other potential mechanisms were operating to reduce binge eating behavior.

Based on these four independent research studies, this thesis gathered converging evidence in support for the underling cognitive-behavioral model of eating disorders. Further, these findings suggest that CBT for eating disorders is likely to contain several theory-specific mechanisms that are responsible for this treatments effectiveness. More broadly, the current thesis offers support for the contention that CBT for eating disorders "works" because of the reason outlined by its underlying model and because of its specific therapeutic mechanisms.

Research Output

Published Peer Reviewed Papers as Chapters of Thesis

Linardon, J., de la Piedad Garcia, X., & Brennan, L. (2016). Predictors, moderators and mediators of treatment outcome following manualised cognitive-behavioural therapy for eating disorders: A systematic review. *European Eating Disorders Review*, 25, 3-12. doi: 10.1002/erv.2492

Linardon, J., Wade, TD, de la Piedad Garcia, X., & Brennan, L (in press). The efficacy of cognitive-behavioural therapy for eating disorders: A meta-analysis. *Journal of Consulting and Clinical Psychology.*

Chapters Currently Under Peer-Review

Linardon, J., de la Piedad Garcia, X., & Brennan, L. Evaluating an expanded cognitive-behavioural model of bulimia nervosa: The role of body checking, body avoidance, and dichotomous thinking. Under review in Eating Behaviors

Additional Peer-Reviewed Papers Published During Candidature

Linardon, J., Brennan, L., & de la Piedad Garcia, X. (2016). Rapid response to eating disorder treatment: A systematic review and meta-analysis. *International Journal of Eating Disorders*, 49, 905-919. doi: 10.1002/eat.22595

Linardon, J. (2017). Correlates of the over-evaluation of weight and shape in binge eating disorder and mixed eating disorder samples: A meta-analytic review. *Eating disorders*, 25, 183-198. doi: 10.1080/10640266.2016.1260374

Linardon, J., & Mitchell, S. (2017). Rigid dietary control, flexible dietary control, and intuitive eating: Evidence for their differential relationship to disordered eating and body image concerns. *Eating Behaviors*, 26, 16-22.

Linardon, J., & Brennan, L. (2017). The effects of cognitive-behavioral therapy for eating disorders on quality of life: A meta-analysis. *International Journal of Eating Disorders, 50,* 715-730. doi: 10.1002/eat.22719

Brennan, L., Teede, H., Skouteris, H., **Linardon, J.,** Hill, B., & Moran, L. (2017). Lifestyle and Behavioral Management of Polycystic Ovary Syndrome. *Journal of Women's Health*. https://doi.org/10.1089/jwh.2016.5792

Brennan, L., Mitchell, S., & **Linardon, J.** (2016) Feeding and Eating Disorders. Abnormal psychology in context: The Australian and New Zealand Handbook. Cambridge University Press.

Linardon, J., Wade, T D., De la Piedad Garcia, X., & Brennan, L. (2017). Psychotherapy for bulimia nervosa on symptoms of depression: A meta-analysis of randomized controlled trials. International Journal of Eating Disorders. doi: 10.1002/eat.22763

List of Tables

List of Figures

List of Acronyms

AN	Anorexia nervosa	EDE	Eating disorder examination
BCQ	Body checking questionnaire	EDE-Q	Eating disorder examination questionnaire
BES	Binge eating scale	EPP	Experimental psychopathology
BED	Binge eating disorder	EOT	End of treatment
BIA	Body image avoidance questionnaire	IPT	Interpersonal psychotherapy
BITE	Bulimia investigatory test – Edinburgh	MRC	Medical research council
BN	Bulimia nervosa	MBCT	Mindfulness-based cognitive therapy
CIA	Clinical impairment assessment	OSFED	Other specified feeding and eating disorder
CBT	Cognitive-behavioural therapy	OBE	Objective binge eating
CBT-E	Enhanced cognitive-behavioural therapy	RCT	Randomized controlled trial
CBT-Eb	Enhanced cognitive-behavioural therapy – broad version	SCED	Single case experimental design
CBT-Ef	Enhanced cognitive-behavioural therapy – focused version	SEM	Structural equation modelling

CFI	Comparative fit index	SRMR	Standardized root mean square

List of Appendices

Chapter 1. Introduction and Thesis Outline

Introduction

The prevalence and consequences of eating disorders and disordered eating are well-documented. Over 900,000 Australians meet diagnostic criteria for an eating disorder (Dekoitte Access Economics, 2012), and epidemiological data suggest that many more Australians exhibit subthreshold symptoms of disordered eating, including dietary restriction, binge eating, and shape and weight over-concern (da Luz et al., 2017; Ghaderi & Scott, 1999; Hay, Mond, Buttner, & Darby, 2008). This is significant, as eating disorders and disordered eating are associated with psychosocial distress and impairment, including reduced quality of life (Hay & Mond, 2005), elevated levels of depression and anxiety (Chen, McCloskey, & Keenan, 2009; Kaye, Bulik, Thornton, Barbarich, & Masters, 2004), interpersonal problems (Arcelus, Haslam, Farrow, & Meyer, 2013; Ghaderi, 2001), personality disorders (Chen, Brown, Harned, & Linehan, 2009; Chen, McCloskey, Michelson, Gordon, & Coccaro, 2011), and increased suicidality (Berkman, Lohr, & Bulik, 2007).

Effective interventions for eating disorders and disordered eating are needed. At present, specific forms of cognitive-behavioural therapy (CBT) are the recommended treatment approaches for bulimia nervosa (BN), binge eating disorder (BED), other specified feeding or eating disorders (OSFED), and anorexia nervosa (AN) according to both national (Hay et al., 2014) and international clinical guidelines (Hilbert, Hoek, & Schmidt, 2017; National Institute of Clinical Excellence, 2017). Numerous randomized controlled trials (RCTs) of these specific forms of CBT have reported large and long-lasting improvements in eating disorder symptoms that are generally superior to alternative psychological (e.g., interpersonal psychotherapy) and pharmacological (e.g., antidepressants) treatments (e.g.,

Agras, Walsh, Fairburn, Wilson, & Kraemer, 2000b; Fairburn et al., 2015). However, improvements in treatment are still needed, as RCTs of CBT and other psychological treatments are characterized by significant attrition, relapse, and treatment non-response (Wonderlich et al., 2014). Further, important questions still remain, such as the best available treatment for AN (Byrne et al., 2017), and the mechanisms through which any given treatment operates (Kazdin, 2007).

Evaluating Complex Interventions: A Framework

A possible pathway to the development of effective eating disorder treatments is through systematic testing and examination of theoretical models of eating disorders (Pennesi & Wade, 2016). This pathway is highlighted and described by the UK Medical Research Council (MRC). The MRC framework put forth a set of guidelines for researchers who intend to develop and evaluate complex interventions for health conditions (Campbell et al., 2000). Figure 1.1 presents the main stages and the interactions between the stages that characterise this process. According to these guidelines, best practice is to develop and evaluate interventions systematically, through the following four stages:

(1) ***Developmental stage:*** includes identifying the existing evidence base for related interventions (e.g., systematic reviews); developing a theory that informs the understanding of the processes of change; and modelling the processes of change outlined in this theory.

(2) ***Feasibility stage:*** includes testing procedures for feasibility through case-series designs or pilot studies; and estimating the likely rates of participant recruitment and retention.

(3) ***Evaluation stage:*** includes a test of the intervention's effectiveness, ideally through experimental RCTs; investigating the intervention's mechanisms of change; and assessing its cost-effectiveness.

(4) ***Implementation stage:*** includes the dissemination of research findings; the evaluation of the intervention in "real world" settings; and an assessment of the long-term impact of the intervention.

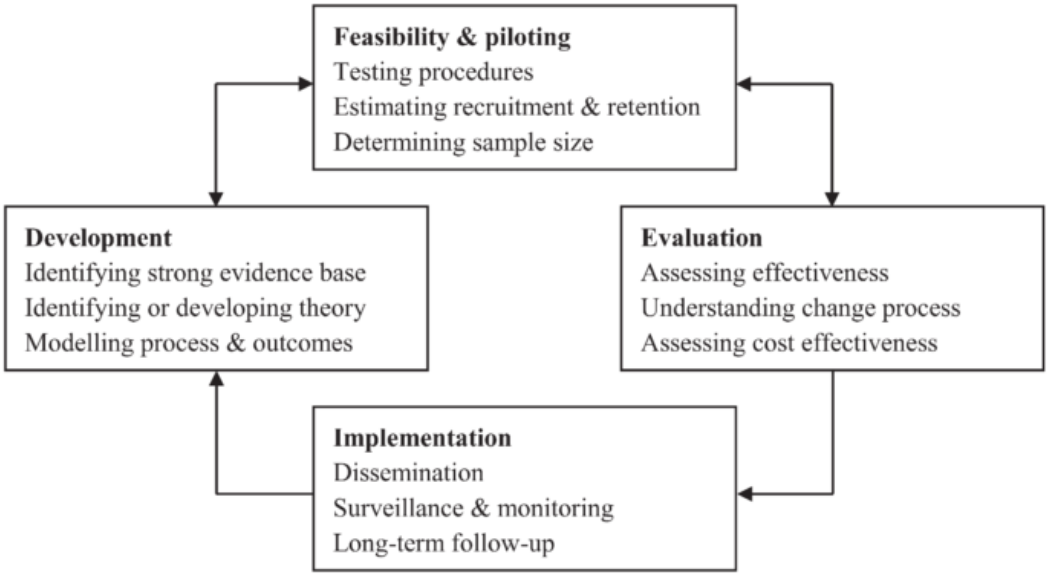


Figure 1.1: Key elements of the Medical Research Council's (MRC) evaluation framework. Figure taken from the Medical Research Council (Craig e al., 2008, 2013).

According to the MRC, these stages will not typically follow a linear sequence (Campbell et al., 2000). Rather, progression from one stage to another is an iterative process, and researchers are encouraged to move back and forth between stages. For example, an intervention might already be disseminated in the real world (*implementation stage*), yet advancements in theory that point toward several plausible mechanisms of action might then be explored in case-series designs (*feasibility stage*). Once there is preliminary evidence

supporting the role of these mechanisms in a case-series design, their causal role can be tested in larger RCTs (*evaluation stage*). Improving an intervention according to this framework is an iterative process that requires continual refinements (Campbell et al., 2000).

In the context of disordered eating, Pennisi and Wade (2016) sought to investigate whether current theoretical models of disordered eating had progressed past the point of theory development and onto the stages of evaluation and implementation. The authors identified 23 theoretical models of disordered eating; of these, only two models — the dual pathway model of BN (Stice, Nemeroff, & Shaw, 1996) and the cognitive-behavioural model of eating disorders (Fairburn, Marcus, & Wilson, 1993b) — had progressed past the point of theory development, and had been examined in RCTs and disseminated in real world settings. The authors determined that these 23 models of disordered eating shared similar overlapping risk and/or maintaining factors (e.g., preoccupation with weight/shape, emotion regulation difficulties). This led to the conclusion that the next generation of research should seek to advance the study of those models that have progressed past theory development, rather than on trying to develop entirely new theories that are likely to overlap with existing models

Thesis Aim and Overview

In line with Pennesi and Wade's (2016) above conclusion, the current thesis will focus on evaluating the cognitive-behavioural model of eating disorders. In particular, this thesis will argue that the large body of evidence supporting the efficacy of cognitive-behavioural *treatment* does not necessarily provide direct empirical support for the underlying cognitive-behavioural *theory*. Testing the key processes of change outlined in the cognitive-behavioural theory is crucial for eventually uncovering CBT's mechanisms of action. Pinpointing CBT's change mechanisms is crucial for improving its effectiveness, as research could then aim to develop new or augment existing therapeutic strategies designed

to target these mechanisms (Murphy, Cooper, Hollon, & Fairburn, 2009). The iterative MRC framework will serve as a guide for this thesis' intention of using several distinct methodological approaches for evaluating the cognitive-behavioural model. It is important to note that while there are several distinct cognitive-behavioural conceptualisations of eating disorders (e.g., Garner & Bemis, 1982; Pike, Loeb, & Vitousek, 2001), the current thesis aims to focus on the cognitive-behavioural theory and treatment devised by Fairburn and colleagues (Fairburn, Cooper, & Shafran, 2003a; Fairburn et al., 1993b).

This research is presented as a thesis by publication in accordance with section five of the Australian Catholic University's Guidelines on the Preparation and Presentation of a Research or Professional Doctoral Thesis for Examination (Australian Catholic University, 2015). This research project consists of four studies, described in four individual journal articles, each of which addresses a specific aim. All of these studies are connected by a unified body of supporting research, and each study builds on the study(s) that came before it.

The thesis structure is as follows: Chapter 2 reviews the evolution of CBT for eating disorders and the current research supporting its efficacy. Chapter 3 provides a discussion of the importance of psychotherapy process-based research. Chapter 4 provides an overview of the methodological rationale for the first two studies of this thesis. Chapter 5 presents a meta-analysis of the efficacy of CBT for eating disorders. Chapter 6 presents a systematic review of mediators, moderators, and predictors of response to CBT for eating disorders. Chapter 7 provides a literature review of studies that have directly tested the cognitive-behavioural model, and provides the rationale for the final two studies. Chapter 8 presents the findings from a cross-sectional evaluation of the cognitive-behavioural model of disordered eating. Chapter 9 presents an overview of the methodological rationale for the final study. Chapter 10 presents the findings from the final study, which examined mechanism of change during

CBT for disordered eating. Chapter 11 presents a general discussion, highlights the implications and limitations of this research, and outlines future directions for research.

Chapter 2: The Evolution of Cognitive-Behavioural Therapy for Eating Disorders

Chapter Outline

The purpose of this chapter is to provide an overview of the evolution of CBT for eating disorders. This chapter will review the development and evolution of the cognitive-behavioural theory and treatment for eating disorders. The evidence related to the efficacy of CBT for eating disorders will also be presented, and gaps in the literature will be highlighted. This literature review will form the rationale for the first study (Chapter 5).

CBT's Evolution

The cognitive-behavioural theory and treatment of eating disorders emerged in the mid-1980s. Research trials evaluating CBT in individuals BN were published before Fairburn and colleagues devised the cognitive-behavioural theory and treatment. These trials, however, treated BN through an approach based on CBT for depressive disorders (e.g., Freeman, Sinclair, Turnbull, & Annandale, 1985). It was not until Christopher Fairburn formulated a theoretical model of the hypothesised *maintaining* mechanisms of BN that a specific cognitive-behavioural treatment protocol for eating disorders was developed. Note that, in contrast to a risk factor, which is a variable that predicts the *onset* of symptoms, maintaining factors are variables that predict symptom persistence among initially symptomatic individuals (Stice, 2002). Fairburn argues that the reason why BN developed may not necessarily be relevant to the reasons why BN persists, and therefore targeting maintaining rather than risk factors is crucial to clinical improvement (Fairburn et al., 1993b).

According to this cognitive-behavioural model (Fairburn et al., 1993b), the core psychopathology of BN is a dysfunctional self-evaluative system, whereby individuals determine their self-worth largely in terms of weight and shape, and their control. This over-concern with weight and shape encourages inflexible dietary restraint, which includes strict dietary rules (e.g., what, when, and how much one can eat) that govern eating behaviour. These dietary rules, however, are difficult to sustain, and the inevitable "breaking" of them prompts an all-or-none reaction ("I have completely ruined my diet"). This reaction then results in episodes of uncontrollable binge eating. Compensatory behaviours (e.g., self-induced vomiting) can follow binge eating as an attempt to counteract any weight gain that may occur as a result. Extreme concerns about weight and shape are exacerbated following episodes of binge eating, thereby maintaining further dietary restraint, which leads to a self-perpetuating cycle (Fairburn et al., 1993b). A schematic representation is presented in Figure 2.1.

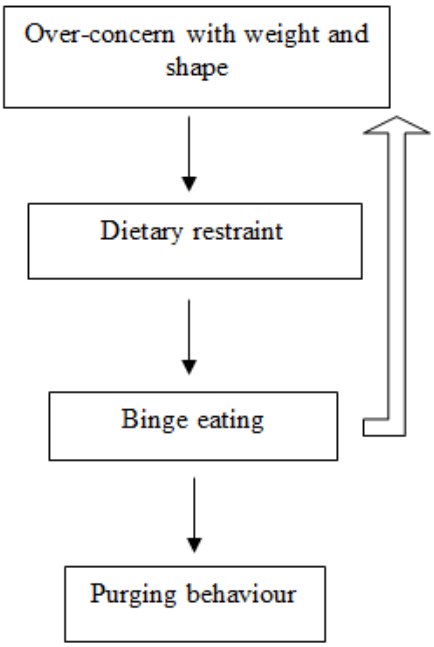


Figure 2: A schematic representation of the cognitive-behavioural model of bulimia nervosa.

A detailed treatment manual of CBT-BN was published in 1993 (Fairburn et al., 1993b). Three RCTs of CBT-BN were conducted prior to the publication of this manual. In the first RCT, CBT-BN (*n*=11) resulted in greater improvements in eating disorder and general psychiatric symptoms than short-term focal psychotherapy (*n*=11) at post-treatment and 8 month follow-up (Fairburn, Kirk, O'Connor, & Cooper, 1986). In the second RCT (Fairburn et al., 1991), although no post-treatment differences between CBT-BN (*n*=21), interpersonal psychotherapy (*n*=19) or behaviour therapy (*n*=22) were observed on binge eating and purge frequencies, CBT-BN resulted in greater reductions in weight and shape concerns and dietary restraint. At 12 month follow-up, only CBT (*n*=20) and IPT (*n*=18) were compared, and no outcome differences were observed, indicating that the beneficial effects of IPT "caught up" to CBT over time (Fairburn, Jones, Peveler, Hope, & O'Connor, 1993a). In the third RCT, Garner et al. (1993) compared CBT-BN (*n*=25) to supportive-expressive therapy (*n*=25), and found that although no significant post-treatment differences in binge eating and purging were observed, CBT-BN led to greater reductions in shape and weight concerns and dietary restraint at post-treatment. This study did not report findings at follow-up. These findings provided initial evidence of the efficacy of CBT-BN.

Many more RCTs of CBT-BN have been conducted since these early trials, and this treatment has been shown to mostly outperform other psychological treatments. For example, Agras and colleagues (Agras et al., 2000b) conducted the largest multisite trial of CBT-BN to date in their comparison of CBT-BN (*n*=110) to IPT (*n*=110). Post-treatment recovery rates were significantly higher for those who received CBT-BN (29%) than for those who received IPT (6%), but the two treatments did not differ at 12 month follow-up (CBT-BN = 28% recovered, IPT= 17% recovered). These findings replicated the previous trial comparing CBT-BN to IPT (Fairburn et al., 1993a), which demonstrated that CBT-BN is a faster acting

treatment than IPT. Note that the superiority of CBT-BN over other psychological interventions has not always been observed. For instance, some trials have reported no significant difference between CBT-BN and schema therapy (McIntosh et al., 2016), physical therapy (Sundgot-Borgen, Rosenvinge, Bahr, & Schneider, 2002), and dialectical behaviour therapy (Chen et al., 2016).

Other trials also compared CBT-BN to antidepressant medication. Since symptoms of depression are purported to be an important risk and maintaining factor for BN symptoms, treating depressive symptoms through antidepressants was hypothesized to have a secondary effect on BN symptoms (Mitchell et al., 2001). In an early trial, for example, Goldbloom et al. (1997) compared the relative efficacy of CBT-BN (*n*=24) and fluoxetine (*n*=23), and found that individuals who received CBT-BN had a significantly greater percentage reduction in vomiting frequency (79.2% reduction) and achieved greater rates of binge eating and purging abstinence (43%) than individuals who received fluoxetine (37.4% reduction and 17% abstinence). In addition, Agras and colleagues compared CBT-BN (*n*=24) to desipramine (*n*=23), and found that CBT-BN was significantly superior to desipramine at reducing binge eating and purging behaviour at post-treatment (Agras et al., 1992). The superiority of CBT-BN over antidepressants has not always been replicated, however. For instance, Jacobi and colleagues reported no significant differences in binge eating and vomiting frequency between those who received CBT and those who received fluoxetine (Jacobi, Dahme, & Dittmann, 2002).

Overall, a large body of evidence has supported the efficacy of CBT-BN. As this treatment has been the most widely investigated eating disorder treatment, with results showing CBT to generally outperform other interventions, early clinical guidelines recommended CBT-BN as the front-running treatment for BN (NICE, 2004).

CBT for binge eating disorder. It was noted that binge eating behaviour often occurred independently from compensatory behaviours (Halmi, Falk, & Schwartz, 1981). This pattern of eating behaviour was later termed Binge Eating Disorder (BED), and early research found that BED was highly prevalent in overweight men and women (Spitzer et al., 1992). BED was a category placed in the DSM-IV appendices identifying it as an important clinical condition that warranted further investigation. Although BED was not formally classified as an eating disorder, it was recognised that BED and BN shared important symptoms (e.g., weight and shape concerns, binge eating). This led to the belief that CBT and IPT interventions may also be effective treatments for this disorder. Consequently, investigators began to examine the efficacy of CBT for BED (Telch, Agras, Rossiter, Wilfley, & Kenardy, 1990; Wilfley et al., 1993). Although the treatment manuals delivered in RCTs of CBT for BED were not the same as Fairburn's CBT-BN manual, they shared important similarities, including, for example, the importance placed on regular eating principles, eliminating restrictive dieting, a providing relapse prevention techniques (Wilfley et al., 1993). The first RCT that compared group-based CBT for BED (*n*=19) to a wait-list (*n*=21) found significantly higher rates of binge eating abstinence for those who received CBT (79%) than the wait-list (0%) at post-treatment (Telch et al., 1990). Two larger RCTs compared group-based CBT to IPT, both showing no significant group differences in abstinence rates at post-treatment and 12 month follow-up (Wilfley et al., 1993; Wilfley et al., 2002). These findings provided promising support for the efficacy of both CBT and IPT for BED.

Refining and improving CBT. In the early 2000s, Fairburn and colleagues attempted to improve CBT-BN, via two major revisions (Fairburn et al., 2003a). These revisions led to the "enhanced" cognitive behavioural theory and treatment (CBT-E). First, CBT-E adopted a

transdiagnostic perspective. It was hypothesised that all eating disorders are maintained by a common set of mechanisms (shape and weight over-evaluation and dietary restraint), which led to the proposition that a treatment capable of targeting such mechanisms should be effective for all eating disorders (Fairburn et al., 2003a). Second, CBT-E was expanded to include and address four additional maintaining mechanisms (i.e., clinical perfectionism; core low self-esteem; mood intolerance; and interpersonal difficulties) thought to interact with, and exacerbate, the core features of eating disorders. These additional maintaining mechanisms were thought to operate in a subset of individuals, particularly those who failed to respond to CBT-BN (Fairburn et al., 2003a). A broad form of CBT-E (CBT-Eb), which contains supplementary modules designed to target these additional mechanisms, is provided to this subset of individuals. For most individuals, a focused version of CBT-E (CBT-Ef) is provided, which exclusively targets the universal eating disorder psychopathology via a collection of new and refined treatment strategies. Thus, CBT-E is a treatment that is delivered flexibly and tailored toward the specific psychopathology present in individuals. A detailed treatment manual of CBT-E was published in 2008 (Fairburn, 2008).

Evidence supporting the efficacy and effectiveness of CBT-E comes from RCTs and open trials. RCTs have documented the short and long-term superiority of CBT-E over wait-lists and active psychological comparisons (IPT, psychodynamic therapy) in transdiagnostic (Fairburn et al., 2015) and BN (Poulsen et al., 2014) samples. In addition, recent RCTs reported significant symptom improvement and weight gain following CBT-E for AN, although no differences between CBT-E, the Maudsley Model, and Specialist Supportive Clinical Management were observed (Byrne et al., 2017). Delivering CBT-E in a community clinic (effectiveness trials) has been found to be effective for treating eating disorders (Byrne, Fursland, Allen, & Watson, 2011), and large symptom improvements have been demonstrated in adolescents with AN (Dalle Grave, Calugi, Doll, & Fairburn, 2013). Taken

together, these findings provide support for the efficacy and effectiveness of CBT-E for a range of eating disorder presentations.

Although the development of CBT-E was an important milestone in eating disorder treatment research, important limitations of CBT-E exist. For example, although remission rates have been reported to be higher in some trials of CBT-E than CBT-BN, a large percentage (30-40%) of individuals still do not fully recover following CBT-E. No trials have directly compared CBT-BN to CBT-E in individuals with BN or with another type of eating disorder (e.g., BED), so it is unknown whether CBT-E is in fact an "enhanced" treatment. This is problematic, as CBT-E was developed with the intention of not only being a transdiagnostic intervention, but to also be a stronger intervention that can effectively treat a larger number of eating disorders. These concerns have led some authors to argue that CBT-E can still be improved, via a number of methods, including, for example, the identification of (a) treatment mechanisms, (b) the ineffective treatment modules, and (c) moderators of change (Lampard & Sharbanee, 2015).

Improving the dissemination of CBT. It was noted that only a small proportion of individuals with eating disorders have access to or receive CBT (Shafran et al., 2009). Hart and colleagues estimated that, globally, only 23% of individuals with eating disorders seek treatment (Hart, Granillo, Jorm, & Paxton, 2011), and earlier work in the U.S. showed that only 7% individuals (*n*=352) with BN presenting to treatment had received CBT in the past (Crow, Pederson Mussell, Peterson, Knopke, & Mitchell, 1999). A major factor contributing to this low level of exposure to CBT across the world was the shortage of therapists with expertise in CBT (Shafran et al., 2009). Therapists require specialised and intensive training to become competent in delivering CBT. This training has not been readily available. For instance, a survey of community-based clinicians in the U.S reported that only 35% of

therapists said that they had delivered CBT to their clients with eating disorders (Von Ranson, Wallace, & Stevenson, 2013). Critically, only half of all therapists surveyed in this study had been trained in CBT, despite the majority reporting a desire to receive CBT training. Another important factor that limits the dissemination of CBT is the cost of treatment. Many health care systems provide limited insurance that does not typically cover the recommended 16-20 sessions of CBT. Consequently, recent efforts to improve the dissemination, accessibility, and affordability of evidence-based eating disorder treatments have been made, with a focus on making treatment cheaper, shorter, and less complex. Guided self-help and technology-based programs may achieve these goals (Agras, Fitzsimmons-Craft, & Wilfley, 2017).

Cognitive-behavioural guided self-help. Guided self-help is a psychological intervention where the client takes home a standardized treatment manual and works through the manual somewhat independently (Cuijpers, Donker, van Straten, Li, & Andersson, 2010). Guided self-help programs are characterised by the use of a therapist who supports, facilitates, or coaches (rather than directs) an individual through the standardized program (Cuijpers et al., 2010). Interactions between therapist and client in this mode of delivery can occur in many ways, including face to face, telephone, email, or online contact.

There are advantages to guided self-help programs. First, guided programs are typically briefer and cheaper than therapist-led CBT (Fairburn, 2013). For example, guided programs have been successfully delivered in fewer than 8 sessions, and have been shown to result in reduced patient use of treatment as usual services over the 12 months following a the use of a guided program (Lynch et al., 2010). Second, guided programs can be delivered by non-specialist health care providers with minimal training. For example, general practitioners, nurses, and undergraduate psychology students have successfully delivered

CBT guided self-help (CBTgsh; Striegel-Moore et al., 2010). Third, because guided programs can be delivered via the internet, they allow an individual to engage in therapy at a time, location, and place of their convenience, thereby minimising potential barriers to accessing treatment, such as distance from treatment clinics (Wilson & Zandberg, 2012).

Guided self-help is effective for treating various psychiatric conditions. Two meta-analyses demonstrated that face to face CBTgsh and therapist-led CBT were not significantly different from each other (with negligible effect sizes) in the treatment of depression, anxiety and somatic (e.g., sexual dysfunction) disorders (Andersson, Cuijpers, Carlbring, Riper, & Hedman, 2014; Cuijpers et al., 2010). Moreover, other meta-analyses have shown that (a) guided self-help e-therapy for depressive and anxiety disorders significantly outperformed (with moderate to large effect sizes) wait-list controls, and (b) e-therapy was equally efficacious to face-to-face therapist-led treatment (Andersson & Cuijpers, 2009; Andrews, Cuijpers, Craske, McEvoy, & Titov, 2010). These promising findings have led researchers to suggest using guided self-help interventions as a complement to face-to-face treatment, or as an alternative approach when standard treatment is not available.

CBTgsh has also been applied to individuals with eating disorders and disordered eating. Fairburn (1995) initially published an abbreviated self-help version of CBT-BN, and then updated this in 2013 to align with the transdiagnostic perspective. Since then, numerous RCTs have documented the efficacy of face-to-face and computerized CBTgsh in individuals with BN (Banasiak, Paxton, & Hay, 2005), BED (Carter & Fairburn, 1998; Chen et al., 2016; Wagner et al., 2016), and in individuals with subthreshold eating disorders (Aardoom et al., 2016; Ghaderi, 2006; Ghaderi & Scott, 2003). In light of the promising outcomes of CBTgsh, clinical practice guidelines have recommended a “stepped care” approach to the treatment of eating disorders, where guided self-help is offered as a first step in treatment, with more

intensive resources reserved for those who fail to respond to guided self-help (National Institute of Clinical Excellence, 2017).

Summary

In summary, significant progress has been made toward developing, adapting, and refining effective eating disorder treatments such as CBT. CBT for eating disorders can be delivered in individual or group formats (Chen et al., 2003), over the internet (Loucas et al., 2014), and by non-specialist health care providers. There have been numerous RCTs evaluating the efficacy of all of these forms of CBT, and the outcomes of these individual trials have largely demonstrated that CBT is an efficacious treatment for eating disorders.

Systematic Reviews and Meta-Analyses of CBT

Numerous researchers have synthesised the results of findings from RCTs of CBT for eating disorders. The primary method for researchers has been to systematically search the literature for all available studies that have answered this pre-defined question (i.e., is CBT efficacious), evaluate the findings collectively, and reach conclusions regarding the [comparative] efficacy of CBT. Some authors have synthesised the results of RCTs of CBT for eating disorders qualitatively (Berkman et al., 2007; Hay, 2013), whereas others have used meta-analytic procedures to quantify the size of the effect of CBT (Ghaderi & Andersson, 1999; Hay, Bacaltchuk, Stefano, & Kashyap, 2009).

Several systematic reviews and meta-analyses of CBT for eating disorders have been conducted. Findings from these reviews consistently show that therapist-led CBT is superior to wait-list controls and alternative psychological comparisons (e.g., any other psychotherapy approach) at reducing behavioural symptoms in BN and BED (Ghaderi & Andersson, 1999; Hay et al., 2009; Spielmans et al., 2013). Several reviews have also shown that specific

modalities (e.g., CBTgsh) and formats (e.g., group-based) of CBT outperformed wait-list controls in BN and BED (Loucas et al., 2014; Polnay et al., 2014). Only one meta-analysis has examined the efficacy of CBT for AN, where CBT was shown to not differ significantly from either family-based therapy or short-term psychodynamic therapy (Hay, Claudino, Touyz, & Abd Elbaky, 2015). However, as will be reviewed, most of the earlier meta-analyses have been narrow in focus, for example, by placing specific emphasis on one diagnosis, one type of symptom, and on a limited number of treatment modalities. Thus, several important questions have been left unanswered.

Key Questions Left Unanswered

Previous systematic reviews and meta-analyses of CBT for eating disorders have not answered a number of important questions. First, it is not known whether CBT has a strong effect on reducing cognitive symptoms (e.g., extreme concerns about weight, shape and eating), as previous reviews have only analysed CBTs effect on behavioural symptoms (e.g., binge eating). This question is crucial, as cognitive symptoms are manifestations of important maintaining mechanisms, and have been shown to be strong predictors of relapse in BN (Fairburn, Peveler, Jones, Hope, & Doll, 1993c). It has been argued that the efficacy of an eating disorder treatment should be based on its effect on both cognitive and behavioural symptoms (Williams, Watts, & Wade, 2012). Currently, CBT's effects on cognitive symptoms for all diagnoses are not known.

Second, each review has focused on CBT's effect on a specific diagnosis (e.g., BN or BED). As a result of the transdiagnostic view of eating disorders (Fairburn, 2008), recent trials of CBT are not limited to treating specific eating disorder presentations. A number of these recent transdiagnostic trails have been excluded from systematic reviews and meta-analyses evaluating CBT or related interventions for specific diagnoses. Therefore, an

updated meta-analysis that includes all eating disorder presentations is needed to determine whether CBT is efficacious across the eating disorder spectrum.

Third, the comparative efficacy of CBT and pharmacotherapy is unknown. One early review found antidepressants to be equally efficacious to CBT for BN on binge/purge abstinence at post-treatment (Hay, Claudino, & Kaio, 2001). However, many more pharmacotherapy versus CBT trials of both BN and BED have been published since 2001, and this review did not assess the comparative efficacy of CBT and pharmacotherapy at follow-up or on cognitive symptoms. Comparing the efficacy of CBT and pharmacotherapy for eating disorders is important, particularly in the Australian context, as national practice guidelines recommend pharmacotherapy as an alternative treatment approach (Hay et al., 2014).

Finally, it is unclear whether CBT outperforms other *specific* psychological treatments. Three meta-analyses have compared CBT to other specific psychological treatments for eating disorders. Two of these compared CBT to behavioural interventions. Hay et al. (2009) found CBT to be superior to behavioural interventions (k=4) on rates of remission in BN, whereas Spielmans et al. (2013) reported no significant difference in outcomes between these treatments in individuals either with BED (k=4) or with BN (k=8). The third meta-analysis compared CBT to IPT on behavioural outcomes for BN and BED, and found CBT to outperform IPT on behavioural symptom improvement (Cuijpers, Donker, Weissman, Ravitz, & Cristea, 2016b). However, this analysis did not include a comparison at follow-up and on cognitive symptoms. Direct comparison of CBT to an alternative psychotherapy is crucial for providing indirect empirical support to the underlying cognitive-behavioural model of eating disorders, and also for guiding future work on the likely mechanisms of change that are operating in these treatments (Lorenzo-Luaces, German, & DeRubeis, 2014).

Chapter Summary

This chapter reviewed the evolution of CBT for eating disorders. It reviewed how CBT has emerged as a transdiagnostic treatment, and how CBT can be implemented through a variety of different formats and modalities. The evidence supporting the efficacy of CBT was presented. While several reviews on the efficacy of CBT for eating disorders have been published, this literature review highlighted several questions that remain unanswered. These questions will be answered in the first study of this thesis. Study 1 is a meta-analysis of CBT's efficacy, which aims to examine whether CBT is efficacious for each eating disorder presentation in the short and long-term, relative to other psychological and pharmacological treatments. Prior to the presentation of this meta-analysis (Chapter 5), the following two chapters will outline the importance of process-based research (Chapter 3) and provide the methodological rationale for employing a systematic review and meta-analysis (Chapter 4).

Chapter 3: A Focus on Psychotherapy Process-Based Research

Introduction

As discussed in the previous chapter, specific forms of CBT are empirically-supported eating disorder treatments that are recommended by clinical practice guidelines. However, outcomes from CBT can still be improved, as 30-40% of individuals fail to fully recover following treatment (Lampard & Sharbanee, 2015). This finding highlights that more research is needed to improve the effectiveness of CBT.

Improving the effectiveness of any psychological treatment requires researchers to move beyond basic questions of treatment efficacy (i.e., whether a psychotherapy works) and towards identifying when, how, and for whom such treatments work (Kazdin, 2007). This process-based research is a critical part of psychotherapy development, evaluation, and refinement; its importance is being increasingly recognised and promoted by international clinical and research guidelines, including the MRC framework and the American Psychological Association of Division 12 Task Force (Society for Clinical Psychology). What follows is a conceptual review of process-based research, the questions it can answer, how it can lead to improvements in psychological treatments, and the current state of process-based research in field of clinical psychology (including eating disorders). This chapter will form the rationale for Study 2.

Process-Based Research

In psychotherapy research, there is renewed interest in studying when, how, and for whom psychotherapy works (Laurenceau, Hayes, & Feldman, 2007). Process-based research therefore focuses on what occurs in the interval *between* pre-treatment and post-treatment.

There are three main areas of research process-based research addresses. These include (1) the trajectory of symptom change over the course of treatment; (2) the mechanisms and mediators of change; (3) moderators and predictors of change.

The trajectory of symptom change. Process research allows us to study the course of symptom change throughout treatment. RCTs typically assess group average level symptoms at pre-treatment and post-treatment, with no attention devoted toward the rich information available during the interval between this period, or in individual differences in treatment response (Laurenceau et al., 2007). Such pre-post designs cannot capture the dynamic and nonlinear process of change during treatment, which is problematic, because it is often assumed that change from pre-treatment to post-treatment is gradual and linear (Hayes, Laurenceau, Feldman, Strauss, & Cardaciotto, 2007b). However, change is rarely linear, and while participants might show similar levels of improvement from pre to post-treatment, such change may be achieved via unique trajectories (Hayes et al., 2007b).

Frequent symptom assessment during the course of therapy enables one to study the trajectory of symptom change. This trajectory can provide information about the rate of symptom change, and whether the rate of change is constant or variable. For example, psychotherapy research for depression has identified several distinct trajectories of change during CBT for depression, including an early response to treatment and sudden treatment gains (Renaud et al., 1998). Specifically, many clients receiving CBT for depression experience a rapid response to treatment, in which improvements in depressive symptoms occur mostly in the early stages (the first four weeks) of treatment (Renaud et al., 1998). Similar to a rapid response, Tang and colleagues also found that many CBT clients experience “sudden gains”, such that at least a 25% reduction in depressive symptoms is observed in one between-session interval (Tang & DeRubeis, 1999). A rapid response to

treatment and sudden gains have been shown to be robust predictors of successful short and long-term treatment outcomes (Aderka, Nickerson, Bøe, & Hofmann, 2012).

Identifying these key transition points (e.g., rapid response, sudden gains) has numerous advantages for research and practice. For example, given that a rapid response to CBT is consistently linked to successful outcome, those who fail to respond quickly to CBT might be recognised early and might therefore be offered more focused, targeted, and intensive treatment to minimize a possible poor outcome (Aderka et al., 2012). In addition, identification of these key transition points allows for an analysis of the possible factors that contribute to these abrupt symptom changes. For instance, in the treatment of depression, Hayes and colleagues found that those who experienced a rapid response to treatment reported greater change in hope and in cognitive-emotional processing immediately prior to this rapid response (Hayes et al., 2007a). Similarly, Tang and DeRubeis (1999) observed that sudden gains in depression treatment were predicted by greater cognitive change in the treatment session that immediately preceded the sudden gain ("pre-gain" session). Thus, it is argued that targeting the factors (i.e., hope, cognitive-emotional processing, and cognitive change) that promote a rapid response or sudden gains will improve the effectiveness, efficiency, and potency of psychological treatments for depression (Lorenzo-Luaces et al., 2014). These crucial non-linear patterns of change and their implications for improved treatment outcome would not have been captured by a simple pre-post assessment.

Mechanisms and mediators of change. Psychotherapy process research is also concerned with studying the mechanisms of change (Kazdin, 2007). Mechanisms, which are defined as the processes and events within treatment that causes clinical change (Kazdin & Nock, 2003), are responsible for answering questions on how and why psychotherapy works. Knowledge about treatment mechanisms is crucial for improving the potency, efficiency and

delivery of psychological interventions. If mechanisms are known, then research can focus on enhancing the effective therapeutic elements known to trigger these mechanisms, while also removing the ineffective therapeutic elements (Murphy et al., 2009). Furthermore, pinpointing mechanisms of change would also enhance our understanding of the nature of psychiatric disorders and the variables associated with their course (Kraemer, Wilson, Fairburn, & Agras, 2002). For example, if a psychotherapy approach "works" because it eliminates a particular psychopathological process, then this would suggest that this particular process is central to the maintenance of that psychiatric disorder (Kraemer et al., 2002).

Pinpointing *mediating* variables is a crucial first step toward uncovering the mechanisms of change (Kazdin, 2007). Mediators are variables that *statistically* explain the effects of treatment on an outcome variable (Laurenceau et al., 2007). Thus, mediators represent *potential* mechanisms. By definition, all mechanisms are mediators, but not all mediators are necessarily causal mechanisms (Kazdin, 2007). Thus, a focus on statistical mediators as a first step narrows down the search for potential causal mechanisms.

There are several requirements to establishing mediating variables. Traditionally, mediation effects in psychological research were tested statistically by the method proposed by Baron and Kenny (1986). Although Baron and Kenny's approach to mediation was typically applied in social psychological research, it is also been used in clinical research (for a discussion, see Lemmens, Müller, Arntz, & Huibers, 2016), and serves as an important comparison for more recent and appropriate methods to test *treatment* mediators (as discussed in the next paragraph). When applying the Baron and Kenny approach to psychotherapy research, four conditions must be met for a variable to be considered a mediator: (1) there is a main effect of treatment; (2) there is a relationship between treatment and the mediator; (3) changes in the mediator are correlated with changes in the outcome; (4)

the effect of treatment on the outcome is absent or reduced when controlling for the mediator (Baron & Kenny, 1986).

As Baron and Kenny's approach to statistical mediation was not developed specifically for testing mediators of treatment effects, the MacArthur Foundation Network Group modified these criteria to make mediation analyses more applicable to clinical research (Kraemer et al., 2002). According to the MacArthur approach, to demonstrate statistical mediation, a change in a hypothesised mediating variable would have to be observed during treatment, then this change must correlate with treatment type (i.e., because it was the treatment that induced this change), which then must *either* have a main or interactive effect with the outcome variable (Kraemer et al., 2002).

The MacArthur approach differs to the Baron and Kenny approach in a few ways: First, although considered desirable, the Baron and Kenny approach does not require that the independent variable temporally precedes a change in the mediator. The MacArthur approach, on the other hand, stipulates that changes in the mediator must occur after the onset of treatment (i.e., correlate with treatment type). Second, unlike the Baron and Kenny approach, the MacArthur approach of mediation does not require a main effect of treatment for mediation to be tested. This means that mediation analyses in the MacArthur approach *can* still be performed in RCTs that demonstrate equivalence between two psychological treatments (no main effect of treatment). This is important for testing theory-specific mediators of change at the same time in two opposing, yet equally effective, psychotherapies (Kraemer et al., 2002). Third, in contrast to the Baron and Kenny approach, the MacArthur approach of mediation always includes a term for the interaction between the independent variable and the mediator in the linear model. An interaction term is included because it can identify cases when a mediator explains the effect of one treatment but not for another equally effective treatment (Kraemer et al., 2002). Fourth, whereas the Baron and Kenny

approach is strongly focused on null hypothesis significance testing, the MacArthur approach strongly emphasises the effect size of the independent on the dependent variable. Hence, the MacArthur approach is considered a hypothesis-generating approach, where each mediation finding is considered an important step for building on future hypothesis-testing studies.

A mediator that is identified from the methods described above provides the necessary but not sufficient steps to establish what Kazdin (2007) refers to as a "true" mediator of change. Kazdin (2007) outlined five *additional* criteria (further to a strong association and temporal precedence) required for a variable to be considered a mediator of treatment. First, *specificity* of the relationship between treatment, mediator, and outcome must be established. That is, clinical change should be explained by specific mediating variables and not by several other factors. Second, *consistency* must be demonstrated, such that the observed mediation effects are replicated across studies, samples, and conditions. Third, *experimentally manipulating* the mediator and observing its effect on the outcome should be performed, as experimental evidence strengthens the case that the mediator accounts for change in that outcome. Fourth, a *gradient* should be shown, such that greater activation of the mediator is shown to predict greater clinical change. Fifth, inclusion of *plausible processes* (i.e. factors that might explain precisely what the mediator does and how it works to affect the outcome) should also be tested directly (Kazdin, 2007).

Kazdin (2007) contends that drawing inferences about a treatment mediator requires convergence of evidence stemming from these multiple criteria. That is, no single study can determine the presence or absence of a mediator. By definition, consistency and replication is required. Thus, a mediator is only established by numerous studies that address different criteria; when all criteria are met across studies, then one can claim that the particular mediating variable is responsible for the treatment effects.

Moderators and predictors of change. Moderators are *baseline,* pre-randomized characteristics that *interact with treatment type* to affect outcome (Kraemer et al., 2002). Moderators therefore specify for whom, and under what condition, a treatment works. Moderators, by definition, are not correlated with treatment type; this means that moderators must (a) be assessed prior to randomization, (b) not change as a result of treatment (because they are not correlated with treatment type), and (c) not explain the effects of treatment on outcome (Kraemer, 2016). However, an interaction between the moderator and treatment means that the effect of a particular treatment *depends* on participants' level of the moderator (prior to randomization). For these reasons, moderators can only be examined in RCTs.

There are numerous benefits to studying moderators. Moderators can aid in treatment matching, such that clinicians are better equipped to decide on which treatment might be most effective for a certain client (Laurenceau et al., 2007). For example, if CBT is highly effective for individuals with low self-esteem (the moderator), then individuals who present to treatment with low self-esteem might be matched CBT over a different treatment. In addition, moderators also help clarify the best choice of inclusion and exclusion criteria to optimize statistical power in RCTs (Kraemer et al., 2002). For example, consider that CBT for depression is only effective for women and not for men (gender is the moderating variable). If this is the case, then future RCTs might restrict their sample to females. Thus, not only would statistical power increase, but resources could be reserved for providing males with an alternative, more effective treatment. For these reasons, moderators are crucial toward ensuring that clients are provided with a treatment that best suits their needs.

Predictors are also baseline variables that correlate with treatment outcome. In the context of RCTs, variables that correlate with outcome, *irrespective of treatment type,* are termed non-specific predictors (Kraemer et al., 2002). Unlike treatment moderators,

predictors of outcome can also be examined in single-treatment pre-post designs (e.g., open trials). Identifying predictors of outcome for a specific treatment is important for prospectively distinguishing between treatment responders and non-responders (Agras et al., 2000a). Thus, predictors can be identified irrespective of whether a comparison is delivered, and are crucial for providing information about how treatment can be tailored toward the needs of a particular individual.

The Current Status of Psychotherapy Process Research

Clinical researchers are increasingly asking questions about when, how, and for whom particular treatments work (Lorenzo-Luaces et al., 2014). Mediators, moderators, and predictors of response are being studied across several psychiatric disorders, including depression (Lorenzo-Luaces et al., 2014), anxiety (Gallagher et al., 2013), and eating disorders (Agras et al., 2000a). The growing interest in process research was recently highlighted by Schneider, Arch, and Wolitzky-Taylor (2015), who reported that over 70% of studies that have assessed treatment moderators across anxiety disorders were published after 2009.

Recent systematic reviews have synthesised the evidence and attempted to identify reliable mediators, moderators, and predictors of response for various psychological disorders. Two recent systematic reviews synthesised the evidence related to mediators of response following mindfulness-based cognitive therapy (MBCT) for mental health conditions (Gu, Strauss, Bond, & Cavanagh, 2015) and MBCT specifically for depression (van der Velden et al., 2015). Both review authors concluded that there was preliminary evidence to suggest that MBCT *might* "work" because of the reasons specified by the underlying theory, i.e., through enhancing mindfulness and psychological flexibility. These conclusions were based on the fact that these hypothesized mechanisms were shown to

consistently mediate treatment outcome. However, both review authors noted that most included studies failed to establish temporal precedence between the mediator and outcome, which prevented them from concluding that MBCT leads to or *causes* changes in these mechanisms, which in turn *causes* symptom change (Gu et al., 2015; van der Velden et al., 2015). Another review investigated whether threat reappraisal mediated symptom improvement during CBT for anxiety disorders (Smits, Julian, Rosenfield, & Powers, 2012). The authors found that while there was strong evidence demonstrating that threat reappraisal was *associated* with symptom improvement, key criteria necessary for establishing mediation (Kazdin, 2007), including temporal precedence and treatment specificity, were not met across studies. These reviews are important to discuss because they provided crucial insights toward the role of several potential explanatory mechanisms of CBT for anxiety disorders, and have since prompted more research in this field.

Many more systematic reviews have focused on identifying reliable moderators and predictors of response following CBT for depressive and anxiety disorders. For example, reliable *predictors* of poor outcome for anxiety disorders include comorbid depressive and personality disorders, higher baseline symptom severity, agoraphobic avoidance, and lower expectancy to change (Eskildsen, Hougaard, & Rosenberg, 2010; Knopp, Knowles, Bee, Lovell, & Bower, 2013; Porter & Chambless, 2015). Reliable *predictors* of poor outcome for depressive disorders include higher baseline symptom severity and comorbid anxiety disorders (Nilsen, Eisemann, & Kvernmo, 2013). Reliable *moderators* are scarce; reviews have shown that CBT for anxiety disorders is less effective than alternative psychological treatments (i.e., psychodynamic therapy and MBCT) in individuals with comorbid personality disorders and higher baseline levels of depression (Schneider et al., 2015), whereas CBT for depression has been shown to be more effective than alternative psychological treatments for older people and in individuals with comorbid addictive

disorders (Cuijpers, Ebert, Acarturk, Andersson, & Cristea, 2016c). These findings have prompted calls for the next generation of research to test and improve the efficacy of treatment outcomes by using a moderator-to-allocate RCT design (Kraemer, 2016) .

Process Research in Eating Disorders

Eating disorder treatment research is also beginning to focus more on identifying reliable mediators, moderators, and predictors of treatment response. Process-based research is a priority to advance the eating disorder field (Murphy et al., 2009).

Several systematic reviews have synthesized research regarding predictors of response (Berkman et al., 2007; Shapiro et al., 2007), and only one review has synthesised the findings on moderators and mediators of response (Vall & Wade, 2015). Some reviews concluded that most predictors explored were unrelated to BN outcome (Steinhausen & Weber, 2009) and BED (Shapiro et al., 2007), while others concluded that high levels of psychopathology and poorer interpersonal functioning were consistent predictors of poor AN outcome (Berkman et al., 2007).

Since the publication of the abovementioned reviews, several other studies have tested predictors, moderators, and mediators of outcome. This prompted Vall and Wade (2015) to conduct an updated meta-analysis on mediators, moderators and predictors of response across all psychological and pharmacological treatments. Vall and Wade (2015) concluded that consistent *predictors* of poor outcome included higher baseline levels of shape and weight concern, and binge eating and purge frequencies, while a consistent predictor of successful outcomes was greater motivation to change. No reliable moderators emerged in this review. The mediational process of an early response to treatment was also found to be associated with successful outcomes (Vall & Wade, 2015).

Although the above mentioned reviews have provided important insights toward the types of factors that might influence treatment outcome across eating disorders, there are still important questions left unanswered. For instance, each review based their conclusions on mediators, moderators, and predictors tested in studies that administered several distinct psychological, pharmacological, or behavioural treatments. Thus, variables that might be a consistent predictor for one treatment but not for others might have been masked in an analysis that aggregates these disparate treatment approaches. Moreover, because psychological treatments are hypothesised to contain several theory-specific mechanisms of action (Murphy et al., 2009), these reviews would not have been able to capture mediators specific to any one treatment. Thus, we still do not know whether any consistent mediators, moderators, or predictors of response exist specifically for CBT.

Chapter Summary

This chapter provided an overview of psychotherapy process-based research. Process research seeks to understand the trajectory of symptom change during the course of treatment, the mediators and mechanisms that account for symptom change, and the factors that moderate or predict change. This chapter also demonstrated that, relative to the depression and anxiety literature, little work has been done to understand the factors that mediate, moderate, and predict outcome for CBT for eating disorders. A number of studies have sought to answer such questions, yet their findings have not been synthesised and evaluated. The objective of Study 2 is to therefore synthesise and appraise the literature on studies that have tested mediators, moderators, and predictors of CBT outcomes for individuals with eating disorders. It is an overarching goal to identify consistent and robust factors that affect CBT outcomes, with the intention of understanding how, why, and for whom CBT works.

Chapter 4: Methodological Rationale for Article 1 and 2

Overview

This chapter outlines the methodological rationale for Study 1 and Study 2 of this thesis, both of which are systematic literature reviews. The specific procedural details of the systematic reviews (e.g., the databases searched, the study selection process) are provided in each article. This chapter will therefore focus on providing a brief rationale for using a systematic review and meta-analysis for these studies.

Systematic Reviews

A systematic review is an appraisal and synthesis of primary research papers using a rigorous and clearly specified methodology both for the search strategy and for the study selection process (Moher, Liberati, Tetzlaff, & Altman, 2009). Unlike narrative reviews, systematic reviews look exhaustively for all available studies that have addressed a particular research question (Higgins & Green, 2011b). Because systematic reviews use a pre-specified eligibility criteria and an explicit reproducible methodology to answer a specific research question, potential sources of bias (e.g., selective citing) are minimized. Systematic reviews may or may not be accompanied by a meta-analysis, which uses statistical procedures to summarize, combine and quantify the results of multiple independent studies (Lipsey & Wilson, 2001).

In the late 1980's, it was noted that the quality of systematic reviews and meta-analyses was suboptimal (Sacks, Berrier, Reitman, Ancona-Berk, & Chalmers, 1987). For example, Sacks et al. (1986) evaluated the quality of 86 meta-analyses published during the period from 1966 to 1986. The authors evaluated these reviews against 23 quality

characteristics covering six broad domains (study design, combinability, control of bias, statistical analysis, sensitivity analysis, and application of results). They found that only 28% of reviews adequately addressed all six quality criteria. This led to the conclusion that there was a need for improved reporting in meta-analyses.

To address the suboptimal reporting of meta-analyses, an international group of researchers developed a set of guidelines for systematic review and meta-analysis reporting (Moher et al., 2009). These guidelines were initially named the Quality of Reporting of Meta-Analyses (QUOROM) statement (Moher et al., 2000), but were since revised to the Preferred Reporting Items for Systematic Reviews and Meta-analyses (PRISMA) statement (Moher et al., 2009). The primary objective of the PRISMA statement was to help authors improve the reporting of systematic reviews and meta-analyses. To achieve this goal, the PRISMA statement devised a four phase diagram outlining the stages of study selection, and also a 27-item checklist (Moher et al., 2009). Items that form the checklist fall under six sections (title, abstract, introduction, methods, results, and discussion). These items are provided to ensure that potential sources of bias from systematic reviews are minimized, and that the reporting of a systematic review is comprehensive enough to allow for the systematic review to be replicated or updated (Moher et al., 2009). Many journals now require authors to submit the PRISMA diagram and checklist with their review.

The quality of reporting in systematic reviews has improved since the development of the PRISMA statement. For example, Tunis, McInnes, Hanna, and Esmail (2013) assessed the quality of reporting of 130 systematic reviews published in radiology journals between 2007 and 2011. They found that systematic reviews reported on average 22.6 of the 27 items after the publication of the PRISMA statement, compared to an average reporting of 20.9 items before its publication. The quality of reporting following the publication of the

PRISMA statement has also improved in other major research areas, including medical (Ge et al., 2014) and nursing (Jin, Ma, Gao, Hua, & Dou, 2014) research.

Strengths. There are several advantages to systematic reviews and meta-analyses. First, it is very difficult for clinicians, researchers, and policy makers to incorporate and consider a large body of literature into their decision making. By comprehensively and objectively synthesising all the research findings relevant to a research question into a single document, systematic reviews make it possible for consumers to keep up to date with this literature. If systematic reviews on a particular topic are lacking, then the decisions made by clinicians, researchers, and policy makers are likely to be based on a smaller set of research findings selected in a non-systematic way, which has the potential to skew clinical decision making. Thus, systematic reviews provide a more objective avenue for clinical and policy decision making, and, as a result, an opportunity for research to influence practice and policy making (Bartolucci & Hillegass, 2010).

Second, systematic reviews and meta-analyses help reduce implicit researcher bias. For instance, traditional narrative reviews are typically restricted to literature that is either already known to the authors, or obtained through rudimentary, exploratory searches. As a consequence, the same studies are frequently cited, analysed, and critiqued, resulting in potential biases to the conclusions from narrative reviews (Garg, Hackam, & Tonelli, 2008). Because a systematic review adopts comprehensive search strategies, predefined search terms, and transparent inclusion/exclusion criteria, systematic reviews ensure that researchers are searching comprehensively for all the relevant literature. This improves the chance of obtaining clearer and objective answers to a pre-defined research question (Higgins & Green, 2011a).

Third, combining results from multiple independent studies through meta-analytic procedures provides more accurate estimates of relationships between variables. More

specifically, many studies do not have sufficient power to detect statistically significant group differences, particularly when group differences are small in magnitude. By aggregating data from multiple studies and computing an overall "summary effect", meta-analyses have greater power to detect significant effects (Borenstein, Hedges, Higgins, & Rothstein, 2009). Thus, inconsistent or conflicting findings across these studies are better answered in meta-analysis. In addition, a meta-analysis has the ability to further explore the possible reasons for conflicting findings through moderator analyses. Moderator analyses enable examination of whether the magnitude of effect differs as a function of various study characteristics (Borenstein et al., 2009), thereby providing information on when, and under what set of circumstances, a particular effect is present, absent, or strongest.

Limitations. It is important to also acknowledge the limitations of systematic reviews and meta-analyses. First, systematic reviews and meta-analyses have been criticized for combining studies that are too heterogeneous (e.g., comparing "apples and oranges"; Lyman & Kuderer, 2005). Computing an overall effect from heterogeneous studies (e.g., different samples, settings, and interventions) is said to misrepresent or even simplify certain complex relationships. However, multiple methods do exist to combat the impact of potential sources of heterogeneity. For example, sources of heterogeneity can be explored through moderator analyses, as discussed earlier (Higgins & Thompson, 2002). In addition, because a large degree of between-study heterogeneity is common in meta-analyses, it is recommended that random effects statistical models are used over fixed effects models. In a random effects model, it is assumed that the included studies are drawn from "populations" of studies that differ from each other systematically. The effect sizes resulting from included studies in the random effects model not only differ because of the random error within studies (which is the basic assumption of fixed effects models), but also because of true variation in the effect size from one study to the next (Borenstein et al., 2009).

Another limitation of systematic reviews and meta-analyses is their reliance on published data. Negative or non-significant results are less likely to be published (Rosenthal, 1991), so only including published studies is likely to overestimate or exaggerate the size of a particular effect. There are, however, several methods available to minimize the impact of publication bias. For example, review authors are encouraged to seek out any relevant unpublished data. In addition, statistical methods, such as, for example, the Fail-Safe *N* (Rosenthal, 1991) and Duval and Tweedie's (2000) trim and fill method, are available to test and correct for the impact of potential publication bias. For this reason, the PRISMA guidelines recommend testing for publication bias.

Finally, systematic reviews, particularly in clinical research, are sometimes criticised (Petticrew, 2003) because they are often unable to provide specific guidance or recommendations on questions that require a conclusive answer (e.g., which intervention is most effective). It is common for reviews in clinical research to conclude that little or inconsistent evidence exists for a particular research question. This, however, is not a limitation of the methodology of systematic reviews; rather, it is a result of the available research that is being evaluated. It is therefore important to carefully consider whether a systematic appraisal of a particular literature is likely to advance the field. It is also important to recognise that absence of clear evidence for a particular research question will still inform future work on what is required to advance a particular area of study.

Choice of measures of effect. Choosing which measure of effect size to use in a meta-analysis is an important consideration. For continuous outcomes, meta-analysts may either use the raw mean difference or the standardized mean difference. The raw mean difference is typically used when the same scale or measurement tool is used for a particular outcome. When different scales are used to assess similar constructs, the standardized mean difference should be selected because scales are converted to a unit that allows for

comparisons across measures or studies. Because measures of eating disorder psychopathology are typically assessed through different scales, this thesis therefore used the standardized mean difference. Each standardized mean difference was then converted to Hedge's *g*. Hedges *g* was used because it takes into account the potential influence or biases due to small sample sizes (Borenstein et al., 2009).

On the other hand, binary outcomes may be expressed as either an odds ratio or a risk ratio. An odds ratio is a measure of the effect size that is defined as the ratio of the odds of an event occurring in one group to the odds of the event occurring in the comparison group (Bland & Altman, 2000). The risk ratio is ratio of the probabilities of achieving an event between the two conditions (Lipsey & Wilson, 2001). Both measures are commonly used in meta-analyses of binary outcomes. However, while the risk ratio is said to be easier to interpret than the odds ratio, there is some evidence showing that the risk ratio may overestimate an effect when the prevalence of a particular event is greater than 10% (Bland & Altman, 2000). To take a conservative approach, the odds ratio was calculated in this thesis over the risk ratio.

The use of systematic reviews and meta-analysis in this thesis. Two systematic reviews were completed in this thesis. The first was a systematic review and meta-analysis that aimed to examine the efficacy of CBT for eating disorders. Meta-analytic procedures were used, as the goal was to quantify the magnitude of the effect of CBT interventions relative to various comparison groups. The second was a systematic review that aimed to synthesise the literature on mediators, moderators, and predictors of response to CBT for eating disorders. Due to the considerable level of heterogeneity (e.g., type of variables explored, mode of treatment delivery, sample characteristics, etc; see Chapter 6) observed in the studies that were included in the second review, a qualitative data synthesis was

performed over a quantitative data synthesis. Both reviews were conducted in accordance to the PRISMA guidelines.

Chapter 5: The Efficacy of Cognitive-Behavioral Therapy for Eating Disorders: A Meta-Analysis (Article 1)

This chapter consists of an article accepted for publication in the *Journal of Consulting and Clinical Psychology* (Linardon et al., in press). As outlined in Chapter 2, a proliferation of RCTs of CBT have been published to date, and although attempts to synthesize the findings of these trials have been made, previous systematic reviews and meta-analyses have left important questions unanswered. This meta-analysis therefore aimed to evaluate the empirical status of CBT for eating disorders. This meta-analysis was not prospectively registered.

Abstract

Objective: This meta-analysis examined the efficacy of cognitive-behavioural therapy (CBT) for eating disorders. **Method:** Randomized controlled trials of CBT were searched. Seventy-nine trials were included. **Results:** Therapist-led CBT was more efficacious than inactive (wait-lists) and active (any psychotherapy) comparisons in individuals with bulimia nervosa and binge eating disorder. Therapist-led CBT was most efficacious when manualized CBT-BN or its enhanced version was delivered. No significant differences were observed between therapist-led CBT for bulimia nervosa and binge eating disorder and antidepressants at post-treatment. CBT was also directly compared to other specific psychological interventions, and therapist-led CBT resulted in greater reductions in behavioural and cognitive symptoms than interpersonal psychotherapy at post-treatment. At follow-up, CBT outperformed interpersonal psychotherapy only on cognitive symptoms. CBT for binge eating disorder also resulted in greater reductions in behavioural symptoms than behavioural weight loss interventions. There was no evidence that CBT was more efficacious than behaviour therapy or non-specific supportive therapies. **Conclusions:** CBT is efficacious for eating disorders. Although CBT was equally efficacious to certain psychological treatments, the fact that CBT outperformed all active psychological comparisons and interpersonal psychotherapy specifically, offers some support for the specificity of psychological treatments for eating disorders. Conclusions from this study are hampered by the fact that many trials were of poor quality. Higher quality RCTs are essential.

Cognitive-behavioural therapy (CBT) is the most widely investigated eating disorder treatment. Randomised controlled trials (RCTs) demonstrate that specific forms of CBT produce large improvements in eating disorder symptoms in individuals with bulimia nervosa (BN), binge eating disorder (BED), Other Specified Feeding and Eating Disorders (OSFED), and anorexia nervosa (AN) (Byrne et al., 2017; Fairburn et al., 2015; Fairburn et al., 1991). Clinical guidelines recommend specific forms of CBT as the treatment of choice for these BN, BED, and OSFED, and also as one of the front-running treatments for AN (Hay et al., 2014; Herpertz et al., 2011; National Institute of Clinical Excellence, 2017).

The results across RCTs have been synthesized in meta-analyses. A summary of these meta-analyses is presented in Table 1 (Appendix C) of the Supplementary Materials. Compared to wait-list or active controls, therapist-led CBT consistently results in greater improvements in eating disorder symptoms in BN and BED (Hay et al., 2009). Moreover, specific modes (e.g., E-therapy CBT) or formats (e.g., group-based CBT) have also been shown to be superior to wait-list controls in BN and BED (Loucas et al., 2014; Polnay et al., 2014). In contrast, one meta-analysis has examined the effects of CBT for AN (Hay et al., 2015), estimating effect sizes for two comparisons: CBT compared to treatment as usual, and CBT compared to interpersonal psychotherapy (IPT) or short-term focal psychodynamic therapy. Effect sizes were based on two studies, and the pooled effect size was not significantly different from zero for BMI and eating disorder symptom outcomes. These findings only included only two effect sizes, and some RCTs of CBT for AN (Touyz et al., 2013) were excluded from this review because the focus of the review was on treatment that assertively promoted weight gain. The power of these comparisons could be improved by comparing CBT to all available active controls in all available studies that have sampled individuals with AN. In sum, there is evidence supporting the efficacy of CBT for BN and

BED, and while numerous trials have documented the efficacy of CBT for AN, CBT has not been shown to outperform comparison interventions.

Important Questions to be Addressed

Previous meta-analyses of CBT for eating disorders have not addressed several important questions. First, recent meta-analyses of CBT for eating disorders have not assessed the effect of CBT on cognitive symptoms. Pooled effect sizes have only been calculated for binge eating and/or purging behaviour. There is evidence to suggest that individuals with eating disorders who are considered clinically recovered because of their abstinence from binge eating or purging still report significant cognitive symptoms (Keski-Rahkonen et al., 2009). This is concerning, as residual cognitive symptoms following CBT have been shown to predict relapse in BN (Fairburn et al., 1993c). Consequently, authors have recently argued that treatment success should be based on both behavioural and cognitive symptoms (Williams et al., 2012).

Second, each meta-analysis has focused only on the effects of CBT for a specific eating disorder diagnosis. The growing interest on transdiagnostic theories across different psychopathologies means that more RCTs are delivering CBT to individuals across diagnostic criteria (Fairburn et al., 2015; Fairburn et al., 2009). It is therefore not known whether CBT is an effective treatment for transdiagnostic samples. An updated meta-analysis including all eating disorder presentations is also timely pertinent.

Third, the relative short and long-term effects of CBT and pharmacological treatments for eating disorders are unknown. Antidepressants are recommended for treating BN and BED, as antidepressants have been shown to outperform placebo-controls (Brownley et al., 2016; Hay et al., 2014). Indeed, an early meta-analysis that compared therapist-led CBT for BN to antidepressants found no significant differences in behavioural remission rates at post-treatment (Hay et al., 2001). Since 2001, several additional trials comparing CBT to

antidepressants have been conducted, so an updated meta-analysis of these comparisons is required. Moreover, it is also unknown whether the equivalence observed between CBT and antidepressants is (a) sustained at follow-up, (b) generalises to cognitive symptoms, and (c) occurs in individuals with BED or OSFED.

Fourth, few moderators of the effectiveness of CBT have been assessed. Identifying moderators is important for enhancing understanding of the specific conditions under which CBT is most effective. Of the few moderators tested (i.e., CBT modality, the use of homework, therapist pre-training, and therapist allegiance), none have been found to relate to CBT's effectiveness (Spielmans et al., 2013). Thus, given that the specific conditions that are associated with CBT's effectiveness have not been identified, it is important to test additional moderating variables so that we can have a clearer understanding of the circumstances and conditions that make CBT more or less effective and for whom they do so.

One potentially important moderator that has not been investigated is the type of cognitive-behavioural protocol delivered. Although several overlapping but distinct CBT protocols for eating disorders exist, a specific manualized form of CBT developed by Fairburn and colleagues' is recommended as the treatment of choice. This treatment was originally designed as a treatment for BN (CBT-BN), but it has since been enhanced to have a transdiagnostic scope (CBT-E; Fairburn, 2008). Both CBT-BN and CBT-E are designed to disrupt the maintaining mechanisms that are outlined in their underlying cognitive-behavioural model, which is empirically supported (Pennesi & Wade, 2016). CBT-BN typically consists of 19 individual treatment sessions. CBT-E typically consists of 20 individual treatment sessions for normal weight eating disorders, and 40 individual treatment sessions for underweight eating disorders (Fairburn, 2008). Although some have suggested that CBT-E might be superior to CBT-BN and other CBT protocols, no trials have directly compared these protocols. Thus, a first step in determining their relative efficacy is to

examine and compare the size of the effect for trials that have administered these distinct cognitive-behavioural treatment protocols.

The fifth question yet to be addressed is whether CBT outperforms other *specific* psychological treatments. To date, three meta-analyses have directly compared CBT to other specific psychological treatments on behavioural symptoms. Two of these compared CBT to behavioural interventions (Hay et al., 2009; Spielmans et al., 2013). While Hay et al (2009) found CBT to outperform behavioural interventions (k=4) on rates of remission in individuals with BN, Spielmans et al (2013) reported no significant difference in outcomes between these treatments in individuals either with BED (k=4) or with BN (k=8). The third meta-analysis directly compared CBT to IPT on behavioural outcomes for BN and BED (Cuijpers et al., 2016b). The authors found a small but statistically significant effect (g= 0.20) in improved behavioural symptoms at post-treatment in favour of CBT. However, these analyses did not include a comparison at follow-up. The importance of such direct comparisons of CBT to other psychological treatments is twofold. First, these may provide direct and stronger evidence of the relative efficacy of CBT, and may therefore confirm current clinical practice guidelines which recommend CBT over other psychological interventions. Second, if they find evidence indicating the superiority of CBT, the result would (a) provide support for the theoretical model underpinning the CBT, and (b) challenge the widely endorsed common factors model.

The Current Meta-Analysis

Over 15 RCTs of any mode of CBT for eating disorders have been published since the last broadly focused meta-analysis. It is therefore timely and pertinent to conduct an updated meta-analysis on the efficacy of CBT for eating disorders, addressing the unanswered questions listed above. The current meta-analysis has three specific aims: First, we aim to investigate whether CBT for each eating disorder presentation is more efficacious than

inactive (e.g., wait-list), active (e.g., alternative psychotherapy approaches), and pharmacological comparisons at post-treatment and follow-up. Second, we aim to test whether these effects are moderated by sample age, CBT format, and CBT protocol. Third, we aim to perform meta-analyses directly comparing CBT to specific alternative psychological treatments at post-treatment and follow-up.

Method

Search Strategy

Five online databases were searched in June 2017: Medline (421 hits), PsycINFO (436 hits), EMBASE (546 hits), CINAHL (181 hits), and the Cochrane library (387 hits). The following terms were combined using the "AND" Boolean operator and searched in the five databases: *eating disorder, bulimi*, anorexi*, EDNOS, OSFED, bing*,* AND *CBT*, cognitive-behav, cognitive behav*,* AND *random*, trial*, RCT, controlled, allocat*, assign*.* Additional searches were conducted to obtain data from unpublished trials. Using the same key terms, several databases containing grey literature were searched: PsycEXTRA (18 hits), ProQuest Central (25 hits), and PsycINFO (21 hits). All authors from the included published trials studies were contacted with a request for unpublished data. Clinical trials registries were also searched for ongoing trials (3 hits). A flowchart of the search strategy is presented in Figure 5.1.

Inclusion Criteria

Studies were included that (a) administered CBT (b) to individuals with any diagnosis of an eating disorder (c) in a RCT where (d) an inactive (e.g., wait-list), active (i.e., a non-CBT psychological treatment), or pharmacological comparison was administered. We excluded trials that either (a) only compared variants of CBT (e.g., group vs. individual

format), or (b) administered a multidisciplinary treatment (i.e., included aspects of CBT and aspects of other distinct psychological treatment approaches).

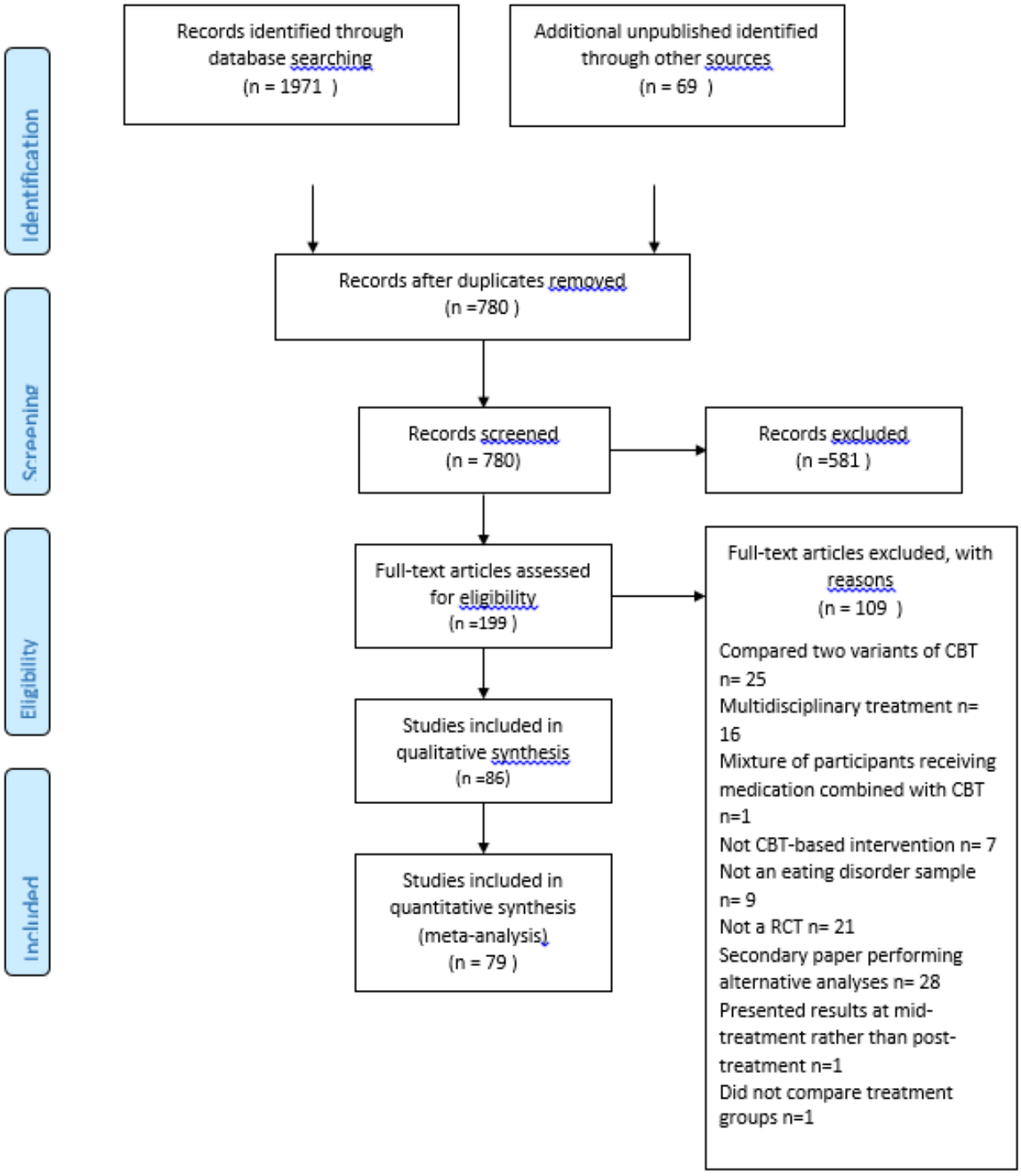


Figure 5.1: Flowchart of Literature Search

Study Selection

Duplicate records were removed once the search strategy outputs were combined. Titles and abstracts were screened to identify studies that administered CBT to individuals with eating disorders. Full-texts of these articles were read to see whether full inclusion criteria were met. All studies that met inclusion criteria were again screened to determine eligibility for the meta-analysis. Eighty-six studies met full inclusion criteria, and 79 studies were included in the meta-analysis. An effect size could not be calculated from five studies as insufficient data were reported and further data could not be obtained (Channon, de Silva, Hemsley, & Perkins, 1989; Fairburn et al., 1986; Freeman et al., 1985; Serfaty, Turkington, Heap, Ledsham, & Jolley, 1999) and two studies did not assess an outcome relevant to the current meta-analysis (Bhatnagar, Wisniewski, Solomon, & Heinberg, 2013; Robinson & Serfaty, 2008).

Quality Assessment

The validity of trials was assessed using four of the criteria of the Cochrane Risk of Bias tool (Higgins & Green, 2011b). This risk of bias tool assesses potential sources of biases in RCTs, such as the adequate generation of allocation sequence, the concealment of allocation to treatment conditions, blinding of outcome assessors, and dealing with incomplete data. Dealing with incomplete data was assessed as low risk when ITT analyses were conducted. The two other criteria of the Cochrane Collaboration tool were not used; there was no indication that there were selective outcome reporting or other potential sources of bias, consistent with previous systematic reviews (Cuijpers, Cristea, Weitz, Gentili, & Berking, 2016a; Hay, 2013). The first author and an independent research assistant performed the quality assessment. Assessments were cross-checked, and any disagreement was discussed in detail.

Meta-Analysis

We compared CBT to (1) inactive comparisons, which included wait-list or treatment as usual (TAU) conditions[2], (2) active psychological comparisons, which included any other psychotherapy condition; and (3) pharmacological comparisons (any medication). If a study compared CBT to multiple conditions that fell within the same comparison category (two active psychological comparisons), then the sample size of the CBT condition was halved to avoid double counting (Borenstein et al., 2009). Analyses were performed at post-treatment, short-term follow-up (< 12 months) and long-term follow-up (≥ 12 months), unless otherwise indicated. Analyses were performed separately for AN, BN, and BED studies. However, eight trials studied a transdiagnostic sample. For each of these, we determined the diagnosis that occurred most frequently in the sample, and included that study in one of the BN or BED analyses mentioned above. Note that none of these eight transdiagnostic trials were included in the AN analyses.

For continuous outcomes (see outcomes below), Cohen's *d* was initially calculated by dividing the difference between the post-treatment CBT group mean and the post-treatment comparison group mean by the pooled standard deviation (Borenstein et al., 2009). If means and standard deviations were not reported, *d* was calculated using conversion equations from significance test statistics. To correct for biases due to small sample size, *d* was converted to Hedges' *g*. To calculate a pooled effect size, each study's overall effect size was weighted by its inverse variance. Positive *g's* indicates that the CBT condition scored better on a particular outcome than the comparison. Small (0.2), medium (0.5) and large (0.8) effects were specified.

[2] Since participants who are assigned to a wait-list condition typically receive some form of TAU, we merged studies that used a wait-list with studies that used a TAU condition for this comparison.

For binary outcomes (remission rates), we calculated the odds ratio (OR) and 95% confidence intervals (CI). The OR is a measure of the effect size that is defined as the ratio of the odds of an event (remission) occurring in the CBT group to the ratio of the event in the comparison group another group. An OR of 1 indicates that the event is equally likely in both conditions. Effect sizes were coded so that ORs greater than 1 indicate that remission was significantly more likely in the CBT group. A small (1.68), medium (3.47) and large (6.71) OR was specified.

Primary outcomes included (1) *remission from binge eating and/or purging* (i.e., cessation of binge eating and/or purging in the last 28 days), (2) *binge/purge frequencies* (i.e., the number of objective binge eating and/or purging episodes over the past 28 days), and (3) *global cognitive symptoms.* For the global cognitive symptoms outcome, we prioritised and selected the interviewer-based or self-report version of the Eating Disorder Examination (Fairburn & Beglin, 1994)global score when reported. However, if studies reported multiple subscales from the EDE (or EDE-Q) or subscales from other measures that assess cognitive symptoms (e.g., EDI), we computed separate effect sizes for each subscale and averaged them to create one overall, omnibus cognitive symptoms effect size.

There were instances where a study reported multiple dependent measures for one of the outcome categories listed above. For example, studies often reported *both* binge eating and purging. In such cases, an aggregated effect size for the study was computed from the mean of the individual effect sizes and the pooled variance, assuming the most conservative correlation (r =1.0) between the outcomes (Tolin, 2010).

Heterogeneity

Pooled effect sizes were calculated using the Comprehensive Meta-analysis program (Borenstein et al., 2009). Since we expected considerable heterogeneity among the studies, a random effects model was used for all analyses. Heterogeneity was assessed through the I^2

statistic. The I^2 statistic assesses the degree of heterogeneity, where a value of 0% indicates no observed heterogeneity, 25% low heterogeneity, 50% moderate heterogeneity, and 75% as high heterogeneity (Higgins & Thompson, 2002).

Subgroup Analyses

For the subgroup analyses, a pooled effect size was calculated for each subgroup, and a test was conducted to determine whether the effect sizes for subgroups differed significantly from each other. A mixed effects model was used, which pools studies within a subgroup using a random effects model, but tests for differences between subgroups using a fixed effects model (Borenstein et al., 2009). Significant differences between subgroups are tested by the $Q_{between}$ statistic. Subgroup analyses were conducted for the following characteristics.

- *Sample age*: Adult or adolescent (≤ 18 years) sample.
- *Therapist-led CBT format*: Individual face to face or group face to face,
- *Therapist-led CBT type*: Fairburn and colleagues' CBT-BN or CBT-E; adaptations/abbreviated versions of CBT-BN, or other cognitive-behavioural protocols or approaches (see Supplementary Table 2). Adapted or abbreviated versions of CBT-BN were coded together when (a) shorter versions of the original treatment were delivered, (b) additional cognitive and/or behavioural strategies were incorporated within the original protocol, or (c) strategies from the original CBT-BN or CBT-E manual were removed.
- *Specific therapist-led manualized CBT type*: Manualized CBT-BN or manualized CBT-E.
- *Self-help format:* Face to face guided self-help, computerised guided self-help, or pure self-help.

Results

Anorexia Nervosa Trials

Study characteristics. The characteristics of all included studies are presented in Table 2 (Appendix C) in the Supplementary Materials. Seven studies delivered CBT for AN. None of the trials included severely underweight individuals with AN (BMI ≤14.5). All seven studies compared individual, therapist-led CBT to an active comparison intervention. Two trials delivered CBT-E. The active comparisons included behavioural family therapy, cognitive remediation therapy, interpersonal psychotherapy, specialist supportive clinical management, dietary counselling, focal psychodynamic therapy, and Maudsley model of therapy.

The quality of included studies varied. Five trials reported an adequate sequence generation, three trial reported adequate allocation concealment, four trials reported blinding of outcome assessment or used self-report questionnaires, and six trials conducted ITT analyses. Three trials met all four quality criteria, one trial met three criteria, one trial met two criteria, one trial met one of the criteria, and one trial met none of the four criteria. Please see Table 2 (Appendix C) in the supplementary materials for domain ratings for each trial.

Therapist-led CBT for AN.

CBT vs. Inactive comparisons. No studies contributed to this comparison.

CBT vs. Active comparisons. There was no statistically significant post-treatment difference in cognitive symptoms between CBT and active comparison treatments. Table 5.1 presents the results from this meta-analysis. There were also no significant differences at short and long-term follow-up. Table 5.2 presents the results from these analyses at follow-up. No studies examined binge/purge frequency or remission rates.

CBT vs. pharmacotherapy comparisons. No studies contributed to this comparison.

Table 5.1
Primary Meta-Analyses Comparing CBT to Inactive, Active, and Pharmacological Comparisons at Post-Treatment

		Remission			Binge/purge frequencies			Cognitive symptoms		
Comparison	Sample	N_{comp}	OR (95% CI)	I^2	N_{comp}	g (95% CI)	I^2	N_{comp}	g (95% CI)	I^2
Therapist-led CBT vs inactive										
	BN	4	**8.89 (2.25, 35.12)**	71%	8	**0.89 (0.56, 1.22)**	66%	8	**0.34 [0.11, 0.56)**	42%
	BED	7	**6.01 (3.13, 11.77)**	0%	11	**1.13 (0.71, 1.55)**	74%	6	0.24 [-0.28, 0.76)	84%
	AN	-	-	-	-	-	-	-	-	-
Therapist-led CBT vs active										
	BN	15	**1.49 (1.00, 2.26)**	53%	25	**0.21 (0.05, 0.36)**	68%	18	**0.20 (0.01, 0.39)**	74%
	BED	5	0.97 (0.61, 1.53)	26%	9	**0.18 (0.01, 0.35)**	41%	8	**0.17 [0.01, 0.33)**	0%
	AN	-	-	-	-	-	-	10	0.13 (-0.05, 0.32)	46%
Therapist-led CBT vs pharmacotherapy										
	BN	3`	1.99 (0.63, 6.27)	55%	4	0.27 (-0.02, 0.56)	52%	4	0.18 (-0.05, 0.12)	0%
	BED	-	-	-	2	1.61 (-1.07, 4.35)	97%	2	**0.73 (0.37, 1.08)**	0%
	AN	-	-	-	-	-	-	-	-	-
Self-help CBT vs inactive										
	BN	4	**3.44 (2.05, 5.78)**	0%	5	0.16 (-0.11, 0.44)	75%	8	**0.47 (0.12, 0.82)**	92%
	BED	16	**4.82 (3.20, 7.27)**	19%	15	**0.57 (0.32, 0.82)**	64%	13	**0.57 (0.31, 0.82)**	84%
	AN	-	-	-	-	-	-	-	-	-
Self-help CBT vs active										
	BN	-	-	-	-	-	-	-	-	-
	BED	4	1.45 (0.71, 2.97)	43	4	0.21 (-0.04, 0.45)	0%	5	0.13 (-0.16, 0.41)	57%
	AN	-	-	-	-	-	-	-	-	-

Note: There were insufficient studies to perform meta-analyses comparing self-help to pharmacotherapy for BN and BED. BN= bulimia nervosa; BED= binge eating disorder; AN= anorexia nervosa. Bolded indicates statistical significance

Table 5.2
Short and Long-Term Outcomes of Therapist-Led CBT for the Three Main Comparisons

			Remission rates			Binge/purge frequency			Cognitive symptoms		
Sample	Comparison	Follow-up point	N_{comp}	OR (95% CI)	*p*	N_{comp}	g (95% CI)	*p*	N_{comp}	g (95% CI)	*p*
AN											
	CBT vs active										
		Short-term	-	-	-	-	-	-	5	-0.02 (-0.23, 0.18)	.822
		Long-term	-	-	-	-	-	-	6	0.03 (-0.20, 0.26)	.802
BN											
	CBT vs inactive										
		Short-term	1	2.33 (0.85, 6.36)	.098	**1**	**0.81 (0.42, 1.19)**	**<.001**	1	0.17 (-0.37, 0.72)	.541
		Long-term	-	-	-	-	-	-	-	-	
	CBT vs active										
		Short-term	**7**	**2.28 (1.25, 4.17)**	**.007**	10	0.22 (-0.01, 0.46)	.060	8	0.03 (-0.17, 0.22)	.779
		Long-term	6	1.10 (0.65, 1.88)	.700	**10**	**0.31 (0.10, 0.52)**	**.003**	9	0.11 (-0.04, 0.26)	.134
	CBT vs pharmacotherapy										
		Short-term	-	-	-	-	-	-	-	-	-
		Long-term	1	4.66 (0.40, 53.95)	.217	1	0.38 (-0.30, 1.07)	.279	1	0.32 (-0.36, 1.01)	.354
BED											
	CBT vs inactive										
		Short-term	1	2.80 (0.12, 63.58)	.517	**1**	**4.11 (2.89, 5.33)**	**<.001**	-	-	
		Long-term	-	-	-	-	-	-	-	-	
	CBT vs active										
		Short-term	2	1.54 (0.88, 2.68)	.125	5	0.12 (-0.17, 0.43)	.415	**6**	**0.18 (0.04, 0.30)**	**.011**
		Long-term	4	1.08 (0.72, 1.62)	.686	4	0.14 (-0.13, 0.37)	.363	4	0.01 (-0.17, 0.18)	.951
	CBT vs pharmacotherapy										
		Short-term	1	8.66 (0.98, 76.11)	.051	1	0.11 (-0.41, 0.63)	.689	**1**	**0.92 (0.25, 1.59)**	**.007**
		Long-term	**1**	**14.44 (1.69, 122.97)**	**.015**	2	1.15 (-0.88, 3.20)	.266	**3**	**0.99 (0.51, 1.48)**	**<.001**

Note: OR= odds ratio; CI = confidence interval; N_{comp}= number of comparisons; CBT-E = enhanced cognitive-behavioural therapy. Short-term = < 12 months; Long-term = ≥ 12 months; - indicates that there were not enough studies to conduct a meta-analysis. Analyses for AN could only be conducted when CBT was compared with active controls. bolded indicates statistical significance

Bulimia Nervosa Trials

Study Characteristics. There were 37 studies that delivered CBT to individuals with BN. Twenty-eight studies delivered therapist-led CBT—six in group format and 22 in individual format. Face to face (*k*=6) and computerised (*k*=2) guided self-help, and pure self-help (*k*=1) was delivered less often. Fourteen studies compared CBT to an inactive comparison, 26 studies compared CBT to an active comparison (see Supplementary Table 2, Appendix C), and five compared CBT to a pharmacological (all antidepressants) comparison.

Twenty-two studies reported an adequate sequence generation, only 11 trials reported adequate allocation concealment, 32 trials reported blinding of outcome assessment or used self-report questionnaires, and 20 trials conducted ITT analyses. Only eight trials met all four quality criteria, nine trials met three criteria, seven trials met two criteria, 12 trials met one of the criteria, and one trial met none of the quality criteria. Please see Table 2 (Appendix C) in the supplementary materials for domain ratings for each trial.

Therapist-led CBT (post-treatment). Table 5.1 present the main results from each meta-analysis comparing therapist-led and self-help CBT for BN and BED to inactive, active, and pharmacological comparisons. The number of comparisons, the pooled effect size and 95% confidence interval, and the degree of heterogeneity is presented in this table. Statistically significant effect sizes are highlighted in bold.

As can be seen in Table 5.1, therapist-led CBT for individuals with BN was significantly more efficacious than inactive and active comparisons at post-treatment on all three outcomes. Effect sizes and the degree of heterogeneity ranged from small to large. There was no evidence suggesting that therapist-led CBT for BN was significantly more efficacious than antidepressants at post-treatment.

Subgroup analyses. A series of subgroup analyses were performed for the comparison between CBT and *inactive* conditions at post-treatment. The results of these

subgroup analyses can be seen in Table 5.3. CBT format (group or individual) and CBT type (CBT-BN variant or "other") did not moderate any effects at post-treatment (see Table 5.3).

Subgroup analyses were also performed for the comparison between CBT and *active* conditions at post-treatment (Table 5.3). Only two moderation effects were observed: Studies that delivered manualized CBT-BN or CBT-E produced significantly larger effect sizes on cognitive symptoms than studies that delivered either a variant of CBT-BN or an alternative protocol. Additionally, studies that delivered CBT for BN in *adults* produced significantly higher remission rates than studies that delivered CBT for BN in *adolescents*.

Consistent trends *within* study subgroups were also found for the CBT versus active comparisons. In particular, therapist-led CBT was significantly superior to active comparisons on all outcomes *only* when full CBT-BN or CBT-E was delivered—the effect sizes for studies that delivered adapted versions of CBT-BN or alternative protocols were not statistically significant across each outcome. The same trends were observed for sample age; CBT was significantly superior to active comparisons *only* in adults. The effect sizes for adolescents were non-significant, though few studies contributed to this subgroup (Table 5.3).

Follow-up findings. Table 5.2 presents the results from the meta-analyses for the three main comparisons at short and long-term follow-up for individuals with BN. As shown, only one study contributed to the analyses comparing CBT to inactive and pharmacotherapy, and no differences were reported. However, there was evidence that CBT for BN was significantly more efficacious than *active* comparisons on behavioural, but not cognitive, symptoms at follow-up periods.

CBT self-help for BN. Table 5.1 presents the meta-analyses comparing CBT self-help for BN to inactive and active comparisons. With moderate effect sizes, self-help CBT for BN was significantly more efficacious than inactive comparisons on remission rates and

cognitive symptoms. It was not possible to perform meta-analyses comparing self-help CBT for BN to an active or pharmacological comparison.

Table 5.3
Subgroup Analyses Across Post-Treatment Outcomes during Therapist-Led CBT for Bulimia Nervosa

		Remission				Binge/purge frequency				Cognitive symptoms			
	Subgroup	N_{comp}	OR (95% CI)	*p*	Qbp	N_{comp}	g (95% CI)	*p*	Qbp	N_{comp}	g (95% CI)	*p*	Qbp
CBT v inactive													
	Format												
	Individual	4	8.89 (2.52, 35.12)	.002		4	0.98 (0.52, 1.43)	<.001		4	0.29 (-0.07, 0.69)	.121	
	Group	-	-	-		4	0.79 (0.29, 1.29)	.002		2	0.46 (-0.11, 1.04)	.115	
					-				.594				.634
	CBT type												
	CBT-BN/E	-	-	-		-	-			-	-	-	
	Adapted CBT-BN	3	15.00 (1.97, 113.90)	.009		5	1.10 (0.67, 1.53)	<.001		4	0.36 (-0.05, 0.78)	.088	
	Other	1	3.35 (0.11, 100.50)	.486		3	0.60 (0.09, 1.14)	.020		3	0.32 (-0.21, 0.86)	.241	
					.458				.114				.900
CBT v active													
	Format												
	Individual	14	1.48 (0.98, 2.23)	.062		20	0.19 (0.02, 0.37)	.021		16	0.21 (0.01, 0.42)	.041	
	Group	1	2.10 (0.15, 28.31)	.574		5	0.24 (-0.13, 0.61)	.200		2	0.11 (-0.51, 0.72)	.745	
					.793				.824				.737
	CBT type												
	CBT-BN/E	7	2.08 (1.23, 3.53)	.006		7	0.42 (0.16, 0.67)	.001		7	0.53 (0.31, 0.74)	<.001	
	Adapted CBT-BN	7	1.01 (0.58, 1.76)	.950		11	0.04 (-0.18, 0.27)	.710		10	-0.03, (-0.23, 0.16)	.731	
	Other	1	1.58 (0.35, 7.19)	.548		7	0.21 (-0.09, 0.51)	.174		1	0.08 (-0.56, 0.74)	.793	
					.183				.099				**.001**
	Specific CBT type												
	Full CBT-BN	4	2.37 (0.98, 5.74)	.054		4	0.32 (-0.09, 0.73)	.127		3	0.53 (0.16, 0.89)	.004	
	Full CBT-E	3	1.78 (0.71, 4.45)	.215		3	0.52 (0.08, 0.96)	.019		4	0.52 (0.21, 0.82)	.001	
					.657				.506				.979
	Age												
	Adults	14	1.70 (1.21, 2.38)	.002		25	0.23 (0.06. 0.37)	.005		16	0.27 (0.08, 0.45)	.004	
	Adolescents	1	0.36 (0.12, 1.06)	.065		2	-0.01 (-0.52, 0.49)	.947		2	-0.25, (-0.76, 0.25)	.322	
					.007				.376				.055

Note: OR= odds ratio; CI = confidence interval; N_{comp}= number of comparisons; Qbp= p value for testing whether subgroups differ significantly from each other; CBT-E = enhanced cognitive-behavioural therapy.

Binge Eating Disorder Trials

Study characteristics. There were 35 studies that delivered CBT to individuals with BED. Twenty-one studies delivered therapist-led CBT: 16 in group format and five in individual format. Face to face (*k*=11) and computerised (*k*=4) guided self-help, and pure self-help (*k*=3) were delivered less often[3]. Twenty-two studies compared CBT to an inactive comparison, 10 studies compared CBT to an active comparison, and three compared CBT to a pharmacological comparison (see Supplementary Table 2, Appendix C).

Twenty-three studies reported an adequate sequence generation, only five trials reported adequate allocation concealment, 30 trials reported blinding of outcome assessment or used self-report questionnaires, and 26 trials conducted ITT analyses. Four trials met all four quality criteria, 12 trials met two criteria, and seven trials met one of the criteria. Please see Table 2 in the supplementary materials (Appendix C) for domain ratings for each trial.

Therapist-led CBT (post-treatment). As can be seen in Table 5.1, therapist-led CBT for BED was significantly more efficacious than *inactive* comparisons on remission rates and binge/purge frequencies (large effect sizes), significantly more efficacious than *active* comparisons on binge/purge frequencies and cognitive symptoms (small effect sizes), and significantly more efficacious than *pharmacotherapy* on cognitive symptoms (large effect size).

Subgroup analyses. Subgroup analyses were performed for the comparisons of CBT to inactive and active conditions (Table 5.4). Only one moderation effect occurred; studies that delivered an alternative CBT protocol produced a significantly larger effect size on cognitive symptoms than studies that delivered an abbreviated version of CBT-BN.

[3] Note that many trials included multiple conditions of different CBT modalities.

Follow-up findings. Table 5.3 presents the results of CBT for BED at follow-up. Only one study contributed to the analyses comparing CBT to inactive conditions, and this study showed a benefit of CBT over pharmacotherapy on binge eating frequency. While therapist-led CBT for BED was equally efficacious to active comparisons at follow-up, therapist-led CBT showed a clear benefit over pharmacotherapy at long-term follow-up.

Self-help CBT for BED (post-treatment). Table 5.1 also presents the meta-analyses comparing CBT self-help for BED to inactive and active comparisons. As shown, self-help CBT for BED was significantly more efficacious than *inactive* comparisons on all outcomes (with moderate effect sizes), but was not more efficacious than *active* comparisons. Analyses comparing self-help to pharmacotherapy were not performed, as only one trial compared these treatments.

Follow-up. Analyses comparing self-help CBT for BED to *inactive* comparisons were performed at follow-up. Follow-up analyses comparing self-help CBT for BED to *active* and *pharmacological* comparisons were not performed, as too few studies provided these data. Given the limited number of studies providing follow-up data, we analysed the last reported follow-up only.

CBT self-help for BED was significantly more efficacious than *inactive* controls at follow-up on remission rates (N_{comp} = 5, OR= 2.81, 95% CI [1.76, 4.49], I^2= 0%). Of these, the mean effect size for the subgroup that delivered *face to face* guided self-help (OR = 2.87, 95% CI [1.68, 4.99]) was statistically significant, while the mean effect size for the studies that delivered guided self-help over the *computer* was non-significant (OR= 2.63, 95% CI [0.99, 6.90]). CBT self-help for BED was also significantly more efficacious than *inactive* controls at follow-up on cognitive symptoms (N_{comp} = 4, g= 0.39, 95% CI [0.14, 0.63], I^2= 66%). Of these studies, three delivered *face to face* guided self-help, and the mean effect size

for this subgroup was statistically significant (g= 0.51, 95% CI [0.36, 0.63]). Follow-up analyses for binge frequencies were not performed given the limited available data.

Direct Comparisons

A series of analyses comparing any type of CBT to specific alternative psychological treatments were performed at post-treatment and follow-up. Given the limited number of studies directly comparing CBT with these specific psychological treatments, we took a transdiagnostic perspective and included all diagnoses into these analyses. The number of comparisons and pooled effect sizes for these comparisons can be seen in Table 5.5.

CBT vs. interpersonal psychotherapy. Seven studies compared CBT to interpersonal psychotherapy (Agras et al., 2000b; Fairburn et al., 2015; Fairburn et al., 1991; McIntosh et al., 2005; Wilfley et al., 1993; Wilfley et al., 2002; Wilson, Wilfley, Agras, & Bryson, 2010). Six delivered therapist-led CBT and one delivered CBT guided self-help. Three studies sampled BN, three sampled BED, and one study sampled AN. Findings show CBT had a significantly larger effect at post-treatment on binge/purge frequencies and cognitive symptoms than interpersonal psychotherapy. Given that there was a trend favouring CBT on remission rates (p=.130), we performed an analysis in which we removed the CBT self-help trial. Therapist-led CBT was significantly more efficacious than interpersonal psychotherapy on remission rates (OR= 2.05, 95% CI [1.07, 3.93]). At follow-up, CBT was only more efficacious than IPT on cognitive symptoms.

CBT vs. behaviour therapy. Eight studies compared CBT to behaviour therapy (Cooper & Steere, 1995; Fairburn et al., 1991; Freeman, Barry, Dunkeld-Turnbull, & Henderson, 1988; Griffiths, Hadzi-Pavlovic, & Channon-Little, 1994; Grilo & Masheb, 2005; Nauta, Hospers, Kok, & Jansen, 2000; Thackwray, Smith, Bodfish, & Meyers, 1993; Wolf & Crowther, 1992). All eight studies delivered therapist-led CBT for BN. At post-treatment, there was no significant difference between CBT and behaviour therapy on any outcome. At

follow-up, however, CBT produced significantly greater rates of remission than behaviour therapy

CBT vs behavioural weight loss. Five studies compared CBT to behavioural weight loss (Agras et al., 1994; Grilo & Masheb, 2005; Grilo, Masheb, Wilson, Gueorguieva, & White, 2011; Munsch et al., 2007; Wilson et al., 2010). All five studies sampled individuals with BED with comorbid overweight obesity—two delivered therapist-led CBT and three delivered guided self-help. CBT was significantly more efficacious than behavioural weight loss at post-treatment and follow-up on binge/purge frequencies. No other differences were observed. We also conducted a meta-analysis examining group BMI differences. Although BMI was lower in BWL, the differences at post-treatment (g= -0.26, 95% CI [-0.56, 0.05]) and follow-up (g= -0.13, 95% CI [-0.35, 0.09]) were not significant.

CBT vs non-specific supportive therapy. Six studies compared CBT to a non-specific supportive therapy (Carter et al., 2003; Freeman et al., 1988; Garner et al., 1993; Kenardy, Mensch, Bowen, Green, & Walton, 2002; Thackwray et al., 1993; Walsh et al., 1997). Five sampled BN and one sampled BED. Five delivered therapist-led CBT and one delivered CBT guided self-help. No statistically significant differences were observed between these two treatments at post-treatment.

Table 5.4
Subgroup Analyses Across Post-Treatment Outcomes During Therapist-Led CBT for Binge Eating Disorder

		Remission			Binge/purge frequency			Cognitive symptoms		
	Subgroup	N_{comp}	OR (95% CI)	Qbp	N_{comp}	g (95% CI)	Qbp	N_{comp}	g (95% CI)	Qbp
CBT v inactive										
	Format									
	Individual	-	-		1	2.04 (0.63, 1.51)		1	-0.43 (-1.76, 0.90)	
	Group	7	6.04 (3.17, 11.72)		10	1.07 (0.63, 3.44)		5	0.36 (-0.20, 0.92)	
							.197			.282
	CBT type									
	CBT-BN/E	-	-		-	-		-	-	
	Adapted CBT-BN	-	-		2	0.75 (-0.42, 1.92)				
	Other	7	6.04 (3.17, 11.72)		9	1.23 (0.74, 1.72)		6	0.24 (-0.28, 0.76)	
				-			.462			-
CBT v active										
	Format									
	Individual	1	0.99 (0.33, 3.02)		2	0.17 (-0.13, 0.46)		2	0.26 (-0.20, 0.73)	
	Group	4	0.93 (0.50, 1.74)		6	0.17 (-0.12, 0.46)		7	0.17 (-0.03, 0.37)	
				.919			.974			.706
	CBT type									
	CBT-BN/E	1	1.31 (0.39, 4.38)		1	0.29 (-0.23, 0.81)		1	0.12 (-0.30, 0.53)	
	Adapted CBT-BN	2	0.72 (0.31, 1.67)		2	0.11 (-0.16, 0.41)		2	-0.12 (-0.36, 0.13)	
	Other	2	1.08 (0.42, 2.78)		5	0.19 (-0.01, 0.41)		6	0.27 (0.13, 0.44)	
				.683			.788			**.030**

Note: OR= odds ratio; CI = confidence interval; N_{comp}= number of comparisons; Qbp= p value for testing whether subgroups differ significantly from each other; CBT-E = e
cognitive-behavioural therapy.

Table 5.5
Direct Comparisons Between CBT and Alternative Psychological Treatments at Post-Treatment and Follow-Up on Primary Outcomes

		Remission			Binge/purge frequencies			Cognitive symptoms		
Comparison	Time point	N_{comp}	OR (95% CI)	*p*	N_{comp}	g (95% CI)	*p*	N_{comp}	g (95% CI)	*p*
CBT vs. interpersonal psychotherapy										
	Post	6	1.66 (0.86, 3.23)	.130	6	**0.24 (0.01, 0.47)**	**.044**	7	**0.32 (0.14, 0.50)**	**<.001**
	Follow-up	4	1.14 (0.75, 1.71)	.530	5	0.06 (-0.07, 0.21)	.348	6	**0.16 (0.04, 0.28)**	**.010**
CBT vs. behaviour therapy										
	Post	5	1.54 (0.82, 2.88)	.173	8	0.17 (-0.17, 0.52)	.323	7	0.13 (-0.32, 0.58)	.569
	Follow-up	3	**3.34 (1.38, 8.07)**	**.007**	4	0.51 (-0.06, 1.06)	.080	4	0.14 (-0.18, 0.74)	.398
CBT vs. behavioural weight loss										
	Post	4	1.23 (0.57, 2.64)	.605	5	**0.30 (0.09, 0.51)**	**.005**	5	0.19 (-0.05, 0.44)	.117
	Follow-up	2	1.45 (0.79, 2.68)	.226	3	**0.24 (0.01, 0.46)**	**.036**	3	0.08 (-0.26, 0.44)	.618
CBT vs. non-specific supportive therapy										
	Post	2	2.29 (0.62, 8.44)	.211	6	0.29 (-0.01, 0.61)	.056	4	0.21 (-0.36, 0.79)	.472
	Follow-up	-	-	-	-	-	-	-	-	-

Discussion

Summary of Findings

The efficacy of CBT for eating disorders was supported in the current meta-analysis. Therapist-led CBT for BN and BED was consistently more efficacious than inactive control conditions at reducing behavioural and cognitive symptoms. Critically, improvements in core behavioural symptoms were sustained at follow-up periods, suggesting that CBT has an enduring effect beyond the end of treatment. In addition, CBT delivered in a guided self-help format was also consistently more efficacious than inactive comparisons at reducing behavioural and cognitive symptoms in BN and BED. The fact that CBT guided self-help was shown to be an efficacious treatment for this population supports recommendations that CBT guided self-help be offered as a first-step for treating BN and BED (Hay et al., 2014; National Institute of Clinical Excellence, 2017)

We also compared therapist-led CBT to active control conditions (any other psychotherapy approach). Therapist-led CBT was shown to be more efficacious than active comparisons at reducing behavioural and cognitive symptoms in individuals with BN and BED. Critically, however, few studies contributed to the analyses at follow-up, which highlights the need for future RCTs to assess the long-term impact of CBT. Moreover, we found no evidence to suggest that CBT was significantly more efficacious than active psychological comparisons in individuals with AN. This was the first study to meta-analyse *all* available RCTs that have delivered CBT to individuals with AN, and no evidence was found to support the superiority of any psychotherapy over others (Byrne et al., 2017).

Which Version of CBT?

A noteworthy finding was that when therapist-led CBT for BN was compared with active controls, statistically significant effect sizes were *only* observed for studies that delivered manualized CBT-BN or CBT-E as described. These findings indicate that the

superiority of CBT over other psychological treatments is only achieved when the techniques, session structure, and theoretical model outlined in the manual developed by Fairburn and colleagues are implemented. There are several possible explanations for this finding. First, this manualized protocol is based on an extensively validated cognitive-behavioural model that outlines the eating disorder maintaining mechanisms. These manualized treatments list specific strategies designed to target these mechanisms, and the success of treatment is hypothesised to depend on how well these mechanisms are targeted. Indeed, targeting these mechanisms is crucial for success; greater reductions in these maintaining mechanisms are linked to better outcomes (Linardon, Brennan, & de la Piedad Garcia, 2016a; Linardon et al., 2016b), and studies that have removed key CBT-BN components designed to eliminate these mechanisms have reported poor outcomes and high rates of relapse (Cooper & Steere, 1995; Fairburn et al., 1991). Another possible reason for the superiority of these manualized protocols may be attributed to higher therapist quality. Trials that delivered CBT-BN or CBT-E were much more likely to audit treatment sessions, assess treatment fidelity and adherence, and report the use of frequent supervision. This idea is consistent with research in depression and anxiety treatment, where a robust relationship between better treatment outcomes and higher quality training/supervision and therapist adherence has been reported (Ginzburg et al., 2012). Although it is assumed that CBT-E is more effective than CBT-BN, we found no differences in effect sizes between these two protocols. To make stronger conclusions about the relative effects of these CBT protocols, additional RCTs that directly compare CBT-BN and CBT-E are required.

CBT versus Dismantled Behavioural Treatments

Our meta-analysis found CBT for BN to be equally efficacious to dismantled behavioural treatments post-treatment, although there was preliminary evidence suggesting that CBT was superior at follow-up. Previous researchers have questioned the benefit of

adding complex cognitive interventions to simpler behavioural treatments to achieve therapeutic change for psychological disorders (Dobson & Khatri, 2000). However, since cognitive mechanisms are considered central to the maintenance of eating disorders, advocates of CBT argue that specific treatment strategies that are designed to target these cognitive mechanisms are critical for therapeutic change (Fairburn, 2008). Despite this argument, the available data does suggest that similar behavioural treatments can lead to improvements similar to CBT. Clearly more trials comparing these treatments are needed to clarify this effect. If behaviour therapy is indeed as effective as complex CBT protocols, dissemination of evidence-based treatments could be greatly improved, as behaviour therapy is proposed to be simpler to learn and requires less skilled, trained, and supervised clinicians.

The Role of Pharmacotherapy

CBT for BN and BED was also compared to pharmacological interventions (antidepressants in all but one case). CBT and pharmacotherapy was equally efficacious at post-treatment in BN and BED. This finding echoes previous research demonstrating that pharmacological therapy, particularly antidepressant medication, has a strong short-term anti-bulimic effect (Brownley et al., 2016). At follow-up, however, CBT was more efficacious than pharmacotherapy only for individuals with BED. All included pharmacotherapy studies discontinued medication use immediately after the treatment phase of the study. This indicates that, unlike the durable effects of CBT, where improvements seem to be sustained after treatment ends, BED symptoms do not seem to be sustained following the discontinuation of medication. This result is consistent with a recent meta-analysis on treatment for adult depression (Cuijpers et al., 2013), which found that while CBT was superior to pharmacotherapy at 12 month follow-up in studies that discontinued medication at post-treatment, CBT and pharmacotherapy were equally efficacious in studies that continued

medication use throughout follow-up. Overall, however, the data suggest that the use of pharmacotherapy alone is not recommended in terms of producing long-term change.

CBT versus Other Psychological Interventions

A novel aspect of this meta-analysis was that we compared CBT directly to other specific psychological interventions. CBT was compared with interpersonal psychotherapy, and although CBT was superior to interpersonal psychotherapy on behavioural symptoms at post-treatment, this difference was not evident at follow-up. Interpersonal psychotherapy takes longer to achieve its effects, and this is thought to be because it targets eating disorder symptoms indirectly. However, we found that this "catch up" effect of interpersonal psychotherapy only applies to behavioural symptoms, as CBT was still significantly superior to interpersonal psychotherapy at follow-up on cognitive symptoms. This finding reinforces the revised NICE guidelines, which recommends CBT over interpersonal psychotherapy as the treatment of choice for eating disorders. Moreover, the fact that CBT outperformed interpersonal psychotherapy demonstrates that CBT has specific mechanisms of change, thereby providing evidence against the common factors model of therapeutic change (Messer & Wampold, 2002).

CBT for BED with overweight/obesity was also compared with behavioural weight loss. CBT was generally superior to behavioural weight loss in the short and long-term at reducing binge eating frequencies. The fact that CBT outperformed behavioural weight loss on binge eating frequency is not unexpected, as behavioural weight loss, unlike CBT, aims to induce one of the core mechanisms hypothesised to maintain binge eating behaviour—dietary restraint. No differences were observed between CBT for BED and behavioural weight loss on cognitive outcomes, suggesting that behavioural weight loss might also have a strong effect on reducing core cognitive symptoms in BED. Unexpectedly, BMI did not differ between the two interventions. Thus, given this lack of observed difference, and given that

CBT has a more powerful effect on reducing binge eating than behavioural weight loss, the data suggest that CBT should be prioritized and selected over behavioural weight loss as a treatment approach for overweight individuals with BED.

Finally, CBT was also compared to non-specific supportive therapies. Broadly, non-specific supportive therapy was typically an unstructured therapy without specific psychological techniques other than those that are common to all approaches (e.g., providing empathy, discussion between client and therapist on experiences and emotions). We found no evidence that CBT was more efficacious than non-specific therapies in individuals with eating disorders. This is similar to what was reported in a recent meta-analysis comparing non-specific supportive therapy to CBT for depression (Cuijpers et al., 2012). However, studies that contributed to the CBT versus non-specific psychotherapy analyses varied. In particular, for this comparison, some studies delivered less intense guided self-help CBT, others delivered group-based CBT, and others delivered individual therapist-led CBT. Critically, only one study in these analyses delivered therapist-led manualized CBT-BN. In this study, Garner et al (1993) found a clear advantage of CBT-BN over supportive therapy on purge frequencies, dietary restraint, and extreme concerns about shape, suggesting that CBT might be more effective than non-specific therapies *only* when this particular therapist-led CBT protocol is delivered. In sum, while the data may be more in favour of treatment specificity for eating disorders, at present the common factors model cannot yet be conclusively ruled out.

Directions for Future Research

To advance the field on psychological treatments for eating disorders, we offer several recommendations for future research. For AN, more large-scale RCTs evaluating specialist psychological treatments (particularly CBT-E) are needed. Only seven RCTs of CBT for AN were identified, and only one had a sample size large enough to detect

statistically significant differences between treatment conditions, assuming a small effect size (Zipfel et al., 2014). The argument that there is no particular psychotherapy for AN that is superior to others may be due to the relatively weak statistical power of available studies. Of course, executing a large sample RCT in this population is challenging, yet large trials of AN are nevertheless underway (Watson & Bulik, 2013).

For BN, a greater understanding of the long-term efficacy of CBT is required. For the few trials that have conducted follow-up assessments, length of assessment has typically been 12 months post-treatment. Relapse in BN, however, is common after this period (Olmsted, Kaplan, & Rockert, 2005). Consequently, the long-term efficacy and durability of CBT for BN is largely unknown. Further, comparing CBT to *continued* antidepressant use at long-term follow-up on symptoms of eating disorders and several indices of health (e.g., quality of life) (Linardon & Brennan, 2017) is an important future direction. Such findings could have significant implications for improving the dissemination of cost-effective BN treatments. Finally, to confirm the specificity of psychological treatments for BN, additional trials comparing CBT to a range of other psychological treatments, including interpersonal psychotherapy, non-specific supportive therapy, and the first and third-wave behaviour therapies, are required.

For BED, few trials have directly compared various intensities of CBT (Peterson, Mitchell, Crow, Crosby, & Wonderlich, 2009; Peterson et al., 1998). For instance, while therapist-led CBT for BED was shown to be superior to guided self-help CBT at post-treatment, no differences between modalities were observed at follow-up (Peterson et al., 2009). Comparing distinct cognitive-behavioural treatment modalities should be examined in more trials, particularly since a stepped-care approach is recommended for BED. The stepped-care approach assumes that therapist-led CBT is more effective than guided self-help CBT and should be given priority for those who respond slowly to self-help treatment (NICE,

2017). However, there is insufficient data to definitively conclude that therapist-led CBT is more potent than guided self-help CBT for BED, particularly at longer term follow-up. Clarifying this with larger trials is important. Additionally, demonstrating treatment specificity for BED is also important, so comparing CBT for BED to a range of psychological interventions (e.g., interpersonal psychotherapy, behavioural weight loss) is needed.

Finally, developing empirically supported treatments such as CBT is not the only goal of eating disorder research. Once the efficacy of a treatment is established, the mechanisms through which this treatment exerts its effects, and factors that alter the efficiency of the treatment within certain subgroups should be elucidated. Analysing mediators, moderators, and predictors of response to CBT is one avenue toward improving the effectiveness of CBT for eating disorders, and this should be a research priority (Linardon et al., 2016a; Linardon et al., 2016b).

Limitations and Conclusions

There are limitations to the current meta-analysis. First, the number of trials was relatively small for many of the comparisons and subgroup analyses. Finding no differences between comparisons when the number of trials is small is not conclusive evidence that there is no meaningful difference present, as the lack of an observed difference may be due to insufficient power. Second, the possibility of publication bias is another limitation. Although we tried to limit the impact of publication bias by searching for and including as many unpublished trials as possible, the possibility that some unpublished trials were missed (and hence inflating effect size estimates) cannot be ruled out. We did not statistically test for publication bias because when the number of studies in an analysis is small, using such statistical methods (e.g., trim and fill method) is not recommended (Hunter et al., 2014). Finally, the quality of included studies was far from optimal; only 15 of 79 trials (19%) met

all four criteria for low risk of bias. Therefore, caution should be exercised in interpreting the findings from the current review.

To conclude, the efficacy of therapist-led and guided self-help CBT for BN and BED was supported in the current study. Therapist-led CBT is most efficacious when a manualized version of CBT-BN or its enhanced version is delivered. CBT was no more efficacious than alternative psychotherapies for AN. CBT for eating disorders was equally efficacious to other specific psychological interventions, most clearly behaviour therapy and non-specific supportive therapy. However, given that few studies contributed to these analyses, and that CBT was shown to clearly outperform an aggregate of active psychological treatments, in addition to interpersonal psychotherapy specifically, the data current favour the specificity of psychological treatment for eating disorders. Given that the quality of included trials was far from optimal, there is more work to be done to ensure future RCTs meet higher standards and can thus offer more useful and robust conclusions.

Chapter 6: Predictors, Moderators, and Mediators of Treatment Outcome Following Manualized Cognitive-Behavioral Therapy for Eating Disorders: A Systematic Review (Article 2).

This chapter presents an article accepted for publication in the *European Eating Disorders Review* (Linardon et al., 2016b). The first study reported that CBT was an efficacious treatment for individuals with eating disorders. However, as reviewed in Chapter 3, many individuals who receive CBT fail to make a full and lasting recovery. Understand how (mediators) and for whom (predictors and moderators) CBT works is one avenue toward improving the effectiveness of CBT. This study aimed to systematically review the literature for reliable mediators, moderators, and predictors of outcome following CBT.

REVIEW

Predictors, Moderators, and Mediators of Treatment Outcome Following Manualised Cognitive-Behavioural Therapy for Eating Disorders: A Systematic Review

Jake Linardon*, Xochitl de la Piedad Garcia & Leah Brennan

School of Psychology, Australian Catholic University, Melbourne, Victoria, Australia

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 was 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 (i) interpret the clinical and statistical significance of findings; (ii) use a consistent definition of outcome so that studies can be directly compared; and (iii) report null and statistically significant findings.

Received 10 August 2016; Revised 11 October 2016; Accepted 15 October 2016

Keywords

eating disorder; cognitive behaviour therapy; mediator; moderator; predictor

***Correspondence**

Jake Linardon, Faculty of Health Sciences, Australian Catholic University, 115 Victoria Parade/Locked Bag 4115, Melbourne, Victoria 3065, Australia. Tel: +613 9953 3662; Fax: +613 9953 3205.

Email: Jake.Linardon@acu.edu.au

Published online in Wiley Online Library (wileyonlinelibrary.com) DOI: 10.1002/erv.2492

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 (eight trials) and alternative psychological treatments (seven 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 (i) direct manipulation of a mechanism and observing its effect; (ii) ensuring change is explained by the hypothesised mechanism only; (iii) demonstrating a dose–response relationships; and (iv) 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-behavioural 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: (i) peer-reviewed papers published in English before June 2016; (ii) include a sample aged over 16 years and meeting a diagnosis for threshold or subthreshold eating disorder; (iii) examine at least one mediator, moderator or predictor of treatment outcome; and (iv) 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: (i) for outcome measures, the authors controlled for baseline severity or reported change instead of raw scores; (ii) 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; (iii) authors provided a rationale for *all* variables tested; (iv) reliability ($\alpha \geq .70$) has been established for all variables examined; (v) Type I error was controlled if ≥ 20 tests were conducted; (vi) both null and statistically significant findings were reported; (vii) the

[1]Diagnostic status was included under secondary outcomes as only five of 56 studies reported these.

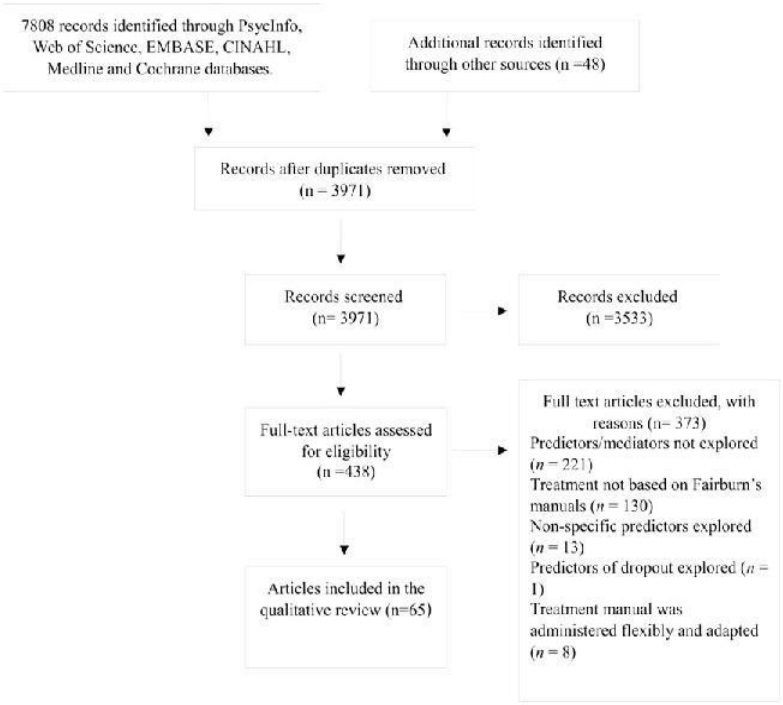


Figure 1. PRISMA flowchart of literature search

study was sufficiently powered[2]; and (viii) 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

A meta-analysis was not feasible for this review because (i) there was considerable heterogeneity across studies with respect to the variables tested and outcomes reported, and (ii) 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. Because the aim was to identify consistent predictors, findings are only presented for variables that were tested in two or more studies (Supporting Information 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 Supporting Information. A few studies also reported secondary outcomes (see above). A brief synthesis is presented in Supporting Information

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 1–3 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.

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.

[2]Underpowered studies were defined by Porter and Chambless (2015) as those that lacked 80% power to detect a medium effect of $f^2 = .15$ at $= .05$ in a linear regression with one predictor. Power analysis showed that 55 participants or more are needed to meet this requirement

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

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			
BN	Mediator variable	Studies	RCT	Other	CBT-BN	CBT-E	CBTgsh	CBT-BN	CBT-E	CBTgsh	Mean quality rating % (range)
	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%)

+ = 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			Comparison treatment		EOT behavioural outcomes			EOT cognitive outcomes			Mean quality
BED	Moderator	RCTs	Psychological	Pharmacology	CBT-BN	CBT-E	CBTgsh	CBT-BN	CBT-E	CBTgsh	rating % (range)
	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%)

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.

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

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			Mean quality
BN	Predictor variable	Studies	RCT	Other	CBT-BN	CBT-E	CBTgsh	CBT-BN	CBT-E	CBTgsh	rating % (range)
	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%)

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.

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 (2008a) found no evidence of moderation, Grilo et al. (2012a) 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 vs. 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 RCTs 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*, were 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,

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.. Because 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). Because 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 its subsequent change. 'Atheoretical' 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). An 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 (i) BMI $\geq$18.5; (ii) abstinence from bingeing, purging, and fasting for three months; and (iii) 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

clinicaland 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.

REFERENCES[4]

Accurso, E. C., Wonderlich, S. A., Crosby, R. D., Smith, T. L., Klein, M. H., Mitchell, J. E., Peterson, C. B. (2016). Predictors and moderators of treatment outcome in a randomized clinical trial for adults with symptoms of bulimia nervosa. *Journal of Consulting and Clinical Psychology, 84*, 178–184. doi: 10.1037/ccp0000073

Agency for Healthcare Research and Quality (2015). *Management and outcomes of binge eating disorder*. U.S: Agency for Healthcare Research and Quality.

Agras, W. S., Crow, S. J., Halmi, K. A., Mitchell, J., Wilson, G., & Kraemer, H. C. (2000). Outcome predictors for the cognitive behavior treatment of bulimia nervosa: Data from a multisite study. *The American Journal of Psychiatry, 157*, 1302–1308. 10.1176/appi.ajp.157.8.1302.

Ålgars, M., Ramberg, C., Moszny, J., Hagman, J., Rintala, H., & Santtila, P. (2015). Readiness and motivation for change among young women with broadly defined eating disorders. *Eating Disorders, 23*, 242–252. 10.1080/10640266.2014.1000100.

Allen, K. L., Fursland, A., Raykos, B. C., Steele, A., Watson, H. J., & Byrne, S. M. (2012). Motivation-focused treatment for eating disorders: A sequential trial of enhanced cognitive behaviour therapy with and without preceding motivation-focused therapy. *European Eating Disorders Review, 20*, 232–239. 10.1002/erv.1131.

Baell, W. K., & Wertheim, E. H. (1992). Predictors of outcome in the treatment of bulimia nervosa. *British Journal of Clinical Psychology, 31*, 330–332. 10.1111/j.2044-8260.1992.tb01000.x.

Bardone-Cone, A. M., Harney, M. B., Maldonado, C. R., Lawson, M. A., Robinson, D. P., Smith, R., et al. (2010). Defining recovery from an eating disorder: Conceptualization, validation, and examination of psychosocial functioning and psychiatric comorbidity. *Behaviour Research and Therapy, 48*, 194–202. 10.1016/j.brat.2009.11.001.

Berkman, N. D., Lohr, K. N., & Bulik, C. M. (2007). Outcomes of eating disorders: A systematic review of the literature. *International Journal of Eating Disorders, 40*, 293–309. 10.1002/eat.20369.

Butryn, M. L., Lowe, M. R., Safer, D. L., & Agras, W. S. (2006). Weight suppression is a robust predictor of outcome in the cognitive-behavioral treatment of bulimia nervosa. *Journal of Abnormal Psychology, 115*, 62–67. 10.1037/0021-843X.115.1.62.

Calugi, S., Dalle Grave, R., & Marchesini, G. (2013). Longstanding underweight eating disorder: Associated features and treatment outcome. *Psychotherapy Research, 23*(3), 315–323.

Calugi, S., Dalle Grave, R., Sartirana, M., & Fairburn, C. G. (2015). Time to restore body weight in adults and adolescents receiving cognitive behaviour therapy for anorexia nervosa. *Journal of Eating Disorders, 3*, 1.

Carter, J. C., & Fairburn, C. G. (1998). Cognitive–behavioral self-help for binge eating disorder: A controlled effectiveness study. *Journal of Consulting and Clinical Psychology, 66*, 616–623. 10.1037/0022-006X.66.4.616.

Castellini, G., Lo Sauro, C., Mannucci, E., Ravaldi, C., Rotella, C. M., Faravelli, C., et al. (2011). Diagnostic crossover and outcome predictors in eating disorders according to DSM-IV and DSM-V proposed criteria: A 6-year follow-up study. *Psychosomatic Medicine, 73*, 270–279. 10.1097/PSY.0b013e31820a1838.

Castellini, G., Mannucci, E., Lo Sauro, C., Benni, L., Lazzeretti, L., Ravaldi, C., et al. (2012). Different moderators of cognitive-behavioral therapy on subjective and objective binge eating in bulimia nervosa and binge eating disorder: A three-year follow-up study. *Psychotherapy & Psychosomatics, 81*, 11–20. 10.1159/000329358.

Constantino, M. J., Arnow, B. A., Blasey, C., & Agras, W. S. (2005). The association between patient characteristics and the therapeutic alliance in cognitive-behavioral and interpersonal therapy for bulimia nervosa. *Journal of Consulting and Clinical Psychology, 73*, 203–211. 10.1037/0022-006X.73.2.203.

Cooper, P. J., Coker, S., & Fleming, C. (1996). An evaluation of the efficacy of supervised cognitive behavioral self-help for bulimia nervosa. *Journal of Psychosomatic Research, 40*, 281–287. 10.1016/0022-3999(96)00535-1.

Cumming, G. (2012). *Understanding the new statistics: Effect sizes, confidence intervals, and meta-analysis*. New York, NY: Routledge.

Dalle Grave, R., Calugi, S., & Marchesini, G. (2008). Compulsive exercise to control shape or weight in eating disorders: Prevalence, associated features, and treatment outcome. *Comprehensive Psychiatry, 49*, 346–352. doi: 10.1016/j.comppsych.2007.12.007

Dalle Grave, R., Calugi, S., & Marchesini, G. (2009). Self-induced vomiting in eating disorders: Associated features and treatment outcome. *Behaviour Research and Therapy, 47*, 680–684. doi: 10.1016/j.brat.2009.04.010

Dalle Grave, R., Calugi, S., & Marchesini, G. (2012). Objective and subjective binge eating in underweight eating disorders: Associated features and treatment outcome. *International Journal of Eating Disorders, 45*, 370–376. 10.1002/eat.20943.

Dawkins, H., Watson, H. J., Egan, S. J., & Kane, R. T. (2013). Weight suppression in bulimia nervosa: Relationship with cognitive behavioral therapy outcome. *International Journal of Eating Disorders, 46*, 586–593. 10.1002/eat.22137.

Fahy, T. A., Eisler, I., & Russell, G. F. (1993). Personality disorder and treatment response in bulimia nervosa. *British Journal of Psychiatry, 162*, 765–770. 10.1192/bjp.162.6.765.

Fahy, T. A., & Russell, G. F. (1993). Outcome and prognostic variables in bulimia nervosa. *International Journal of Eating Disorders, 14*, 135–145. 10.1002/1098-108X(199309).

Fairburn, C. G. (2008). *Cognitive behavior therapy and eating disorders*. New York, NY: Guilford Press.

Fairburn, C. G., Bailey-Straebler, S., Basden, S., Doll, H. A., Jones, R., Murphy, R., et al. (2015). A transdiagnostic comparison of enhanced cognitive behaviour therapy (CBT-E) and interpersonal psychotherapy in the treatment of eating disorders. *Behaviour Research and Therapy, 70*, 64–71. 10.1016/j.brat.2015.04.010.

Fairburn, C. G., & Beglin, S. J. (1994). Assessment of eating disorders: Interview or self-report questionnaire? *International Journal of Eating Disorders, 16*, 363–370.

Fairburn, C. G., Cooper, Z., Doll, H. A., O'Connor, M. E., Bohn, K., Hawker, D. M., et al. (2009). Transdiagnostic cognitive-behavioral therapy for patients with eating disorders: A two-site trial with 60-week follow-up. *American Journal of Psychiatry, 166*, 311–319. 10.1176/appi.ajp.2008.08040608.

Fairburn, C. G., Cooper, Z., & Shafran, R. (2003). Cognitive behaviour therapy for eating disorders: A "transdiagnostic" theory and treatment. *Behaviour Research and Therapy, 41*, 509–528. 10.1016/S0005-7967(02)00088-8.

Fritz, M. S., & Mackinnon, D. P. (2007). Required sample size to detect the mediated effect. *Psychological Science, 18*, 233–239. 10.1111/j.1467-9280.2007.01882.x.

Garner, D. M., Olmstead, M. P., & Polivy, J. (1983). Development and validation of a multidimensional eating disorder inventory for anorexia nervosa and bulimia. *International Journal of Eating Disorders, 2*, 15–34.

Ghaderi, A. (2006). Does individualization matter? A randomized trial of standardized (focused) versus individualized (broad) cognitive behavior therapy for bulimia nervosa. *Behavior Research and Therapy, 44*, 273–288. 10.1016/j.brat.2005.02.004.

Grilo, C. M., Masheb, R. M., White, M. A., Gueorguieva, R., Barnes, R. D., Walsh, B. T., 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. 10.1016/j.brat.2014.04.002.

Grilo, C. M., Masheb, R. M., Wilson, G. T., & Crosby, R. D. (2012a). Predictors and moderators of response to cognitive behavioral therapy and medication for the treatment of binge eating disorder. *Obesity, 18*, S84–S84. 10.1037/a0027001.

Grilo, C. M., White, M. A., Gueorguieva, R., Wilson, G. T., & Masheb, R. M. (2013). Predictive significance of the overvaluation of shape/weight in obese patients with binge eating disorder: Findings from a randomized controlled trial with 12-month follow-up. *Psychological Medicine, 43*, 1335–1344. 10.1017/S0033291712002097.

Grilo, C. M., White, M. A., Wilson, G. T., Gueorguieva, R., & Masheb, R. M. (2012b). Rapid response predicts 12-month post-treatment outcomes in binge-eating disorder: Theoretical

[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.

and clinical implications. *Psychological Medicine*, *42*, 807–817. 10.1017/S0033291711001875.

Hay, P. J., Bacaltchuk, J., Stefano, S., & Kashyap, P. (2009). Psychological treatments for bulimia nervosa and binging. *Cochrane Database of Systematic Reviews*, *2009*(4), 1–170. 10.1002/14651858.CD000562.pub3.

Henderson, M., & Freeman, C. P. (1987). A self-rating scale for bulimia. The'BITE'. *The British Journal of Psychiatry*, *150*(1), 18–24.

Hilbert, A., Hildebrandt, T., Agras, W. S., Wilfley, D. E., & Wilson, G. T. (2015). Rapid response in psychological treatments for binge eating disorder. *Journal of Consulting and Clinical Psychology*, *83*, 649–654. 10.1037/ccp0000018.

Högdahl, L., Birgegård, A., & Björck, C. (2013). How effective is bibliotherapy-based self-help cognitive behavioral therapy with Internet support in clinical settings? Results from a pilot study. *Eating And Weight Disorders*, *18*, 37–44. 10.1007/s40519-013-0005-3.

Kazdin, A. E. (2007). Mediators and mechanisms of change in psychotherapy research. *Annual Review of Clinical Psychology*, *3*, 1–27. 10.1146/annurev.clinpsy.3.022806.091432.

Kraemer, H. C. (2016). Messages for clinicians: Moderators and mediators of treatment outcome in randomized clinical trials. *American Journal of Psychiatry*, *173*, 672–679. doi: 10.1176/appi.ajp.2016.15101333

Kraemer, H. C., Wilson, G. T., Fairburn, C. G., & Agras, W. S. (2002). Mediators and moderators of treatment effects in randomized clinical trials. *Archives of General Psychiatry*, *59*, 877–883. 10.1001/archpsyc.59.10.877.

Lammers, M. W., Vroling, M. S., Ouwens, M. A., Engels, R. C., & van Strien, T. (2015). Predictors of outcome for cognitive behaviour therapy in binge eating disorder. *European Eating Disorders Review*, *23*, 219–228. 10.1002/erv.2356.

Leung, N., Waller, G., & Thomas, G. (2000). Outcome of group cognitive-behavior therapy for bulimia nervosa: The role of core beliefs. *Behavior Research and Therapy*, *38*, 145–156. 10.1016/S0005-7967(99)00026-1.

Linardon, J., Brennan, L., & de la Piedad Garcia, X. (2016). Rapid response to eating disorder treatment: A systematic review and meta-analysis. *International Journal of Eating Disorders*, *49*, 905–919. 10.1002/eat.22595.

Loeb, K. L., Wilson, G. T., Gilbert, J. S., & Labouvie, E. (2000). Guided and unguided self-help for binge eating. *Behavior Research and Therapy*, *38*, 259–272. 10.1016/S0005-7967(99)00041-8.

Loeb, K. L., Wilson, G. T., Labouvie, E., Pratt, E. M., Hayaki, J., Walsh, B. T., et al. (2005). Therapeutic alliance and treatment adherence in two interventions for bulimia nervosa: A study of process and outcome. *Journal of Consulting and Clinical Psychology*, *73*, 1097–1106. 10.1037/0022-006X.73.6.1097.

Marrone, S., Mitchell, J. E., Crosby, R., Wonderlich, S., & Jollie-Trottier, T. (2009). Predictors of response to cognitive behavioral treatment for bulimia nervosa delivered via telemedicine versus face-to-face. *International Journal of Eating Disorders*, *42*, 222–227. 10.1002/eat.20603.

Masheb, R. M., & Grilo, C. (2007). Rapid response predicts treatment outcomes in binge eating disorder: Implications for stepped care. *Journal of Consulting and Clinical Psychology*, *75*, 639–644. doi: 10.1037/0022-006X.75.4.639

Masheb, R. M., & Grilo, C. M. (2008a). Examination of predictors and moderators for self-help treatments of binge-eating disorder. *Journal of Consulting and Clinical Psychology*, *76*, 900–904. 10.1037/a0012917.

Masheb, R. M., & Grilo, C. M. (2008b). Prognostic significance of two sub-categorization methods for the treatment of binge eating disorder: Negative affect and overvaluation predict, but do not moderate, specific outcomes. *Behavior Research and Therapy*, *46*, 428–437. 10.1016/j.brat.2008.01.004.

Moher, D., Liberati, A., Tetzlaff, J., & Altman, D. G. (2009). Preferred reporting items for systematic reviews and meta-analyses: The PRISMA statement. *Annals of Internal Medicine*, *151*, 264–269. 10.7326/0003-4819-151-4-200908180-00135.

Murphy, R., Cooper, Z., Hollon, S. D., & Fairburn, C. G. (2009). How do psychological treatments work? Investigating mediators of change. *Behaviour Research and Therapy*, *47*(1), 1–5. 10.1016/j.brat.2008.10.001.

Porter, E., & Chambless, D. L. (2015). A systematic review of predictors and moderators of improvement in cognitive-behavioral therapy for panic disorder and agoraphobia. *Clinical Psychology Review*, *42*, 179–192.

Poulsen, S., Lunn, S., Daniel, S. I., Folke, S., Mathiesen, B. B., Katznelson, H., et al. (2014). A randomized controlled trial of psychoanalytic psychotherapy or cognitive-behavioral therapy for bulimia nervosa. *American Journal of Psychiatry*, *171*, 109–116. 10.1176/appi.ajp.2013.12121511.

Raykos, B. C., McEvoy, P. M., Erceg-Hurn, D., Byrne, S. M., Fursland, A., & Nathan, P. (2014). Therapeutic alliance in enhanced cognitive behavioural therapy for bulimia nervosa: Probably necessary but definitely insufficient. *Behaviour Research and Therapy*, *57*, 65–71. 10.1016/j.brat.2014.04.004.

Raykos, B. C., Watson, H. J., Fursland, A., Byrne, S. M., & Nathan, P. (2013). Prognostic value of rapid response to enhanced cognitive behavioral therapy in a routine clinic sample of eating disorder outpatients. *International Journal of Eating Disorders*, *46*, 764–770. 10.1002/eat.22169.

Schlup, B., Meyer, A. H., & Munsch, S. (2010). A non-randomized direct comparison of cognitive-behavioral short- and long-term treatment for binge eating disorder. *Obesity Facts*, *3*(4), 261–266. 10.1159/000319538.

Shapiro, J. R., Berkman, N. D., Brownley, K. A., Sedway, J. A., Lohr, K. N., & Bulik, C. M. (2007). Bulimia nervosa treatment: A systematic review of randomized controlled trials. *International Journal of Eating Disorders*, *40*, 321–336. 10.1002/eat.20372.

Spangler, D. L., Baldwin, S. A., & Agras, W. S. (2004). An examination of the mechanisms of action in cognitive behavioral therapy for bulimia nervosa. *Behavior Therapy*, *35*, 537–560. 10.1016/S0005-7894(04)80031-5.

Steketee, G., & Chambless, D. L. (1992). Methodological issues in prediction of treatment outcome. *Clinical Psychology Review*, *12*(4), 387–400.

Striegel-Moore, R. H., Wilson, G. T., DeBar, L., Perrin, N., Lynch, F., Rosselli, F., et al. (2010). Cognitive behavioral guided self-help for the treatment of recurrent binge eating. *Journal of Consulting and Clinical Psychology*, *78*, 312–321. 10.1037/a0018915.

Thompson-Brenner, H., Shingleton, R. M., Sauer-Zavala, S., Richards, L. K., & Pratt, E. M. (2015). Multiple measures of rapid response as predictors of remission in cognitive behavior therapy for bulimia nervosa. *Behavior Research and Therapy*, *64*, 9–14. 10.1016/j.brat.2014.11.004.

Thompson-Brenner, H., Shingleton, R. M., Thompson, D. R., Satir, D. A., Richards, L. K., Pratt, E. M., et al. (2016). Focused vs. broad enhanced cognitive behavioral therapy for bulimia nervosa with comorbid borderline personality: A randomized controlled trial. *International Journal of Eating Disorders*, *49*, 36–49.

Turner, H., Bryant-Waugh, R., & Marshall, E. (2015). The impact of early symptom change and therapeutic alliance on treatment outcome in cognitive-behavioural therapy for eating disorders. *Behaviour Research and Therapy*, *73*, 165–169. doi: 10.1016/j.brat.2015.08.006

Vall, E., & Wade, T. D. (2015). Predictors of treatment outcome in individuals with eating disorders: A systematic review and meta-analysis. *International Journal of Eating Disorders, Advanced online publication*. 10.1002/eat.22411.

Williams, S. E., Watts, T. K. O., & Wade, T. D. (2012). A review of the definitions of outcome used in the treatment of bulimia nervosa. *Clinical Psychology Review*, *32*, 292–300. 10.1016/j.cpr.2012.01.006.

Wilson, T., Fairburn, C. C., Agras, W. S., Walsh, B. T., & Kraemer, H. (2002). Cognitive-behavioral therapy for bulimia nervosa: Time course and mechanisms of change. *Journal of Consulting and Clinical Psychology*, *70*, 267.

Wilson, T., Wilfley, D. E., Agras, W. S., & Bryson, S. (2010). Psychological treatments of binge eating disorder. *Archives of General Psychiatry*, *67*, 94–101. 10.1001/archgenpsychiatry.2009.170.

Wonderlich, S., Peterson, C., Crosby, R., Smith, T., Klein, M., Mitchell, J. E., et al. (2014). A randomized controlled comparison of integrative cognitive-affective therapy (ICAT) and enhanced cognitive-behavioral therapy (CBT-E) for bulimia nervosa. *Psychological Medicine*, *44*, 543–553. 10.1017/S0033291713001098.

Zendegui, E. A., West, J. A., & Zandberg, L. J. (2014). Binge eating frequency and regular eating adherence: The role of eating pattern in cognitive behavioral guided self-help. *Eating Behaviors*, *15*, 241–243. 10.1016/j.eatbeh.2014.03.002.

Zipfel, S., Wild, B., Groß, G., Friederich, H.-C., Teufel, M., Schellberg, D., et al. (2014). Focal psychodynamic therapy, cognitive behaviour therapy, and optimised treatment as usual in outpatients with anorexia nervosa (ANTOP study): Randomised controlled trial. *The Lancet*, *383*, 127–137.

Supporting information

Additional Supporting Information may be found in the online version of this article at the publisher's web site.

Chapter 7: The Cognitive-Behavioural Theory of Eating Disorders

Introduction to Chapter

A central goal of this thesis was to evaluate the cognitive-behavioural theory and treatment of eating disorders. The first study (chapter 5), which documented the efficacy of cognitive-behavioural *treatment*, provided initial, foundational, and indirect evidence in support of the underlying cognitive-behavioural *theory*. A better test of the cognitive-behavioural theory comes from studies that have examined CBT-specific mediators, moderators, and predictors of outcome. The second study (Chapter 6) reviewed this literature and did not identify any reliable mediators, moderators, or predictors. Therefore, the current chapter aims to review a broader body of research (e.g., observational, longitudinal, and cross-sectional) supporting the cognitive-behavioural theory. Given that, at present, the enhanced CBT model (which includes the additional four maintaining mechanisms of mood intolerance, perfectionism, low self-esteem, and interpersonal difficulties) has showed no clear benefit over the original CBT model (see Chapter 1), the following chapter will focus on reviewing the literature that has tested the pathways specified in the original model (see Figure 1.1). The literature review will be followed by identification of clear research gaps, thus establishing the basis for the third and fourth study of this project.

Evidence in Support for the Cognitive-Behavioural Model

A description and schematic representation of the original cognitive-behavioural model was presented in Chapter 2 Figure 1.1. What follows is a review of evidence supporting this model from longitudinal research (prospective and treatment research) and cross-sectional research.

Prospective research. A number of longitudinal studies have examined the role of the cognitive-behavioural factors thought to maintain BN symptoms. Two longitudinal studies have tested the proposed pathways in the cognitive-behavioural model in clinical samples. Fairburn et al. (2003b) examined the natural course of BN (n = 105) and found that higher levels of shape and weight over-concern predicted greater persistence in binge eating over a 15 month period. This relationship was also shown to be mediated by a concurrent increase in dietary restraint (Fairburn et al., 2003b). Note, however, that because there was a simultaneous increase in dietary restraint and binge eating, the direction of the relationship between these variables could not be confirmed. This finding does not therefore provide sufficient evidence to support the hypothesis that dietary restraint prospectively predicts or leads to binge eating. In a later prospective study, Bohon, Stice, and Burton (2009) examined a number of different predictors of binge eating and purging persistence in women with BN (n=96). The authors found that the only significant predictor of binge eating persistence was elevated scores on "expected reward from food", while the only statistically significant predictor of purging persistence was, consistent with the cognitive-behavioural model, elevated levels of dietary restraint (Bohon et al., 2009). Overall, both studies of clinical samples provide support for the idea that dietary restraint and shape and weight over-concern may be crucial maintaining mechanisms of BN symptoms.

Three other prospective studies have also tested the role of cognitive-behavioural maintaining factors in the persistence of bulimic symptomatology in non-clinical samples, including female adolescents (Stice & Agras, 1998), obese women (Rohde, Stice, & Gau, 2016), and male students (Dakanalis et al., 2016). For instance, the association between over-concern with weight and shape/dietary restraint and bulimic symptoms has been consistently supported in these longitudinal studies, while there is mixed support for the pathway linking dietary restraint to binge eating. The only study to test the association between binge eating

and compensatory behaviours longitudinally did not report a significant relationship between these variables (Rohde et al., 2016). Together, findings from these longitudinal studies of clinical and non-clinical samples provide preliminary support for the shape and weight concern-dietary restraint pathway and the dietary restraint-binge/purge symptom pathway specified in the cognitive-behavioural model.

Mediators of response research. A more rigorous evaluation of the cognitive-behavioural model comes from studies that have tested mediators of change during CBT. To date, two studies have tested whether changes in the hypothesised maintaining mechanisms (dietary restraint, and shape and weight over-concern) mediate symptom improvement during CBT. In the first study, Wilson et al (2002) conducted secondary analyses using data (n=154) collected in a multisite RCT comparing CBT-BN to IPT. The authors examined whether changes in binge eating and purge frequencies at post-treatment and follow-up were mediated by changes in both theory-specific (i.e., weight/shape concerns, dietary restraint) and common treatment mechanisms (i.e., perceived self-efficacy, therapeutic alliance). Consistent with the predictions outlined by the cognitive-behavioural model, it was found that changes in dietary restraint at week four mediated or explained the changes in binge eating and purge frequencies at post-treatment and eight month follow-up. Critically, however, this effect did not interact with treatment type, which suggests that both CBT and IPT might "work" through modifying dietary restraint, despite IPT not using strategies to directly target this maintaining mechanism. In addition, changes in self-efficacy from baseline to week 10 also mediated post-treatment purge frequency (Wilson, Fairburn, Agras, Walsh, & Kraemer, 2002). However, because self-efficacy was measured only at baseline and at week 10, and because purge frequency had already decreased by week 10, it could not be determined whether the changes in self-efficacy were a cause or consequence of symptom improvement.

A second study that used a subset of data (*n*=56) from the same multisite trial of CBT-BN also examined a wider range of treatment mediators (Spangler, Baldwin, & Agras, 2004). These authors tested whether CBT treatment manipulations (i.e., behavioural, cognitive, relational, or structural treatment strategies) or purported client mechanisms (i.e., changed in dietary restraint, dysfunctional body-related beliefs, and the degree of engagement in treatment) were associated with changes in weight and shape concerns and vomiting frequency. Using linear growth curve modelling, it was found that (a) an increased use of behavioural treatment strategies[4] was associated with greater changes in shape and weight concerns, (b) an increase in dietary restraint was associated with greater vomiting frequencies, and (c) reductions in dysfunctional body beliefs were associated with greater change in weight concerns (Spangler et al., 2004). Although this study lends support to the predictions outlined in the cognitive-behavioural model, interpretations should be made with caution. In particular, this study considers associations and did not test the direction of these effects, meaning that conclusions about the causal or temporal sequence of the association between these variables could not be made.

The previous two studies found that reductions in dietary restraint during treatment might be a crucial mechanism underpinning CBT's effectiveness. This idea was further reinforced in a study of CBTgsh for binge eating (Zendegui, West, & Zandberg, 2014). Specifically, these authors wanted to explore the relationship between regular eating and binge eating in individuals with BN and BED (*n*=38) who completed a CBTgsh program. Establishing a regular eating pattern, according to Fairburn (2013), is the most potent element of the CBT protocol. Regular eating is prescribed in the first few weeks of CBT to disrupt dietary restraint and restriction, which, according to the cognitive-behavioural model,

[4] To assess use of behavioral treatment strategies, the Coding Scale for Bulimia Nervosa (CCS-BN) was used. The behavioral scale assesses the extent to which therapists probe for problematic behaviors, plan and practice alternative behaviors, attempt to teach behavioral skills, schedule and structure alternative activities, and help identify cues for specific behaviors.

maintains binge eating. Zendegui and colleagues assessed adherence to regular eating principles (i.e., defined as consuming three meals and three snacks) and binge eating frequency throughout the course of the 8 week treatment. The authors found that a higher number of regular eating adherent days per week was significantly associated with lower frequencies of binge eating. The authors argued that the rapid behavioural symptom change that is typically observed within first few weeks of CBT for eating disorders could be a result of this regular eating strategy (Zendegui et al., 2014). While this study lends further support to the hypothesised maintaining role of dietary restraint on binge eating, an important limitation of this study was that the authors did not control for "time" or "session number" in their analyses. Thus, this cross-sectional relationship between regular eating and binge eating frequency cannot tell us whether regular eating led to binge eating reductions or whether it was a consequence of it.

Cross-sectional research. Numerous cross-sectional studies have directly evaluated this model, typically using structural equation modelling (SEM). The cognitive-behavioural model lends itself well to SEM, as this statistical approach can estimate the relationships between a complex set of independent and dependent variables at one time (Muthén & Muthén, 2010).

The cognitive-behavioural model has been directly tested in BN and transdiagnostic samples, and this research has found evidence in support of most of the specified pathways. In particular, shape and weight over-concern has been shown to consistently predict dietary restraint, and binge eating has been shown to consistently predict purging behaviour (e.g., Dakanalis et al., 2015). Although some studies have shown dietary restraint to predict binge eating (Lampard, Tasca, Balfour, & Bissada, 2013), others have reported no relationship between dietary restraint and binge eating (Lampard et al, 2011). This latter finding led to the

speculation that dietary restraint might not be as important in maintaining binge eating as was originally thought. These inconsistencies may, however, be a result of how binge eating was measured in these studies. For instance, Lampard et al (2013) argued that dietary restraint is only a strong predictor of binge eating when binge eating is measured through scales (e.g., binge eating scale) that assess all underlying components of binge eating, including behavioural (frequency), cognitive (perceived loss of control), and affective (guilt) components. Studies that have failed to support this relationship have typically only assessed the behavioural components of binge eating (Lampard, Byrne, McLean, & Fursland, 2011). Overall, cross-sectional research in clinical samples has provided the necessary statistical support for the cognitive-behavioural pathways.

Many more cross-sectional studies have directly tested the cognitive-behavioural model using SEM in non-clinical samples (Hoiles, Egan, & Kane, 2012; Schnitzler, von Ranson, & Wallace, 2012). Although this model was originally designed to understand and treat BN, some have argued that there are multiple benefits to studying this model in those without an eating disorder. First, CBT-BN (or guided self-help versions) is being increasingly administered to individuals who do not meet exact diagnostic criteria for BN or BED, and evidence suggests that CBT-BN is efficacious for these subthreshold cases (Ghaderi & Scott, 2003). This provides indirect evidence that the same maintaining mechanisms are operating to maintain subclinical BN symptomatology, and that CBT can be applied to a broad spectrum of individuals who exhibit symptoms of disordered eating. Second, Fairburn (2008) argues that transdiagnostic CBT-E is a treatment designed to target eating disorder psychopathology, irrespective of diagnosis. This implies that anyone may benefit from CBT-E, so long as eating disorder psychopathology is present. Third, some authors contend that subthreshold eating disorders are understudied (Striegel-Moore et al., 2010), particularly since (a) their prevalence is increasing, and (b) the psychological impairment reported by

subthreshold cases is significant, and (c) they often lead to the development of clinical eating disorders (Hay, 2003). Finally, using a community sample allows for the recruitment of sufficient participants to ensure adequate power for tests of this model, which ultimately provides foundational evidence for the development of future studies that test this model in clinical cases.

These cross-sectional studies have consistently reported a positive relationship between shape and weight concerns and dietary restraint, and binge eating and purging in elite athletes (S. Byrne & McLean, 2002), undergraduate men and women (Dakanalis, Timko, Clerici, Zanetti, & Riva, 2014; Schnitzler et al., 2012), female-only community samples (Hoiles et al., 2012), and overweight children (Decaluwé & Braet, 2005). In addition, several of these have also reported a positive relationship between dietary restraint and binge eating, though this relationship has not always been replicated in studies of non-clinical samples (e.g., Byrne & McLean, 2002). Together, these studies mostly support the pathways specified in the cognitive-behavioural model. Again, the cross-sectional nature of these data do not allow for conclusions about the temporal nature of these paths.

Mechanisms Underpinning the Cognitive-Behavioural Pathways

While numerous longitudinal and cross-sectional observational studies provide support for the pathways specified in the cognitive-behavioural model, the mechanisms that explain or underpin these pathways remain unclear. More specifically, it is not known *how* shape and weight over-concern increases dietary restraint, or *how* dietary restraint increases binge eating. Understanding mediating variables of the cognitive-behavioural pathways is important for several reasons. First, it would eventually enhance and further our understanding of the nature of disordered eating symptomatology, its possible course, and the reasons why certain disordered eating symptoms persist. Second, understanding additional

mediating variables could, in the long-term, lead to improvements in treatment outcomes. This is because there would be more clarity around the possible factors that serve to maintain disordered eating symptomatology, which could then lead to the development of new (or the refinements of existing) therapeutic strategies that eliminate these maintaining mechanisms (Kraemer et al., 2002). I will now propose three additional maintaining factors (body checking, body avoidance, and dichotomous thinking) that might serve to maintain the core mechanisms highlighted in the cognitive-behavioural model. These variables were a focus because cognitive-behavioural conceptualisations of eating disorders clearly highlight their importance toward maintaining certain symptoms, yet, as will be reviewed, limited work has been done to understand the function of these variables. A greater understanding of body checking, body avoidance and dichotomous thinking is important for validating the cognitive-behavioural model.

Body checking. The process of repeatedly examining and scrutinizing aspects of one's body is referred to as body checking (Reas, Whisenhunt, Netemeyer, & Williamson, 2002). Body checking behaviour includes frequent self-weighing, mirror checking, and pinching of body parts to assess for fat. Body checking is common in weight concerned individuals, and is also a strong predictor of psychological impairment (Latner, Mond, Vallance, Gleaves, & Buckett, 2012). Body checking is considered to be a behavioural expression of extreme concerns about weight and shape. This behavioural expression is then postulated to exacerbate and maintain efforts to restrain and restrict eating, thereby making body checking a potential mediating variable between shape and weight concerns and dietary restraint (Fairburn et al., 2003a). More specifically, individuals who are highly concerned about their weight and shape frequently monitor their weight, sometimes more than once a day. These individuals typically pay close attention to any minor or natural body weight

fluctuation. It is hypothesised that this attentional bias fuels further concerns about weight and shape and reinforces subsequent efforts to restrain and/or restrict eating, regardless of the outcomes. This is because (a) if weight increases or remains constant it is taken as evidence that the previous diet was not strict enough, and (b) if weight decreases it is taken as evidence that the previous diet was effective (Shafran, Fairburn, Robinson, & Lask, 2004).

CBT for eating disorders addresses frequent weight and shape checking. For instance, the "weekly weighing" strategy is an important component of CBT that addresses this potential maintaining mechanism (Fairburn, 2008). The goal of the weekly weighing strategy is to reduce a client's over-concern with weight (the core psychopathology). The therapist initially provides the client with a rationale for weekly weighing, in particular by outlining the aversive effects (e.g., attentional biases, further weight concerns) of frequent or obsessive weight checking. The therapist then educates the client about normal week-to-week weight fluctuations. By having supervised weekly "weigh-ins", the therapist can help the client accurately interpret any changes in body weight over the weeks. Eventually, the client should come to accept any minor week-to-week body weight fluctuations; this acceptance is purported to correlate with reductions in weight concerns and in dietary restraint (Fairburn, 2008).

The mediating role of body checking on the relationship between weight and shape concerns and dietary restraint is implicit in Fairburn's cognitive-behavioural conceptualisation. Despite body checking being a core focus during CBT, body checking behaviour has received remarkably little research attention. Cross-sectional research has reported consistent correlations between body checking and eating disorder symptoms (Shafran et al., 2004), general psychopathology (Mountford, Haase, & Waller, 2006), and quality of life impairment (Latner et al., 2012), and preliminary longitudinal evidence indicates that body checking predicts dietary restraint in AN (Lavender et al., 2013).

However, no research has tested (a) the role of body checking in the context of evaluating the cognitive-behavioural model, and (b) whether body checking can account for or explain the robust link between shape and weight over-concern and dietary restraint. Such a test is important for enhancing our understanding of the breadth of factors that maintain eating disorder psychopathology.

Body avoidance. Body avoidance refers to behaviour that is designed to prevent the individual from seeing their body and being aware of it (Rosen, Srebnik, Saltzberg, & Wendt, 1991). Body avoidance behaviours include a refusal to be weighed, wearing baggy clothes to disguise one's shape, or covering up mirrors. Body avoidance is highly correlated with body checking; it is very common for weight and shape concerned individuals to repeatedly switch from checking (e.g., self-weighing) to avoiding (e.g., wearing baggy clothes) throughout the day (Shafran et al., 2004). Like body checking, body avoidance is also considered a behavioural expression of weight and shape concerns — it is considered problematic because it provides little opportunity to disprove maladaptive assumptions and beliefs held toward weight and shape, and, like body checking, exacerbates dietary restraint (Fairburn et al., 2003).

Body avoidance is also a target during CBT for eating disorders. First, like weight checking, weight avoidance is also addressed via the "weekly weighing" procedure. Second, the "body image module" administered during the mid to later stages of CBT contains a collection of treatment procedures designed to target body avoidance (Fairburn, 2008). For example, a particular form of exposure treatment is implemented in this module, where the client is encouraged to get used to the sight and feel of their own body. Some individuals, for instance, avoid the site of their body by getting dressed and undressed in the dark. If this is the case, the therapist might encourage the client to make small changes to this behavioural

repertoire. This change may be initially putting candles in room while getting dressed and undressed. The end objective is to get the individual to become comfortable getting dressed and undressed with the lights on. Another example is that some people may avoid touching their body. If this is the case, the therapist might encourage the client to be more focused or mindful during instances where "body touching" is required. When washing oneself, the client might be encouraged to first wash the self with a sponge, focusing on the neutral body parts (e.g., feet, hands). Then the client is encouraged to gradually work toward being able to wash the whole body comfortably using bare hands (Fairburn, 2008). These exposure-related techniques are purported to reduce body avoidant behaviours, which is therefore proposed to indirectly target shape concerns.

Despite being an important part of CBT for eating disorders, there has been relatively little research examining the role of body avoidance on eating disorder psychopathology. Cross-sectional research has reported strong links between body avoidance and eating disorder psychopathology (Latner et al., 2012; Reas, Grilo, Masheb, & Wilson, 2005; Rosen et al., 1991). Higher baseline levels of body avoidance have also been shown to predict poor treatment outcome (Olmsted, MacDonald, McFarlane, Trottier, & Colton, 2015). Critically, while body avoidance might be a key mechanism underpinning the relationship between shape and weight concerns and dietary restraint, no research has tested this hypothesis. Furthermore, no studies that have evaluated the cognitive-behavioural model have examined the role of body avoidance on the cognitive-behavioural mechanisms.

Dichotomous thinking. Dichotomous thinking refers to the tendency for one to appraise situations or events in a polarized fashion (Egan, Piek, Dyck, & Rees, 2007). Dichotomous thinking could be a key maintaining mechanism underpinning the link between dietary restraint and binge eating. The restraint theory of eating behaviour (Herman & Mack,

1975) and the cognitive-behavioural model (Fairburn et al., 1993b) proposes that one of the reasons why an individual with elevated levels of dietary restraint regularly binge eats is because of an all-or-none thinking style around food and dieting. More specifically, any deviation from a rigid dietary rule (e.g., consumption of "forbidden" food) typically triggers a dichotomous thinking reaction (e.g., "I've blown my diet"), which in turn prompts an individual to abandon their dieting regime, resulting in episodes of uncontrollable binge eating (Fairburn et al., 1993b).

One treatment component of focused versions of CBT is specifically designed to target dichotomous thinking. This treatment component, called the "dietary rules" strategy, is implemented during the later stages of CBT (Fairburn, 2008). This procedure addresses food avoidance — the tendency to completely exclude so-called forbidden foods in fear of weight gain (Fairburn, 2008). Like body avoidance, food avoidance is addressed through a form of exposure therapy. In this exposure technique, clients are encouraged to identify, list, and group foods ranging from "least forbidden" to "most forbidden". Clients are then encouraged to gradually introduce small amounts of these foods into their diet, starting from the least forbidden, and working up to the most forbidden. After this particular exposure, an individual's cognitions around these foods are "restructured", and the thought or act of eating these foods should not elicit anxiety, should not prompt an all-or-none reaction to dietary transgressions, and should therefore prevent binge eating episodes (Fairburn, 2008).

Research on the role of dichotomous thinking in disordered eating symptomatology is scarce. Prospective research has shown dichotomous thinking to predict weight regain in obese individuals (Byrne, Cooper, & Fairburn, 2004), and cross-sectional research has shown dichotomous thinking to mediate the relationship between dietary restraint and self-reported weight regain in a community sample (Palascha, van Kleef, & van Trijp, 2015). These findings provide preliminary and indirect support for the hypothesis that dichotomous

thinking negatively impacts eating behaviour. Early experimental research also highlights the important role of dichotomous thinking. In one study (Knight & Boland, 1989), female students were asked to rate certain foods from zero (permitted foods) to eight (forbidden foods). Participants were then categorised as restrained or unrestrained eaters, and were randomized to either a control group, a one or two serve "forbidden food" (chocolate milkshake) preload group, or a one or two serve "non-forbidden food" (cottage cheese) preload group. Critically, while the calorie content of the milkshake and cottage cheese was matched, in the early rating task, the chocolate milkshake was rated as significantly more "forbidden". The dependent variable was the amount of ice-cream consumed (a measure of eating control) during a taste test that followed the preload. The authors found that, while the type of preload had no effect on *unrestrained* eaters ice-cream consumption, *restrained* eaters randomized to the forbidden preload (i.e., hypothesised to trigger the all-or-none reaction) condition consumed significantly more ice-cream on the later taste test than restrained eaters randomized to the non-forbidden food preload. The authors then compared the amount of ice-cream consumed for restrained and unrestrained eaters in the *same* preload condition. Here, for those randomized to the non-forbidden food preload, unrestrained eaters consumed significantly more ice-cream than restrained eaters (because non-forbidden food should not elicit a reaction in restrained eaters). However, for those randomized to the forbidden food preload, restrained eaters consumed significantly more ice-cream than non-restrained eaters (Knight & Boland, 1989). These findings lend support to the idea that an all-or-none thinking style around food and dieting plays a pivotal role in affecting an individual's control over their eating, particularly for those with elevated levels of dietary restraint.

Chapter Summary

In sum, research has sought to directly evaluate the cognitive-behavioural model of eating disorders. There is limited and preliminary longitudinal evidence supporting the cognitive-behavioural model and its pathways. Much more research has evaluated this model cross-sectionally, and there is support for the proposed cognitive-behavioural pathways in both clinical and non-clinical samples. At present, however, little is known about the potential mechanisms that underpin these pathways or the mechanisms of change that underpin CBTs effectiveness. Identification of additional maintaining mechanisms is one important avenue toward understanding CBT's mechanisms of change. This review highlighted how body checking, body avoidance, and dichotomous thinking could be additional eating disorder maintaining mechanisms. Yet, empirical research studying the role of the variables is limited. Thus, the third study of this thesis will aim to cross-sectionally evaluate the cognitive-behavioural model, and to consider whether body checking, body avoidance, and dichotomous thinking mediate these proposed pathways. The forth study will then examine the proposed mechanisms of action outlined in the cognitive-behavioural in participants who were treated with a CBT guided self-help program.

Chapter 8: Evaluating an expanded cognitive-behavioural model of bulimia nervosa: The role of body checking, body avoidance, and dichotomous thinking (Article 3).

This chapter consists of an article submitted for publication (currently revised and resubmitted) to the journal *Eating Behaviours*. As outlined in chapter 8, several cross-sectional studies using clinical and non-clinical samples have evaluated the cognitive-behavioural model using SEM, and have mostly found support for the cognitive-behavioural pathways. However, additional maintaining factors hypothesized to mediate these pathways have not been tested. These additional factors include body checking, body avoidance, and dichotomous thinking. Therefore, the aim of this study is to cross-sectionally evaluate an expanded cognitive-behavioural of eating disorders (which includes these additional maintaining factors) using SEM in a large non-clinical sample.

Abstract

Although empirical support for the cognitive-behavioural model of bulimia nervosa (BN) exists, the mechanisms that explain the pathways outlined in this model are unclear. Body checking and body avoidance are two key variables that might explain how and why shape and weight over-concern is associated with dietary restraint, and dichotomous thinking might be a key variable explaining how and why dietary restraint is associated with binge eating. While these three variables are recognised as important, the precise role they play in maintaining eating disorder psychopathology is unclear. This study aimed to validate the original cognitive-behavioural model of BN, and to test an expanded cognitive-behavioural model of BN (i.e., with body checking, body avoidance, and dichotomous thinking included). Data were collected from 397 participants recruited through the community. The original and expanded cognitive-behavioural model was analysed using structural equation modelling. The original model provided an acceptable model fit and the hypothesised pathways were supported. In the expanded model, body checking (but not avoidance) partially mediated the relationship between shape and weight over-concern and dietary restraint. Dichotomous thinking fully mediated the relationship between dietary restraint and binge eating. The expanded model explained three times the amount of variance in bulimic symptoms than the original model, but only when a path from purging to shape and weight concerns was added. This study directly validated the cognitive-behavioural model, and provides empirical support for the inclusion of dichotomous thinking, body checking, and body avoidance as potentially important maintaining mechanisms to be addressed during interventions.

Keywords: Cognitive-behavioural model; bulimia nervosa; eating disorder; body checking; dichotomous thinking; body avoidance

The cognitive-behavioural model of bulimia nervosa (BN, see Figure 8.1), which is the foundation for cognitive-behavioural therapy for BN (CBT-BN), outlines the cognitive and behavioural processes that maintain BN (Fairburn et al., 1993b). Empirical support exists for the cognitive-behavioural model in clinical and non-clinical samples. Two longitudinal studies have found evidence supporting this model in individuals with BN (Fairburn et al., 2003b; Wilson et al., 2002). Recently, cross-sectional studies have directly tested the cognitive-behavioural model using structural equation modelling (SEM), and research has consistently found shape and weight concerns to be associated with dietary restraint, and binge eating to be associated with purging behaviours in a range of clinical and non-clinical samples (Lampard et al., 2011; See Figure 8.1 for all studies that have tested this model). However, the relationship between dietary restraint and binge eating has received mixed findings, leading some authors to speculate that dietary restraint may not be as important at maintaining binge eating as originally proposed (Lampard et al., 2011).

Although there is empirical support for most of the pathways proposed in the model, little is known about the mechanisms that explain these pathways. Since shape and weight concerns and dietary restraint are, according to the cognitive-behavioural model, two of the main maintaining mechanisms that are a primary target for CBT-BN (and the latest version, CBT-E), it is therefore important to identify the variables that explain why shape and weight concerns is associated with restraint, and why restraint is associated with binge eating. Identification of such mechanisms is likely to improve CBT outcomes (Murphy et al., 2009).

Below we discuss three potential mechanisms that might underlie the pathways outlined in the cognitive-behavioural model.

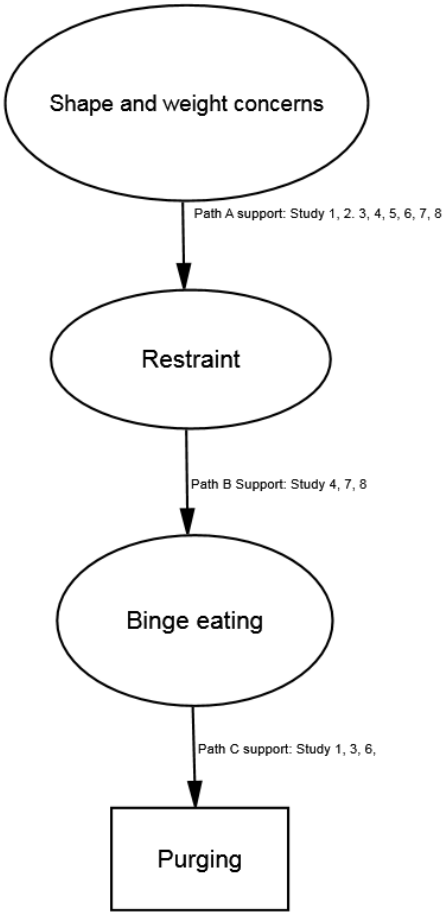


Figure 8.1: The Original Cognitive-Behavioural Model of Bulimia Nervosa

Note. Study 1= S. M. Byrne and N. J. McLean (2002); 2= Dakanalis et al. (2015); 3= Dakanalis et al. (2014); 4= Decaluwé and Braet (2005); 5= Hoiles et al. (2012); 6=Lampard et al (2011); 7= Lampard et al. (2013); 8= Schnitzler et al. (2012); For path B, study 2, 3, 5, and 6 observed no relationship, while study 1 observed a negative relationship. Study 1, 3 and 6 were the only studies to test path C.

Body Checking

Body checking refers to the repeated checking and scrutinising of one's body (Reas et al., 2002). Body checking behaviours include self-weighing and mirror checking. Body checking is considered a behavioural expression of weight and shape concerns, and is hypothesised to maintain dietary restraint in several ways (Fairburn et al., 2003a). For example, for those who weigh themselves frequently, unwarranted attention is typically devoted to natural body weight fluctuations. This frequent monitoring reinforces subsequent dieting, regardless of whether weight has increased or decreased, because (a) weight increases are taken as evidence that the previous diet was not strict enough or (b) weight decreases are taken as evidence that the previous diet was effective (Fairburn, 2013). Indeed, CBT interventions address repeated weight checking via the "weekly weighing" procedure. Although cross-sectional research has reported links between these variables (Linardon & Mitchell, 2017; Mountford et al., 2006), no research has tested whether body checking explains the link between shape and weight concerns and dietary restraint.

Body Avoidance

Body avoidance refers to the active avoidance of situations that elicit concerns about body weight or shape (Rosen & Ramirez, 1998). Avoidance behaviours include a refusal to be weighed, wearing baggy clothes as a "disguise", or the covering up of mirrors. It is common for checking (self-weighing) and avoidance (wearing baggy clothes) to co-occur. Indeed, body checking and avoidance are moderately correlated (Legenbauer et al., 2017). Body avoidance is assumed to mediate the relationship between shape and weight concerns and dietary restraint (Shafran et al., 2004). Avoidant behaviours, which are behavioural expressions of shape and weight concerns, provide little opportunity to disprove maladaptive assumptions or fears regarding weight and shape ("I know I will have put on weight if I

check"), so that a conclusion is made to persist with dieting (Fairburn et al., 2003a). Body avoidance is also addressed during CBT via the "weekly weighting" and body shape exposure techniques (Fairburn et al., 1993b). Little research has tested the role of body avoidance on disordered eating, and while associations between these variables exist (Rosen et al., 1991), research has yet to test whether body avoidance explains the link between shape and weight concerns and dietary restraint.

Dichotomous Thinking

Dichotomous thinking refers to the tendency to interpret situations in a polarised manner (Egan et al., 2007). Dichotomous thinking is considered a key maintaining mechanism of eating disorder psychopathology. It is assumed that one reason why individuals with elevated levels of dietary restraint binge eat is because of their polarised view of food ("good" and "bad") and dieting ("success and "failure"). The cognitive-behavioural model hypothesises that any deviation from a diet primes a dichotomous thinking tendency because the behavioural transgression (breaking a diet rule) is interpreted as a catastrophic failure ("my diet is ruined"). This then prompts an individual to abandon their diet and, because cognitive control over eating is disrupted, engage in uncontrolled binge eating (Fairburn et al., 1993b). The potential mediating role of dichotomous thinking on dietary restraint and binge eating has yet to be tested.

The Current Study

Overall, this study aims to validate the original cognitive-behavioural model of BN, and to test an expanded cognitive-behavioural model (i.e., with body checking, body avoidance, and dichotomous thinking). The expanded model, including the proposed mediational mechanisms, can be seen in Figure 8.2. It is important to note that, although an enhanced transdiagnostic theory and treatment (CBT-E) was more recently developed (Fairburn, 2008), the focus of the current study is on the original model. This decision was

made for two reasons. First, the additional maintaining mechanisms posited by the enhanced model (i.e., self-esteem, mood intolerance, interpersonal difficulties, and perfectionism) are purported to operate in only a subset of clinical cases. On the other hand, the maintaining mechanisms proposed in the original model are said to apply to all cases. Second, the focused version of CBT-E (i.e., the "default" version) targets the same maintaining mechanisms proposed in the original model, but can now instead be applied across diagnoses rather than just for BN (Fairburn, 2008). Thus, improving the treatment of eating disorders will come from advancing our understanding of the maintaining mechanisms (Figure 8.1) that have strong empirical support (Byrne & McLean, 2002).

The proposed models are to be tested in a non-clinical community sample. Standard CBT-BN (or guided self-help variants) is being increasingly administered to community-based men and women, and there is evidence supporting the efficacy of this treatment in non-clinical samples, suggesting that the same maintaining mechanisms operate in non-clinical cases (Kenardy et al., 2002). In addition, testing complex models using structural equation modelling (SEM) techniques requires a large sample. Obtaining a large clinical sample of individuals with eating disorders is sometimes not feasible. The use of a community sample therefore allows for the recruitment of enough participants to ensure adequate statistical power for testing this model using SEM. Testing the proposed models in a community sample therefore provides important foundational evidence for future studies that aim to test these models in clinical cases. For instance, if variables tested in this model show robust associations to disordered eating symptoms in non-clinical cases, then these findings might indicate that the same variables could also serve important functions in clinical cases; hence narrowing does the search for additional maintaining mechanisms in clinical cases.

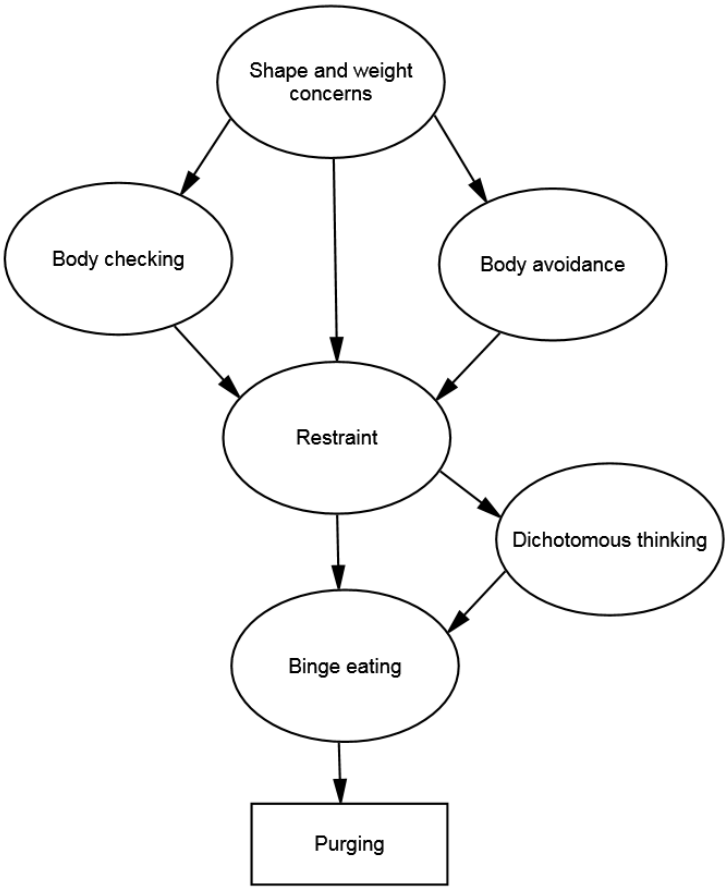


Figure 8.2: The Expanded Cognitive-Behavioural Model with Body Checking, Body Avoidance, and Dichotomous Thinking Included.

Method

Participants

Data from 397 (77 males and 320 females) participants recruited through the community were analysed. Participants' age ranged between 18-65 years. The mean age was 24.89 years (*SD*=8.58) and the mean BMI was 25.17 years (*SD*=5.43). Most participants lived in Australia (96%).

Measures

Weight and shape concern, dietary restraint and purging. The dietary restraint and weight and the shape concern subscales of the Eating Disorder Examination Questionnaire (EDE-Q, version 6.0) were used (Fairburn & Beglin, 1994). Each item from these subscales is rated along a seven point scale, ranging from zero to six, where higher scores indicate greater levels of weight and shape concerns and dietary restraint. Internal consistency for these subscales was acceptable (Cronbach's alpha of .81, .86 and .90 respectively). Purging was also assessed via the EDE-Q, and was measured as the number of vomiting, laxative misuse, and/or diuretic misuse episodes over the past month.

Body checking. The 23-item Body Checking Questionnaire (BCQ) was used to assess the extent to which one repeatedly checks and scrutinizes their body (Reas et al., 2002). Each item is rated along a five point scale, ranging from one (never) to five (very often), and higher scores indicate more frequent body checking. Internal consistency was acceptable in the current sample (α=.94) and good test re-test reliability (*r*=.94) has been reported (Reas et al., 2002).

Body avoidance. The 19-item Body Image Avoidance Questionnaire (BIA) was used to assess the extent to which an individual avoids situations that elicit concerns about body weight and shape (Rosen et al., 1991). Each item is rated along a six point scale, ranging from zero (never) to five (always), and higher scores indicate higher levels of body

avoidance. Internal consistency was adequate in the current sample (α=.87) and good test-retest reliability (*r*=.87) has been reported (Rosen et al., 1991).

Dichotomous thinking. The eating subscale of the Dichotomous Thinking in Eating Disorder Scale (DTES) was used to assess the extent to which an individual thinks about food or dieting in an all-or-none fashion (Byrne, Allen, Dove, Watt, & Nathan, 2008). Each item is rated along a four point scale, ranging from one (not at all true) to five (very true), and higher scores indicate greater levels of dichotomous thinking. Internal consistency was acceptable (α=.87) in the current sample.

Binge eating. The 16-item Binge Eating Scale (BES) was used to assess behavioural, cognitive, and affective components of binge eating (Gormally, Black, Daston, & Rardin, 1982). Behavioural components of binge eating include, for example, the frequency or speed of eating (e.g., "I have a habit of bolting down my food without really chewing it, and when this happens I usually feel uncomfortably stuffed because I've eaten too much"), cognitive components include thoughts associated with binge eating (e.g., "It seems to me that most of my waking hours are pre-occupied by thoughts about eating or not eating. I feel like I'm constantly struggling not to eat"), and affective components include feelings after binge eating (e.g., "almost all of the time I experience strong guilt or self-hate after I overeat"). Internal consistency was adequate (α=.91), and good test-retest reliability(*r*=.87) has been reported (Timmerman, 1999).

Procedure

Ethics approval was granted by the Human Research Ethics Committee and Australian Catholic University (ACU). An advertisement detailing that the study sought to understand the factors that maintain problem eating behaviour was distributed to a variety of locations, including undergraduate psychology programs, social media forums, and the researchers' personal contacts. Participants who expressed interest were provided with a link

to that took them directly to the consent form, and once consent was obtained, the questionnaire battery was presented. Participants completed the questionnaire battery at a time and location of convenience. Details about the characteristics of all participants are presented in Table 8.1

Data Analysis

SEM with robust maximum likelihood estimations was performed using Mplus (Muthén & Muthén, 2010). SEM estimates a measurement and structural model. The measurement model estimates the relationship between indicators (items) and the latent construct while taking into account measurement error. Items that loaded poorly on their construct were removed. The structural model retains the components of the measurement model and tests the relationships between the latent constructs. Model fit was evaluated by several indices (see below). Non-significant pathways were trimmed; paths were added based on modification indices (MI). Model fit was then re-evaluated for the trimmed/revised model and compared to the hypothesised model using the Satorra-Bentler scaled chi-square statistic. The scaled chi-square statistic is robust to violations of normality (Satorra & Bentler, 2001). Significant chi-square differences indicate that the trimmed/revised model provides a better fit.

Measurement and structural model fit was determined through various fit indices, including the comparative-fit-index (CFI), the Tucker–Lewis Index (TLI), the standardized root-mean-square residual (SRMR), and the root-mean-square-error of approximation (RMSEA). An acceptable model fit is indicated by CFA and TLI values between .90 and .94 and SRMR and RMSEA values between .06 and .10, while an excellent model fit is indicated by CFA and TLI values between .95 to 1.00 and SRMR and RMSEA values between .00 to .05 (Hu & Bentler, 1999). Values outside these ranges indicate a poor model fit.

Indirect effects outlined were tested using bootstrapping procedures (10,000 bootstrap samples). If the 95% bias-corrected confidence interval does not include zero, then the indirect is statistically significant ($p < .05$). Partial and full mediation was specified. Full mediation occurs when the indirect path is significant, but the direct path is not; partial mediation occurs when both the direct and the indirect paths are significant (Preacher & Hayes, 2008).

Results

Outliers were examined. As only one case exhibited a Mahalanobis value (27.5) that exceeded the critical region, χ^2 (7) = 24.32, $p<.001$, this case was retained. Normality was inspected through histograms. Bingeing and purging distributions were positively skewed. Therefore, robust likelihood estimation and scaled chi-square statistics were used to account for these violations to normality (Satorra & Bentler, 2001).

Measurement Model

The measurement model was estimated for the latent variables. Items with low factor loadings were removed until a good fit was achieved. This approach is consistent with previous research testing this model (Byrne & McLean, 2002; Lampard et al., 2011; Lampard et al., 2013). Seven EDE-Q items loaded on the shape and weight concern construct, 8 BCQ items loaded on the body checking construct, 5 BIA items loaded on the body image avoidance construct, 3 EDE-Q items loaded on the restraint construct, 4 DTES items loaded on the dichotomous thinking construct, and 4 BES items loaded on the binge eating construct. Table 8.1 presents the items retained in the measurement model and descriptive statistics. The measurement model resulted in an acceptable fit, χ^2 (390) = 1040.59, $p<.001$ (CFI=.90, TFI=.89, RMSEA=.06, SRMR=.04), suggesting that all latent variables were adequately operationalised by their respective indicators. The MIs indicate that allowing the error terms of EDE-Q items 22 and 23 (over-evaluation) to correlate would improve model fit

(MI=197.61). Correlated errors indicate that the items measure something else in common in addition to the construct specified (Brown, 2015). Indeed, the over-evaluation of weight and shape, which differentiates clinical from non-clinical cases, has been shown to be a distinct but related construct to the more normative weight and shape concerns experienced by many individuals (Linardon, 2016; Wade, Zhu, & Martin, 2011). Given it was theoretically reasonable, we allowed these errors to correlate, which resulted in a significantly better measurement model fit, $\Delta\chi^2$ (10) = 215.524, p<.001 (CFI=.93, TLI=.93, RMSEA=.053, SRMR=.04).

Table 8.1
Demographic and Psychological Characteristics of the Sample

Variable	***M (SD)***	**Range**	**Clinical norms or M (SD) reported in previous clinical samples**
Demographic variables			
Age in years	24.89 (8.58)	18 - 65	-
BMI	25.17 (5.43)	17.14 - 36.16	-
Psychological Variables			
Shape concerns	2.86 (1.65)	0 – 6	4.72 (1.31)[1]
Weight concerns	2.37 (1.67)	0 – 6	4.07 (1.38)[1]
Dietary restraint	1.92 (1.58)	0 – 6	3.67 (1.59)[1]
Body checking	50.36 (18.54)	23 – 114	82.10 (18.00)[2]
Body avoidance	30.87 (13.82)	0 – 74	40.17 (10.90)[3]
Dichotomous thinking	2.36 (0.91)	1 – 4	3.41 (0.68)[4]
Binge eating severity	13.55 (9.51)	0 – 42	28.80 (6.10)[5]
Purging frequency	1.61 (2.68)	0 – 10	38.14 (44.41)[6]

Note: Shape concern, weight concern, dietary restraint, and purging was measured through the Eating Disorder Examination Questionnaire; Body avoidance was measured by the Body Image Avoidance Questionnaire; Body Checking was measured by the Body Checking Questionnaire; Dichotomous thinking was measured by the Dichotomous Thinking in Eating Disorder "eat subscale"; binge eating was measured by the Binge Eating Scale. Measures are described below. 1= Clinical norms obtained from Welch, Birgegård, Parling, and Ghaderi (2011); 2 = M SD reported in a clinical sample by Reas et al (2002) ; 3= M SD reported in a clinical sample by Rosen et al (1999); 4= M SD reported in a clinical sample by Byrne et al (2008); 5= M SD reported in a clinical sample by Telch, Agras, and Linehan (2001); 6= M SD reported in a clinical sample by Lampard et al (2011).

Table 8.2

Means, Standard Deviations, and Correlations between Model Variables

Construct/Variable	1	2	3	4	5	6	7
1. Shape and weight concerns (EDE-Q items 22, 23, 24, 25, 26, 27, 28)							
2. Body checking (BCQ items 5, 8, 12, 13, 16, 17, 22)	.685**						
3. Body avoidance (BIA items 1, 2, 4, 15, 17)	.713**	.556**					
4. Dietary restraint (EDE-Q items 1, 3, 4)	.489**	.448**	.459**				
5. Dichotomous thinking (DTES items 1, 4, 6, 8)	.667**	.581**	.596**	.503**			
6. Binge eating (BES items 2, 4, 5, 8)	.486**	.420**	.384**	.225**	.515**		
7. Purging	.305**	.334**	.303**	.467**	.343**	.120*	
Mean	2.91	19.70	8.67	2.44	2.36	3.62	1.61
Standard Deviation	1.73	6.79	5.07	2.05	.92	2.32	2.69

Note: ** = p<.001; * p<.05; BCQ= body checking questionnaire; BIA= Body image avoidance questionnaire; EDE=Q= Eating disorder examining questionnaire; DTES= Dichotomous thinking in eating disorders scale; purging score indicates the average number of purge episodes over the 28 days.

Structural Model of the Original Cognitive-Behavioural Model

The structural model tested the relationships between latent variables outlined in the original cognitive-behavioural model (Figure 3). The structural model provided a marginally acceptable fit, χ^2 (87) = 374.652, p<.001 (CFI=.91, TFI=.90, RMSEA=.09, SRMR=.10). All paths were statistically significant and in the expected direction. The model accounted for 32.6%, 18.4%, and 6.2% of the variance in restraint, binge eating, and purging, respectively (See Figure 8.3).

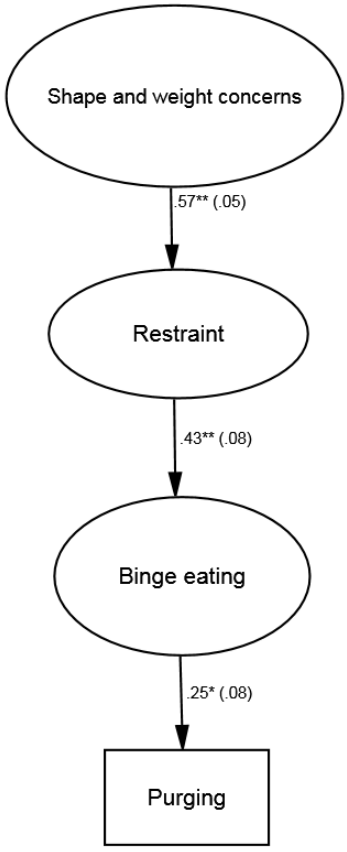


Figure 8.3: The Original Cognitive-Behavioural Structural Model Observed in the Current Study

Note* Standardised coefficients and standard errors are presented; **= p<.001, *= p<.01

Expanded Structural Model

The expanded structural model provided an acceptable to poor fit, χ^2 (425) =7108.60, p<.001 (CFI=.90, TFI=.89, RMSEA=.06, SRMR =.10). All paths were significant, with the exception of the path from body avoidance to dietary restraint and the path from dietary restraint to binge eating. The trimmed model, where these two paths were deleted, did not improve the structural model, $\Delta\chi^2$ (1) = .629, p>.05. These paths were retained.

An un-estimated path in the structural model with a large MI (27.97) was observed: the pathway from purging to shape and weight concerns. This path was added to improve model fit. This revised model provided a significantly better fit than the original expanded structural model, $\Delta\chi^2$ (1) = 67.08, p<.001 (CFI=.92, TLI=.91, RMSEA =.05, SRMR=.05), and was retained as the final expanded structural model (Figure 8.4). The variance accounted for on endogenous variables were as follows: 58.3% for body checking, 68% for body avoidance, 32.6% for restraint, 27.3% for dichotomous thinking, 51.1% for binge eating, and 5.6% for purging. Significant paths were observed between shape and weight concerns and body checking; shape and weight concerns and dietary restraint; body checking and dietary restraint; dietary restraint and dichotomous thinking; dichotomous thinking and binge eating; binge eating and purging; and purging and shape and weight concerns.

Three indirect effects were estimated. Body checking partially mediated the relationship between shape and weight concerns and dietary restraint (indirect effect: β=.213, 95% CI [.105, .322]). Body avoidance did not mediate this relationship (indirect effect: β=.012, 95% CI [-.126, .150]). Dichotomous thinking fully mediated the relationship between dietary restraint and binge eating (indirect effect: β=.474, 95% CI [.375, .573]).

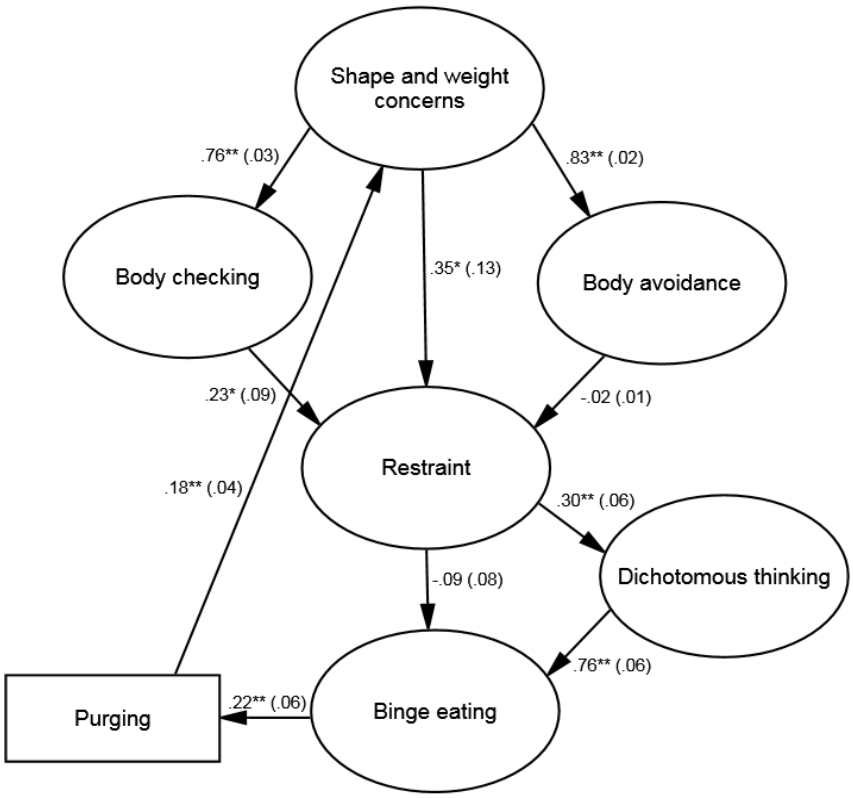


Figure 8.4: The Final and Expanded Structural Model.

Note* Standardised coefficients and standard errors are presented; **= p<.001, *= p<.01

Discussion

This study aimed to test the original cognitive-behavioural model of BN, and to also test an expanded cognitive-behavioural model in which the mediating roles of body checking, body avoidance, and dichotomous thinking were tested. Both the original and expanded model provided an acceptable fit to the data; however, only when a path from purging to weight and shape concerns was added did the expanded model provide a significantly better model fit. The final expanded model also accounted for three times the amount of variance in bulimic symptoms than the original model.

All of the hypothesised paths in the original cognitive-behavioural model were supported. Shape and weight concerns correlated with dietary restraint, dietary restraint correlated with binge eating, and binge eating correlated with purging. The relationship between dietary restraint and binge eating has received mixed findings, which has led some to propose that restraint may have less of a role in the maintenance of binge eating than originally thought (Lampard et al., 2013). Such inconsistencies may be a result of the choice of binge eating measurement. Lampard and colleagues argued that dietary restraint is strongly related to binge eating only when binge eating is measured with scales that assess all underlying behavioural, cognitive and affective components of binge eating, rather than merely how frequently this behaviour occurs. In the current study, binge eating was assessed via the BES, which assesses the severity of all components of binge eating. These findings support the claim that dietary restraint might actually be associated with binge eating severity and its underlying cognitive and affective components, rather than how frequently it occurs.

In the expanded model, after inclusion of the proposed mediators, and after adding a path from purging to weight and shape concerns, most of the relationships in the original model, with the exception of the path from dietary restraint to binge eating, remained

statistically significant. Shape and weight concerns were associated with both body checking and avoidance, and dietary restraint was associated with body checking, but not body avoidance. The relationship between shape and weight concerns and dietary restraint was partially mediated by body checking (but not by avoidance). This supports suggestions that a reason why individuals with elevated levels of shape and weight concerns restrain their eating is because of the repeated checking and scrutinising of their body (Fairburn, 2013). These findings demonstrate how repeated checking could be a maladaptive behaviour that should be sufficiently addressed during prevention and intervention programs (Fairburn et al., 1993b).

By contrast, body avoidance did not mediate this relationship. This finding suggests that avoidance may play less of a role in the maintenance of specific disordered eating symptoms such as dietary restraint. However, the fact that shape and weight concerns was associated with body avoidance suggests that body avoidance might serve to maintain shape and weight concerns. Although theory suggests that avoidant behaviours occur as a result of extreme shape and weight concerns (Fairburn et al., 2003a), a bidirectional relationship between these variables has also been proposed, where shape and weight concerns encourage avoidance which in turn maintains further concerns (Shafran et al., 2004). Since our data are cross-sectional, such feedback maintenance loops could not be tested. This is an interesting focus for future research.

Once dichotomous thinking was included in the expanded model, the relationship between dietary restraint and binge eating became non-significant. Dichotomous thinking fully mediated the dietary restraint-binge eating relationship. This is the first study to demonstrate that dichotomous thinking explains this relationship, further providing empirical support for the cognitive-behavioural model, which proposes that dichotomous thinking is a key mechanism explaining how and why dietary restraint is related to binge eating (Fairburn et al., 1993a). This model proposes that an individual with elevated levels of dietary restraint

is primed to think in an all-or-none fashion following dietary transgressions. This transgression is interpreted as a catastrophic failure ("my diet is ruined"), leading the person to temporarily "give up" their restraint and engage in binge eating (Fairburn et al., 2003a). The present findings highlight the importance of ensuring that dichotomous thinking is directly targeted during CBT for binge eating-related disorders, as this as this may indirectly reduce binge eating. Indeed, several cognitive-behavioural treatment manuals already place emphasis on targeting a dichotomous thinking style, through the use of cognitive restructuring and exposure-based techniques (Pike, Walsh, Vitousek, Wilson, & Bauer, 2003; Touyz, Polivy, & Hay, 2008). These findings also suggest that incorporating such strategies early rather than later during intervention and prevention programs might be beneficial, as this might prompt a rapid response in binge eating reductions, which was shown to be a robust predictor of successful outcomes (Linardon et al., 2016a; Linardon et al., 2016b).

Certain limitations need to be considered. The cross-sectional nature of this study prevents us from drawing conclusions about the causal direction of pathways. Although results provide the statistical evidence that is necessary if a causal relationship existed, this is not sufficient evidence to conclude that causality is present. Experimental and longitudinal research is required to establish that a temporal relationship exists. Furthermore, future research should aim to understand whether CBT works because of the reasons specified by this model or because of common factors (e.g., therapeutic alliance). Testing how CBT-BN works requires future trials to test whether changes in the proposed mechanisms (dichotomous thinking) explain subsequent symptom change (binge eating) over the course of treatment (Murphy et al., 2009). This would provide a more rigorous test of this model. Another limitation was that we relied on self-report data, which means that it might be possible that participants did not accurately report their responses. Future research should assess these constructs using semi-structured interviews (e.g., Eating Disorder Examination

Interview). A final limitation to this study was that participants self-selected to complete the survey. This may have thus led to biases in the sample, such that only individuals with access to the internet or who are interested in their eating patterns provided their responses. Consequently, there is a need to examine whether the present findings are generalizable across participants of various social, cultural, and ethnic identities, particularly those who may not have access to the internet.

The use of a community sample does not allow us to generalise our findings to clinical populations. This model was designed to understand and treat BN (Fairburn et al., 1993a). Since this is the first study to highlight the important roles of dichotomous thinking, body checking, and body avoidance within the context of the cognitive-behavioural model, this research will ideally provide the impetus for further research focused on exploring the mechanisms of CBT. Although generalisations to clinical populations cannot be made, results demonstrate that this model can also account for the variability in behaviours observed in non-clinical groups. Rates of disordered eating in individuals who do not meet diagnostic criteria for an eating disorder significantly outnumbers rates of threshold eating disorders (Hay et al., 2008). Thus, demonstrating that the same maintaining mechanisms apply to clinical and community cases is critical; it demonstrates that intervention programs designed to disrupt these maintaining mechanisms can be potentially applied to individuals who exhibit symptoms of disordered eating, but do not quite meet the diagnostic threshold for an eating disorder. Crucially, this would broaden the dissemination of cognitive-behavioural intervention and prevention programs.

In sum, the relationships specified in the original cognitive-behavioural model of BN were validated in a community sample. This study was the first to test plausible mechanisms underlying the relationships outlined in this model. Body checking and dichotomous thinking contributed to the maintenance of disordered eating symptoms, and explained three times the

amount of variance in bulimic symptoms in the expanded model (when the path from purging to weight and shape concerns was added) than the original model. However, the role of body avoidance requires further elucidation. These findings highlight the importance of ensuring that body checking and dichotomous thinking are addressed during prevention and intervention programs. Future research testing the impact of changes in body checking and dichotomous thinking during treatment is needed to better understand the maintaining role of these variables.

Chapter 9: Methodological Rationale for Article 4

Overview

The present chapter briefly outlines the methodological approach employed to meet the objectives of the fourth study of this project. The objective of this study is to examine the mechanisms of change during a CBTgsh intervention. The mechanisms of action during CBT for eating disorders are not yet known and have not yet been investigated (see Study 2). As a preliminary first-step, the final study will employ a single case experimental design (SCED) to explore the hypothesised CBT change mechanisms, with the intention of providing foundational evidence for future large scale RCTs testing these CBT mechanisms. Specific procedural and methodological details are presented in the study chapter, so this chapter will briefly provide a rationale for the use of the SCED methodology.

Single Case Experimental Design

Single-case experimental designs (SCEDs) are a rigorous, scientific methodology used to define principles of behaviour (Kazdin, 2011). SCEDs have played an important role in developing and evaluating interventions that have been established to modify some facet of human functioning. Some authors (Lundervold & Belwood, 2000) and the Task Force on Promotion and Dissemination of Psychological Procedures (1995) have noted that SCEDs could also qualify as either a stand-alone experiment or as a research method that complements RCTs. The unique aspect of SCEDs is their ability to evaluate interventions experimentally with one or a few individuals. SCEDs also provide an important opportunity to find out whether an intervention works for *individual* people, rather than just comparing the mean of a large group of people (e.g., the experimental group) to the mean of another large group (e.g., the control group) of people (Barlow & Hersen, 1984).

In SCEDS, a single participant serves as his or her own control. Typically, a behaviour or symptom is assessed repeatedly throughout an intervention. The experimenter may then systematically introduce and/or withdraw components of an intervention (a "component analysis"), and then measure the effects of these components on certain behaviours or symptoms in participants (Brossart, Parker, Olson, & Mahadevan, 2006). The components of the intervention include the specific procedural strategies that make up the "treatment package". Each intervention strategy might serve a unique purpose that targets only one aspect of a particular clinical condition (e.g., the "weekly weighing" strategy of CBT is purported to address weight concerns). By tracking behaviours or symptoms repeatedly throughout an intervention for individual participants, SCEDs can demonstrate causal or functional relationships between independent (e.g., weekly weighing strategy) and dependent (e.g., weight concerns) variables (Barlow & Hersen, 1984). Thus, the critical characteristics that make up SCEDs include (a) repeated assessment of a behaviour or symptom, (b) analysing change at the individual, rather than group, level, and (c) manipulation of one or more independent variables and observing its effects on the dependent variable (Lundervold & Belwood, 2000).

The traditional approach to data analysis in SCEDs involves systematic visual comparisons of responding within and between conditions of a study (Kazdin, 2011). Visual inspection of graphed data is considered the most appropriate method of data analysis in SCEDs; visual analysis will show any intervention effect large enough to be considered clinically significant and relevant to practitioners (Barlow & Hersen, 1984). Thus, visual analysis yields lower error rates and is a conservative method for identifying treatment effects (Brossart et al., 2006). Visual analysis involves interpretation of the trend, stability, slope, and overlap. A trend indicates whether a symptom or behaviour is increasing, decreasing, or staying the same (stability). The slope tells us about the *magnitude* of the trend. Overlap is

the extent to which data patterns across phases (e.g., a baseline phase to an intervention phase) overlap with each other. Less overlap provides a stronger case for the functional or causal relationship between the independent and dependant variable (Lundervold & Belwood, 2000).

There are numerous advantages to SCEDs in psychotherapy research. SCEDs can help bridge the scientist-practitioner gap that is so pervasive in modern clinical psychology research; practitioners can engage in SCED research that is clinically relevant to them and that does not require prior knowledge of complex statistical analyses (Lundervold & Belwood, 2000). Further, SCEDs are important for demonstrating the *preliminary* efficacy of an intervention or the *possible* mechanisms of change during an intervention. Once preliminary evidence from SCEDs are gathered, researchers can then decide whether it would be worth trying to replicate these findings using expensive, time consuming, and laboursome group designs (i.e., the RCT). In that regard, SCEDs are suggested to be a "hypothesis-generating" design, in that they can, for example, help future research narrow down the search for causal mechanisms (Barlow & Hersen, 1984). For these reasons, a SCED was employed in the fourth study of this project.

Chapter 10: Exploring mechanisms of change during a cognitive-behavioural guided self-help intervention for disordered eating: A single case experimental design.

This chapter presents the findings from the fourth study of this project that is currently being prepared for publication. Chapter 8 highlighted the dearth of research testing the mechanisms of action during CBT. Thus, as a natural fist-step, this study used a single-case experimental design to explore hypothesized cognitive-behavioural mechanisms of change during a CBT program delivered to women exhibiting subthreshold symptoms of eating disorders.

Abstract

Objective. Although the efficacy of various modalities of cognitive-behavioural therapy (CBT) for treating disordered eating is well documented, little is known about the mechanisms that underpin CBT's effectiveness. This preliminary study utilized a single case experimental design (SCED) to explore several theoretically-relevant mechanisms of change (i.e., regular eating adherence, dietary restraint, shape and weight over-concern) in a CBT guided self-help program. **Method.** Eight women who self-reported binge eating or concerns about weight and shape were randomized to either an eight week CBT (*n*=4) or health at every size (*n*=4) guided self-help program. In addition to pre-post assessments, the purported mechanisms of action were assessed weekly. These weekly data were graphed, and the impact of treatment mechanisms on binge eating behaviour was analysed visually. **Results.** Data from these graphs suggest that an adherence to regular eating principles in the early stages of CBT was associated with a concurrent reduction in dietary restraint and binge eating frequency. There was no evidence that changes in weight and shape concerns were associated with changes in dietary restraint or binge eating. **Discussion.** This preliminary study highlighted how the regular eating strategy in CBT could be an important mechanism that is responsible for the rapid improvements typically observed in CBT. This SCED provides foundational evidence for future large-scale randomized trials testing the purported mechanisms of action during CBT for disordered eating, and highlights the importance of research examining the causal role of regular eating.

There is renewed interest in psychotherapy research that not only asks whether psychotherapy works, but also when, why, and how it works (Laurenceau et al., 2007). Although such questions on the process of therapeutic change are an integral part of psychotherapy development and evaluation, process-based research has long been overshadowed by the need to establish treatment efficacy in randomised controlled trials (RCTs), i.e., whether the aggregated group mean of the outcome differs across treatment conditions (Hayes et al., 2007b). While RCTs are critical for identifying empirically supported therapies, the consequence of being too focused on RCTs is that questions about the therapeutic process (e.g., when and why change occurs) have been neglected. This issue was recently highlighted in a systematic review of cognitive-behavioural therapy (CBT) for eating disorders, where it was demonstrated that almost no attention has been devoted to uncovering the processes of change during CBT (Linardon et al., 2016b).

One benefit of psychotherapy process research is that it allows the trajectory of symptom change to be studied. RCTs typically report group average level symptoms at pre and post-treatment, and a common assumption in psychotherapy is that change is therefore gradual and linear for all individuals (Laurenceau et al., 2007). Psychotherapy, however, is a dynamic process, and the trajectory of change from pre- to post-treatment can take many forms and can differ from person to person (Hayes et al., 2007a). For example, for one person change might occur smoothly over the course of treatment, yet for another person change might occur mostly in the early stages of treatment. These distinct trajectories cannot be captured by pre-post group designs.

Once the trajectory of symptom change is known, mediators and mechanisms of action can be identified (Kazdin, 2007). If, for example, therapeutic change is concentrated mostly during the early stages of treatment, then the next generation of research might begin to test what early therapeutic strategies are operating to cause this change. Thus, researchers

are then better able to inform clinicians on what strategies facilitate this early response, and research can also focus on how to augment or bolster these effective therapeutic strategies (Murphy et al., 2009).

In the context of CBT for eating disorders, many individuals (40-50%) experience a rapid response to treatment, defined by a 65-70% reduction in bulimic symptoms by week four of treatment (Linardon et al., 2016a). This rapid response to CBT implies that powerful therapeutic strategies are operating during the early stages of treatment. However, a recent meta-analysis that aimed to understand predictors of rapid response to CBT for eating disorders concluded that the mechanisms contributing to this early response have not yet been investigated (Linardon et al., 2016a).

The current study therefore used a single-case experimental design (SCED) to identify possible mechanisms of change during a CBT guided self-help (CBTgsh) intervention for subthreshold eating disorders. SCEDs are a useful research tool for evaluating intervention effects, insofar as they serve to complement traditional large sample RCTs (Barlow & Hersen, 1984). In SCEDS, interventions are evaluated experimentally with one or few participants. Participants serve as their own control, as symptoms and behaviours are assessed repeatedly throughout the intervention (Kazdin, 2011). The experimenter then systematically introduces components of an intervention (i.e. the "treatment strategies"), and examines whether a functional relationship exists between the introduction of a treatment component and subsequent changes in symptoms and/or behaviours. An obvious strength of SCEDs is their ability to identify possible mechanisms of change that warrant further investigation in large sample RCTs (Brossart et al., 2006). More specifically, if evidence for a potential mechanism of change comes from one or more SCEDs, future research aiming to examine change mechanisms via an expensive RCT has a starting point and a rationale for selecting candidate mechanisms to test. Thus, SCEDs are an important "hypothesis-generating"

approach for narrowing down the search for causal mechanisms of CBT (Barlow & Hersen, 1984).

The present SCED reports data from participants who were randomized to either CBTgsh or a health at every size guided self-help (HAESgsh) intervention for subthreshold eating disorders. Guided self-help programs are suitable, very effective, and represent a first-line treatment for individuals presenting with less severe and complex eating disorder psychopathology (National Institute of Clinical Excellence, 2017). They also provide an excellent opportunity to study treatment-specific therapeutic mechanisms. Unlike manualized therapist-led CBT, which is typically delivered in a flexible fashion where the content of therapy is tailored toward an individual's specific psychopathology (Fairburn, 2008), guided programs are typically delivered more rigidly and hence more consistently, such that the delivery of guided interventions is more or less the same across participants. Also, since therapists have far less input in guided programs, the confounding effects common mechanisms (e.g., therapeutic alliance) are minimized.

Studying mechanisms of an experimental treatment when a comparison treatment is implemented is also ideal. This is because comparison treatments can help rule out the possibility of what appears to mediate change being simple an effect of other extraneous variables (e.g., regression to the mean) rather than the specific experimental treatment itself (Murphy et al., 2009). The present study used a HAES-based comparison intervention, which is also appropriate since HAES principles, at times, oppose CBT principles. For instance, whereas CBT promotes a pattern of regular eating and weekly weight checking, HAES promotes a pattern of intuitive eating and weight avoidance. Therefore, using HAES as a comparator, the overall objective of this SCED is to explore several CBT-specific mechanisms of change during CBTgsh for subthreshold eating disorders.

Method

Participants

All participants were recruited through online advertisements distributed throughout the community and through local GPs. Eight Caucasian women participated in this study. Ages ranged between 41 and 57 years (*M*= 48.75, *SD*= 6.36), and participants BMI ranged from 22.9 to 43.9 (*M*= 32.42, *SD*= 6.94). Table 10.1 presents the demographic and clinical characteristics of each participant. To be eligible to participate in this RCT, participants had to be over the age of 18 years, and exhibit subthreshold symptoms of disordered eating (i.e., objective or subjective binge eating less than once per week over the past three months) and/or attitudinal disturbances in eating, weight and shape. Attitudinal disturbances was defined as an Eating Disorder Examination Questionnaire (EDE-Q) global score greater than Australian norms (1.52) for women (Mond, Hay, Rodgers, & Owen, 2006). Exclusion criteria were as follows: receiving current psychologist treatment for disordered eating; pregnancy; comorbid psychiatric disorder, and meeting diagnostic criteria for BED, bulimia nervosa (BN), or anorexia nervosa (AN). Note that we did not limit our inclusion criteria to overweight/obese females for two reasons. First, previous research evaluating non-dieting, health at every size interventions has demonstrated that these particular treatments can effectively improve eating behaviour and body image in non-overweight women (Cole & Horacek, 2010; Steinhardt, Bezner & Adams, 1999). This is also the case for CBT guided self-help (e.g., Carter & Fairburn, 1998). Second, practical restraints (e.g., recruitment difficulties) prevented us from only recruiting and sampling a subset of the population (e.g., only people over a certain BMI with disordered eating symptoms). For these reasons, we decided to include both overweight and underweight women.

Table 10.1

Demographic and clinical characteristics of the eight participants

Condition	Case	Sex	Age	Height	Weight	BMI	EDE-Q	CIA	Binge eating
CBT									
	1	Female	44	172cm	110kg	37.2	4.20	31	14
	2	Female	48	155cm	55kg	22.9	1.41	8	0
	3	Female	57	177cm	73kg	23.3	4.80	20	28
	4	Female	53	163cm	87kg	32.7	2.95	17	14
HAES									
	1	Female	42	163cm	98kg	33.1	3.74	16	3
	2	Female	57	169cm	85kg	31.2	1.88	18	0
	3	Female	48	167cm	98kg	35.1	1.31	6	5
	4	Female	41	164cm	118kg	43.9	4.09	33	5

Note: EDE-Q = Eating disorder examination questionnaire global score; CIA= clinical impairment assessment; binge eating= number of binge eating episodes.

Interventions

Both guided interventions were delivered in eight, 50 minute sessions, preceded by one 90 minute assessment session. Therapists, who were masters-level psychology students, were instructed to pay consistent focus to the treatment manual while also attending to client motivation. The guided nature of these interventions meant that therapists primarily focused on (a) maintaining and improving motivation; (b) correcting any misunderstanding of the information provided; (c) enhancing problem solving abilities (i.e., barriers to completing prescribed homework tasks); and (d) reinforcing the successful implementation of key strategies outlined in the program. Guided sessions were video recorded for supervision purposes. Therapists received weekly individual supervision from a clinical psychologist with expertise in the assessment and treatment of eating disorders.

Cognitive-behavioural guided self-help. The "Overcoming Binge Eating" book (Fairburn, 2013) was used as the CBTgsh intervention. This intervention targets the mechanisms that, according to the cognitive model of eating disorders, are hypothesized to maintain eating disorder psychopathology. These maintaining mechanisms include (a) an over-concern with weight and shape; (b) shape and weight checking and avoidance; and (c) dietary restraint and restriction. This book consists of two sections. The first section is psychoeducational, where eight short chapters are devoted to describing binge eating, the factors that maintain binge eating, and the rationale for the self-help program. The second section contains the self-help program, which consists of six steps that address how to change binge eating. Participants were instructed to read and implement the strategies in the relevant section prior to discussing them with the therapist the following week. See Table 10.1 for a detailed overview of the session plan.

Health at Every Size guided self-help: The "If Not Dieting, Then What?" book (Kausman, 2004) was used as the HAESgsh intervention. A HAES-based intervention served

as the comparison condition because the strategies implemented in this approach contrast the strategies implemented in CBT. For example, whereas CBT prescribes the "weekly weighing" technique, HAES interventions assert that weighing serves no useful function and should therefore be avoided. Similarly, whereas CBT strongly encourages clients to eat at regular intervals and initially ignore any hunger and satiety cues, a core element of HAES is to teach clients to eat intuitively and in accordance to physiological cues rather than following externally imposed eating rules. The If Not Dieting book contains psychoeducational material on physical, psychological, and social effects of dieting, and a series of self-help strategies are also provided throughout the book on how to address "yo-yo dieting", "non-hungry eating", and body image concerns. Participants were instructed to read the relevant section and implement the strategies prior to discussing them with the therapist in the following session. See Table 10.1 for a detailed overview of the session plan.

Measures: Baseline and Post-Intervention

The Eating Disorder Examination Questionnaire (EDE-Q). The EDE-Q (Fairburn & Beglin, 1994) is a 28-item self-report measure that assesses the severity of disordered eating behaviours and attitudes over the past 28 days. A global score is computed from the average of the four subscales: dietary restraint, weight concerns, shape concerns, and eating concerns. The global score ranges from zero to six, where higher scores indicate more severe disordered eating attitudes. The global score was used as a primary outcome. Several items assess behavioural symptoms of disordered eating, and for the current study, the frequency of objective binge eating (i.e., eating a large amount of food given the circumstances and accompanied by a sense of loss of control) was used as a primary outcome. Acceptable internal consistency, test re-test reliability, and construct validity of the EDE-Q has been established (Berg, Peterson, Frazier, & Crow, 2012).

The Clinical Impairment Assessment (CIA). The CIA (Bohn et al., 2008) is a 16-item self-report measure that assesses psychosocial impairment secondary to disordered eating symptoms over the past 28 days. Items (e.g., "to what extent have your eating habits stopped you from going out with others") are rated along a four-point scale, ranging from 0 (not at all) to four (a lot), and are summed to produce a total score. Higher scores indicate greater levels of psychosocial impairment. CIA total scores were used as a primary outcome. Acceptable internal consistency, test re-test reliability, and discriminant validity of the CIA has been established (Bohn et al., 2008).

Measures: Within-Treatment Assessment

Several variables were also assessed weekly at the end of each guided session. Self-reported *binge eating frequency* (i.e., "how many times have you binged, i.e., felt out of control of your eating, and eaten far more than a personal normally would in one go) and *regular eating days* (i.e., "how many days out of the past seven have you engaged in regular eating patterns, i.e., consumed 3 meals and 2-3 snacks") were assessed over the past 7 days. *Shape and weight concerns* were assessed via the ED-15, which is a brief measure of the EDE-Q that assesses disordered eating symptoms over the past seven days (Tatham et al., 2015). The combined shape and weight concerns subscale consists of six items that are rated along a seven point scale, ranging from zero (not at all) to six (all the time), where higher scores indicate greater concerns about weight and shape. A single item, which was placed on a 100mm visual analogue scale, was extracted from the EDE-Q to assess *dietary restraint* ("have you been deliberately trying to limit the amount of food you eat in order to influence your weight and shape")[5].

[5] All within-treatment variables were standardized, such that each variable was reported on a scale ranging from 0 to 10.

Table 10.2
Session Outline and Structure for the Two Guided Self-Help Interventions

Session	CBT content	HAES content
1	Getting ready: Review of psychoeducation material. Learn about maintaining factors. Enhancing motivation. Commence self-monitoring and weekly weighing.	Diets don't work - Choose the right goals: Review of past diet tactics and their success. Eliminate any self-weighing.
2	Starting well: Review self-monitoring. Discuss the rationale and importance of regular eating. Identify barriers to engaging in regular eating, and come up with strategies for adhering to regular eating.	Non-hungry eating: Learn about non-hungry eating. How to recognize it using self-monitoring records.
3	Avoiding binges: Review progress. Devise list of alternative activities for when urges to binge eat are present. Implement distraction-based tasks.	Three concepts that can change your life: Learn about three concepts related to food and eating: (1) there is no good or bad foods; (2) tell yourself 'I can have it if I want, but do I feel like it'; (3) Start to eat slowly and start listening to hunger/satiety signals.
4	Problem solving: Discuss effective problem solving. How to implement a 6-step problem solving strategy for when binge eating urges appear.	What is normal? Learn about the balance between nutrition and intuition
5	Taking stock: Review session. Focus on barriers to change. Discuss progress and continue with strategies implemented.	Eating with awareness: Discussion on intuitive eating. Provide a rationale for it, assist with implementing it, and practice eating intuitively.
6	Dieting module: Address food-based based rules using exposure-related techniques.	Nurturing: The importance of self-care. How to effectively self-care – devise a list of activities that demonstrate self-care.
7	Body image module: Address over-evaluation of shape and weight; increase the importance of other facets of life.	Body image + being active: Promoting positive body image. The importance of healthy, enjoyable, and sustainable exercise
8	Ending well: Review progress. Relapse prevention plan.	Going off focus: Review progress. Relapse prevention plan

Procedure

This study received approval from the Australian Catholic University Human Research Ethics Office. Respondents to community advertisements contacted the researcher via email and expressed their interest to participate. The researcher then contacted respondents via the telephone to (a) determine the absence of exclusion criteria, and (b) outline the nature of the intervention programs. After this, an initial assessment was scheduled with Masters-level Provisional Psychologists. During this session, the Eating Disorder Examination and the Mini-International Neuropsychiatric Interview were administered to determine full eligibility for the study. Written consent was obtained prior to these interviews. Eligible participants were then randomized to either intervention, using a randomization sequence of permuted blocks of 50. The randomization list was prepared with a table of random numbers and transferred to a numbered sequence of opaque sealed envelopes by an individual not involved in the study. Therapists were not blind to baseline assessment information. Once participants were randomized, they completed the baseline questionnaires. Participants were not blind to the treatment condition they were allocated to. Participants then scheduled eight weekly sessions with their allocated therapist. Participants completed the weekly treatment questionnaire at the end of each guided session. At the end of the last session, participants then completed the post-treatment assessment.

Data Analysis

Participants' pre and post-treatment scores on each of the three outcomes (i.e., EDE-Q global, binge frequency, and CIA scores) were calculated to determine whether or not they improved from baseline to post-treatment. A reliable change index (RCI) was also calculated to determine whether the magnitude of change from pre to post-treatment for each participant was statistically reliable, and not just due to measurement error. The RCI was calculated by

dividing the difference between the pre and post-treatment test score by the standard error of measurement[6]. A RCI score greater than 1.96 indicates that the change was not just due to measurement error.

For within-treatment data, analyses were conducted visually and in accordance to established guidelines for SCEDs (Barlow & Hersen, 1984). Visual inspection of graphed data is the recommended method of data analysis in SCEDs, as visual analysis will elucidate any intervention effects large enough to be considered clinically significant and relevant to practitioners (Kazdin, 2011). Visual analysis is therefore said to yield low error rates and is a conservative method for identifying potential mechanisms of action (Kazdin, 2011). Visual analysis involves interpretation of the trend (whether behaviours or symptoms are increasing or decreasing), stability (whether behaviours or symptoms are staying the same), and slope (the magnitude of such trends) of the graphed data.

[6] To calculate the SEM, the standard deviation of relevant norms and the test re-test reliability is required. These values were obtained from published reports

Results

Pre-Post Intervention Effects

The scores at pre and post-treatment for each outcome for all eight participants were calculated. These scores are presented in Table 10.2 and discussed below.

EDE-Q Global Scores. EDE-Q global scores decreased at post-treatment for all participants. Two participants from each condition (CBT1, CBT3, HAES1, and HAES4) made reliable change improvements. Of note, those that did not make reliable improvements had low EDE-Q scores at baseline.

Binge Eating. Reductions in binge eating were also observed across participants. CBT3 went from binge eating daily to binge eating abstinence. CBT1 and CBT4 reduced their binge eating by 79% and 58%, respectively. These were all reliable changes. Binge eating behaviour increased in three participants, two of which did not report any binge eating at baseline (CBT2, HAES2), and one reported that she binged infrequently (HAES1).

CIA Scores. Reductions in CIA scores were observed across most participants. CBT3 made large improvements in CIA scores, whereas CBT1 and CBT2 made small improvements. CBT4's CIA score did not change. In the HAES condition, all four participants made large improvements in CIA scores.

Table 10:3
Pre and Post-treatment Scores on Each Outcome for all Eight Participants

		EDE-Q			Binge eating			CIA		
Treatment	Case	Pre	Post	RCI	Pre	Post	RCI	Pre	Post	RCI
CBTgsh										
	1	4.20	0.98	**3.03**	14	3	**3.37**	31	28	**1.94**
	2	1.41	1.18	0.21	0	4	-1.22	8	6	1.29
	3	4.80	0.74	**3.83**	28	0	**8.58**	20	5	**9.74**
	4	2.95	1.21	1.64	14	6	**2.45**	17	17	0
HAESgsh										
	1	3.74	1.46	**2.15**	3	4	-0.31	16	5	**7.14**
	2	1.88	1.56	0.30	0	2	-0.61	18	7	**7.14**
	3	1.31	0.53	0.95	5	0	1.53	6	3	**1.95**
	4	4.09	1.72	**2.23**	5	4	0.30	33	6	**17.53**

Note: CBT= cognitive-behavioural therapy; HAES= health at every size; EDE-Q eating disorder examination global score; CIA= clinical impairment assessment; RCI= reliable change index. Bolded = participant met criteria for reliable change.

Within-Treatment Change

Figures 10.1 and 10.2 present the weekly graphed data on all variables assessed during treatment for all participants. The overall pattern of results demonstrates that each participants made improvements in all or most of the variables assessed within treatment. However, the trajectory of change in each variable differed across participants.

The Shape of Change. Three participants (CBT3, CBT4, HAES2) demonstrated a rapid response trajectory of change (i.e., at least a 65% reduction in binge eating by week four). This rapid response was observed in three participants. After this rapid response, improvements in binge eating were generally sustained throughout the course of treatment. Two of these rapid responders (CBT3 and HAES3) had the best outcomes at post-treatment. Many individuals (CBT2, HAES1, HAES3, and HAES4) reported very few binge eating episodes at baseline (≤ 2 episodes); therefore, a rapid response was not possible in these participants.

Mechanisms of Action. The data were also inspected visually to identify whether any mechanisms of change could be observed.

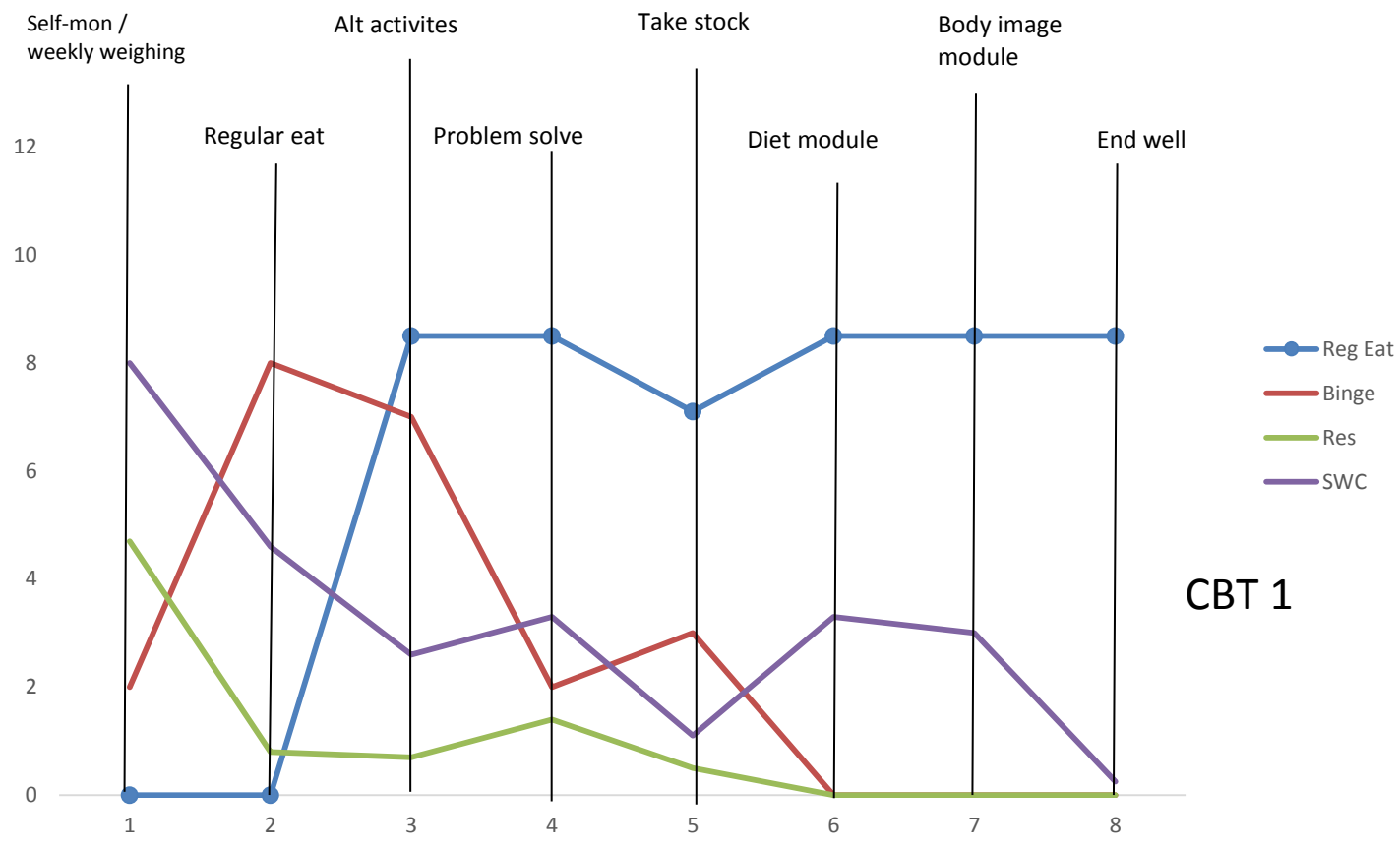
Regular eating. Regular eating frequency showed clear and consistent trends, and appeared to be associated with concurrent reductions in dietary restraint and binge eating. After regular eating was introduced in week two of CBTgsh, CBT1, CBT3, and CBT4 increased their regular adherence to "every day". This reported adherence appeared to be associated with the large and rapid response to treatment.

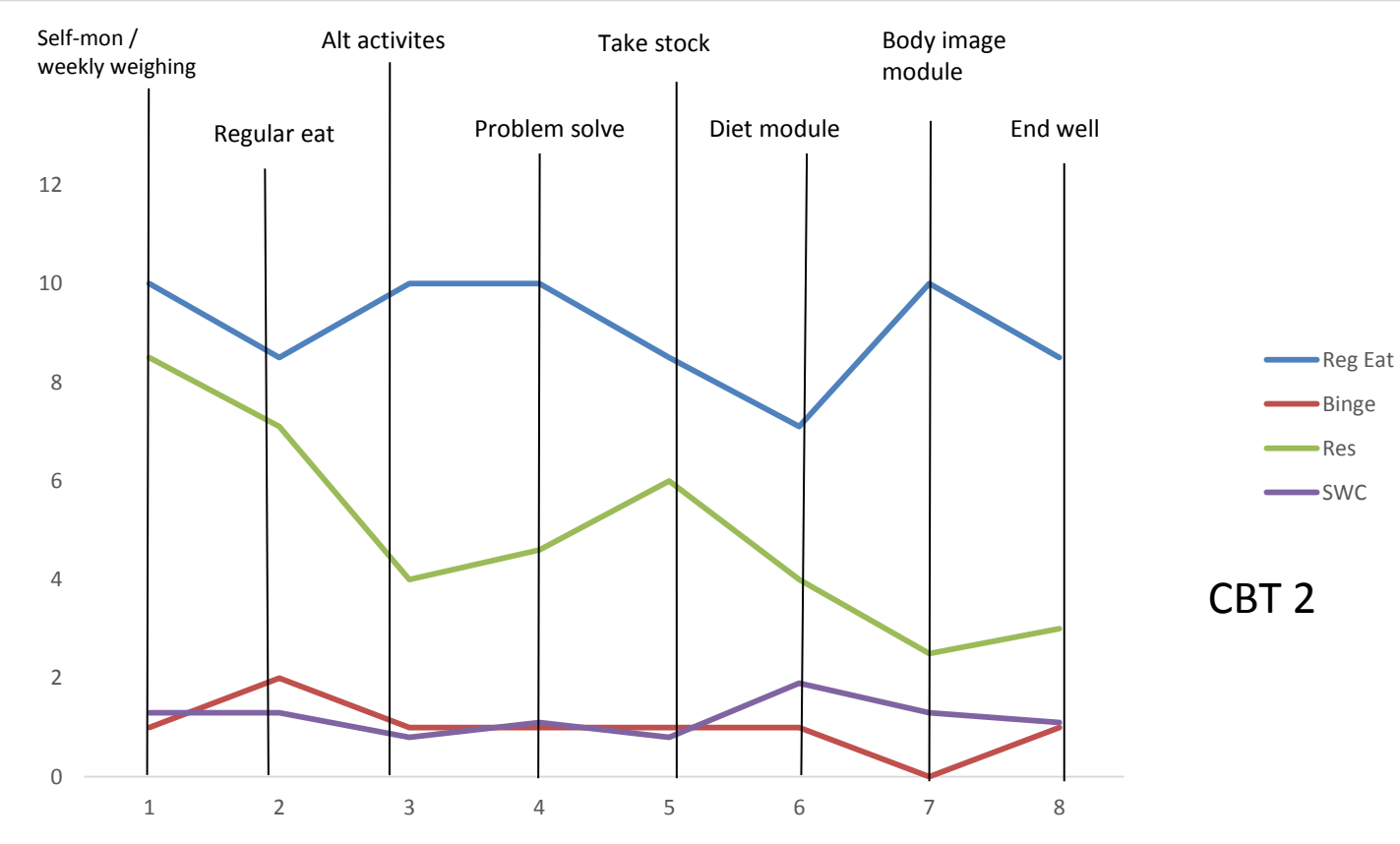
In contrast, all HAES participants reported high levels of regular eating days at baseline. A similar association between regular eating and binge eating was still observed. HAES1 and HAES3 reported regular eating daily throughout the eight weeks. Both participants also reported consistently low levels of binge eating throughout treatment. Also, HAES4 was eating regularly until week four. During week four, her regular eating adherence dropped considerably. This drop corresponded with a binge eating "spike". After she began eating regularly during week five, her self-reported binge eating behaviour ceased.

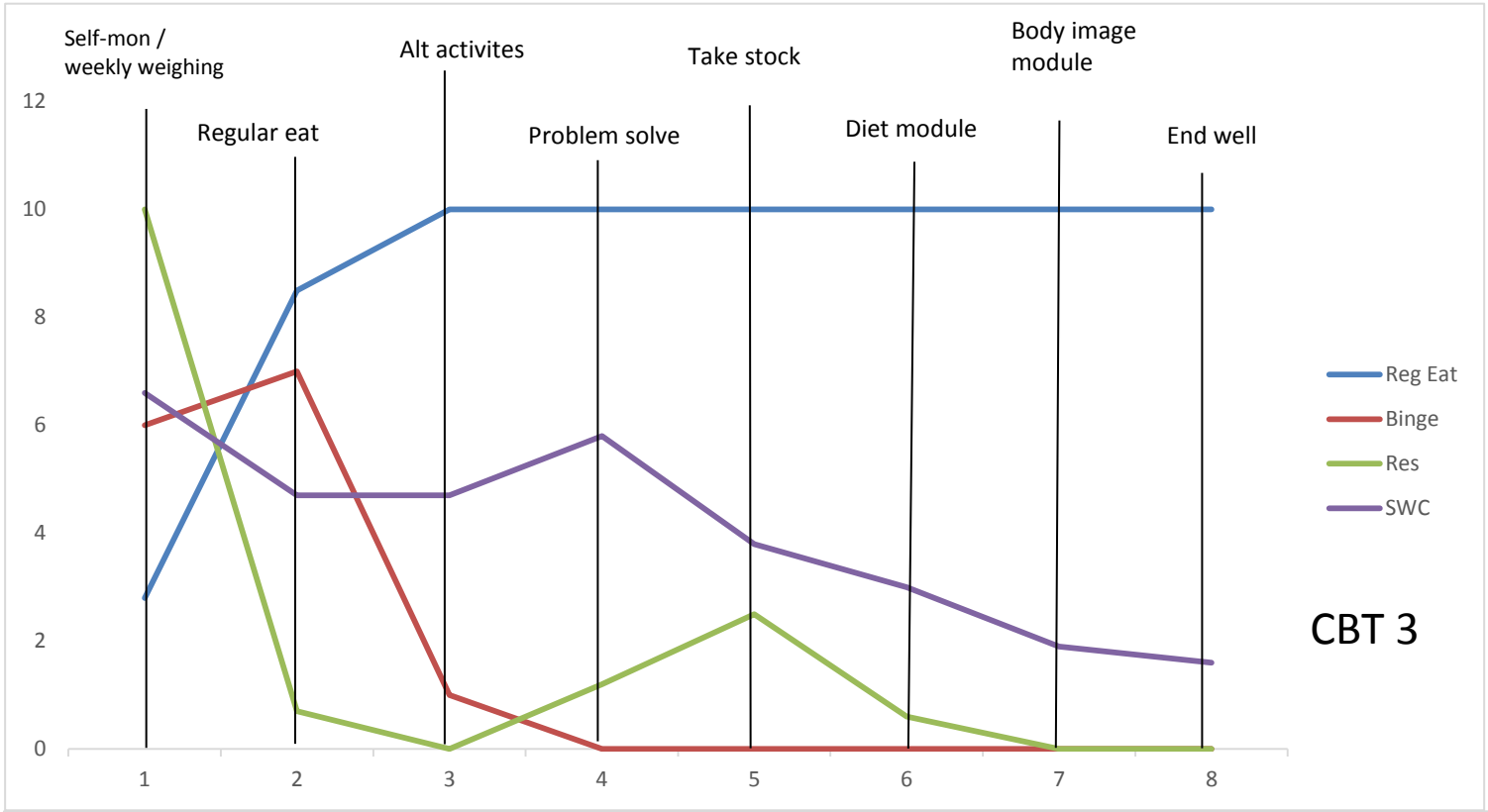
Weight checking. Weight checking was directly manipulated in the first week of both interventions. In particular, whereas CBT instituted weekly in-session weighing, HAES prescribed complete weight avoidance. This enabled us to observe the relationship between weighing checking and weight concerns. The graphs suggest that while each participant reduced their total scores on the weight and shape concern subscale from week one to week eight, these changes tended to be more gradual and linear for most participants. Faster reductions in weight concerns appeared to occur for those with higher scores at baseline (see CBT1 and CBT4).

Other treatment strategies*:* A variety of additional treatment strategies were implemented during the mid to later stages of CBT. These strategies included "alternative activities" (week3), problem solving (week 4), the dietary rules procedure (week 6), and shape checking/avoidance (week 7) techniques. Based on the graphed data, we found little evidence to suggest that the purported maintaining factors (i.e., dietary restraint, shape and

weight concerns) and binge eating behaviours changed in response to these treatment strategies. This was because change either occurred rapidly for some participants or smoothly for others.


Self-mon / weekly weighing
Regular eat
Alt activites
Problem solve
Take stock
Diet module
Body image module
End well
12
10
8
6
4
2
0
1
2
3
4
5
6
7
8
Reg Eat
Binge
Res
SWC
CBT 1


Self-mon / weekly weighing
Regular eat
Alt activites
Problem solve
Take stock
Diet module
Body image module
End well
12
10
8
6
4
2
0
1
2
3
4
5
6
7
8
Reg Eat
Binge
Res
SWC
CBT 2



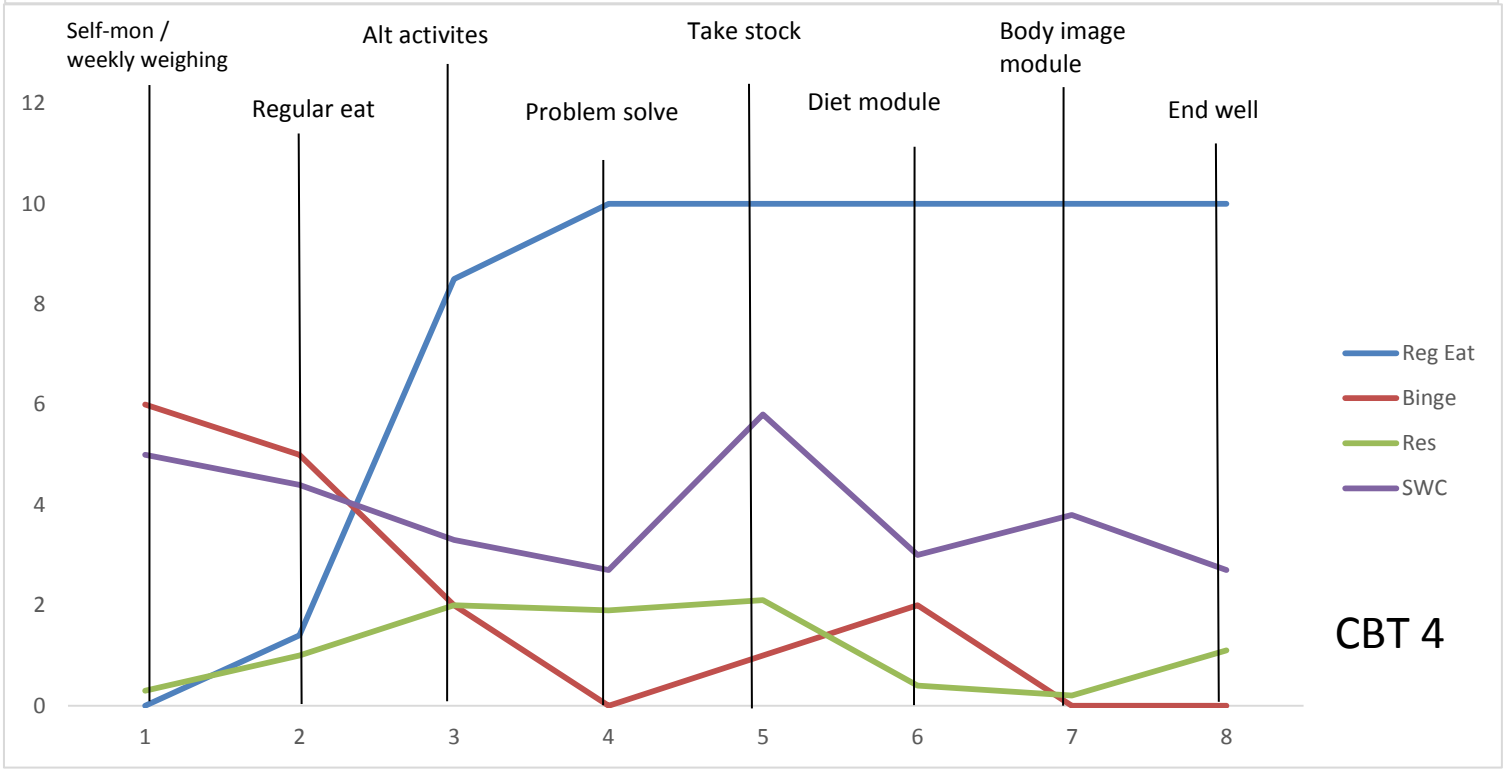
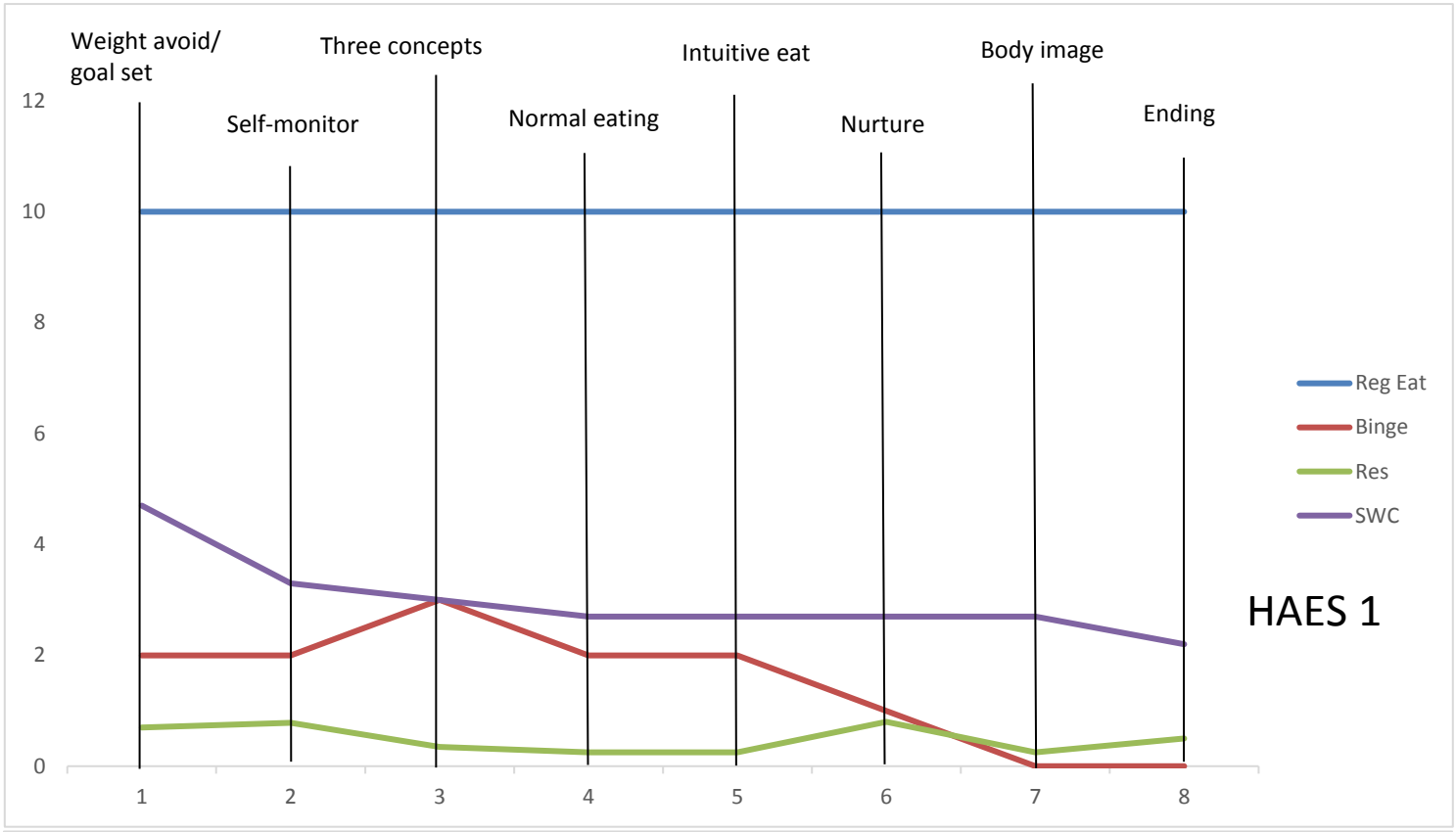
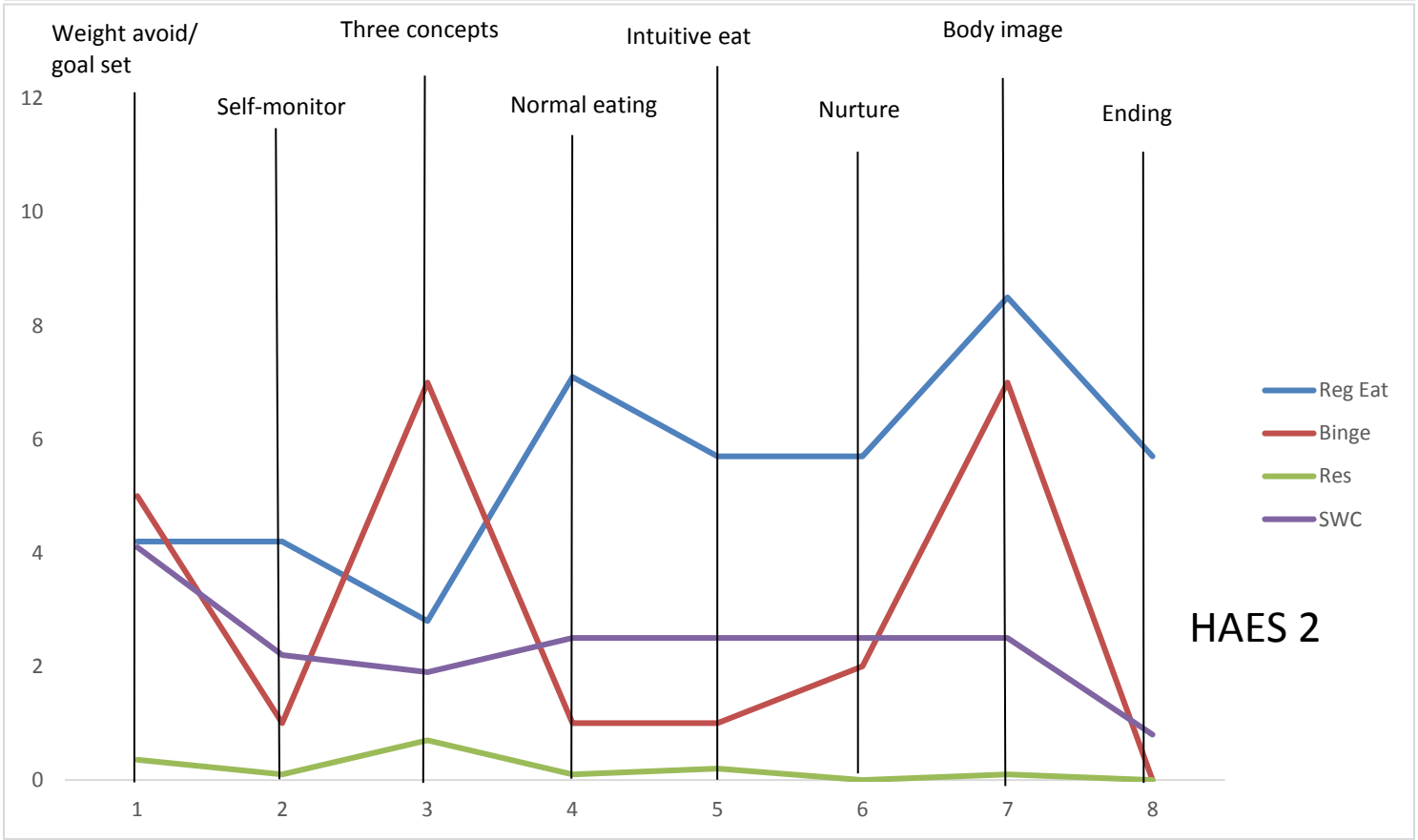


Figure 10.1: Graphed data on treatment mechanisms and symptom change for CBTgsh participants
Note: Vertical axis indicates scaled score and horizontal axis indicates number of weeks.


Weight avoid/ goal set
Self-monitor
Three concepts
Normal eating
Intuitive eat
Nurture
Body image
Ending
12
10
8
6
4
2
0
1
2
3
4
5
6
7
8
Reg Eat
Binge
Res
SWC
HAES 1


Weight avoid/ goal set
Self-monitor
Three concepts
Normal eating
Intuitive eat
Nurture
Body image
Ending
12
10
8
6
4
2
0
1
2
3
4
5
6
7
8
Reg Eat
Binge
Res
SWC
HAES 2

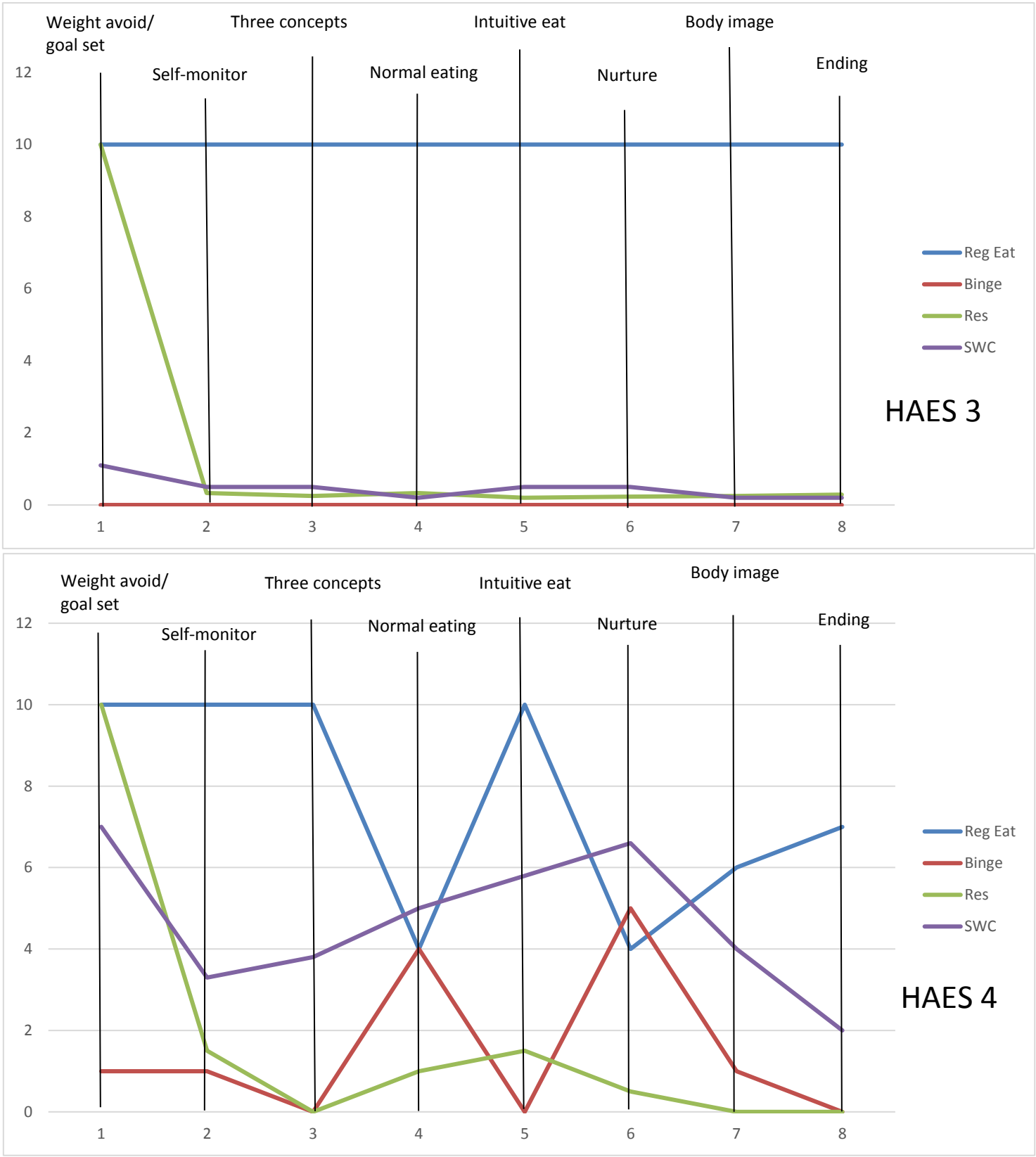


Figure 10.2. Graphed data on treatment mechanisms and symptom change for HAES gsh participants
Note: Vertical axis indicates scaled score and horizontal axis indicates number of weeks.

Discussion

This paper employed a SCED to study the mechanisms of change during CBTgsh for women exhibiting subthreshold symptoms of eating disorders. Although consistent improvements from pre to post-treatment were made across all participants, the trajectory of change varied markedly. In particular, whereas some participants made gradual and linear improvements throughout treatment, others achieved a rapid response to treatment (defined as a 65% reduction in binge eating by week four). These different patterns of symptom change support the idea that change in psychotherapy is discontinuous and punctuated by marked shifts in symptoms for some individuals (Hayes et al., 2007b). Importantly, those who experienced a rapid response had the best outcomes at post-treatment, which is consistent with recent reviews highlighting the prognostic importance of early change (Linardon et al., 2016a; Vall & Wade, 2015) .

Recent calls have been made to identify the mechanisms facilitating this rapid response (Linardon et al., 2016a). Our findings provide preliminary support for the hypothesis that an adherence to regular eating principles could be one important factor responsible for early change in dietary restraint and binge eating. Regular eating was introduced in week two of CBT, and an increased adherence to regular eating was associated with decreased dietary restraint and binge eating in most CBT participants. In addition, a *reduction* in the number of days where participants adhered to regular eating was associated with binge eating "spikes", while those who ate regularly to begin with tended to not report any binge eating behaviour. This relationship, however, occurred across both conditions. Although HAES interventions emphasise intuitive eating over regular eating, it might be that individuals who start learning to eat based on their hunger and satiety cues also eat more regularly than those who attempt to restrain or delay their eating. Overall, these preliminary findings offer some support to the cognitive-behavioural model that underpins cognitive-

behavioural treatments, which proposes that introducing a pattern of regular eating should disrupt the key mechanisms (i.e., dietary restraint and restriction) that maintain binge eating (Fairburn, 2008).

A unique aspect of this study was that we were able to manipulate weight checking. Whereas participants randomized to CBT were weighed weekly by the therapist, those randomized to HAES were instructed to avoid self-weighing. The rationale for the CBT "weekly weighing" strategy is that regular supervised weighing disrupts extreme weight checking or weight avoidance, and addresses the unhelpful cognitions associated with weighing. This is considered beneficial, because obsessive weight checking or weight avoidance is hypothesized to maintain and amplify extreme concerns about weight and shape and dietary restraint (Fairburn, 2008). In addition to targeting these problematic behaviours, Fairburn (2013) speculates that because the therapist teaches the client to focus on long-term weight changes (≥ 4 weeks), and because weight remains relatively stable during treatment, reduction in weight concern should occur during the mid-later stages of treatment. Our data somewhat support this hypothesis. Specifically, rather than observing rapid changes in weight and shape concerns when weight checking was initiated, we instead observed gradual and linear changes in weight and shape concerns for CBT participants (CBT1, CBT3, CBT4) over the course of treatment. However, as this also occurred for two HAES participants (HAES1 and HAES3), it is possible that other mechanisms (possibly common therapeutic mechanisms, such as clinician warmth or regular clinician contact) are responsible for this effect.

Findings from the current study must be interpreted in light of its limitations. First, the sample was relatively homogenous (Caucasian adult women), which limits the generalizability of the current findings. Additionally, despite the fact that participants were randomly allocated to each condition, baseline differences (i.e., binge eating) were apparent

between the two conditions due to the very small sample size. Second, although participants were encouraged to refer back to their self-monitoring forms when filling out their weekly questionnaire, the variables assessed within-treatment relied on participant's recall. This has the potential to introduce social desirability biases, i.e., that participants were responding in a way that made them appear to be adhering to the program. Future research should consider more objective measures of key variables. Third, our interpretations of the data were based entirely on visual analysis. Visual inspection is the recommended method of data analysis in SCEDs (Kazdin, 2011). However, given that this type of analysis relies on subjective interpretation, some have questioned it on the grounds that the interpretations made can be prone to researcher expectations and biases (Brossart et al., 2006). Replicating this using statistical methods in a larger sample (e.g., latent growth curve modelling) is important, and efforts to do so are indeed underway (as reported by Fairburn et al., 2015). Fourth, in SCEDs it is recommended that participants are repeatedly assessed for a period of time (e.g., a few weeks) *prior* to implementing any treatment procedures (Kazdin, 2011). This baseline assessment allows for a period of stability in the behaviours or symptoms that are being measured. Thus, any changes in these variables after the baseline period are said to be a result of the treatment strategies implemented. As delaying treatment through an extended baseline period might have had the potential to exacerbate disordered eating symptoms in this sample (hence raising ethical issues), this multiple baseline design was not employed. A multiple baseline design is an important future direction. Finally, no information on the acceptability of either intervention was collected. Given that this was the first study to examine this particular HAES intervention, it would have been valuable to gain insight toward participants experiences of the intervention, what aspects were or were not valuable, and what could be improved in future for larger-scale studies testing its efficacy. This is an important limitation of this research and should be a consideration in future research.

In sum, this preliminary study built on a small body of literature that had examined mechanisms of change during CBT for eating disorders and disordered eating. This study found that the regular eating strategy implemented during the early stages of CBT *could* be an important therapeutic mechanism that underpins CBT's effectiveness. Future large sample trials are needed to confirm this finding. For example, the dismantling design, where the regular eating strategy is removed from one group but not another group, is well suited to test the causal role of regular eating on binge eating. Overall, this paper ideally brought attention to the fact that there is a paucity of work testing CBT's change mechanisms, and that to advance the field, investigators of future RCTs should plan to test theory-specific and common treatment mechanisms. This is one avenue toward improving the effectiveness of CBT for eating disorders.

Chapter 11: General Discussion

The overarching objective of this thesis was to evaluate the cognitive-behavioural theory and treatment of eating disorders. While most of the current research in the area has focused on evaluating the efficacy and effectiveness of cognitive-behavioural *treatment*, this research does not provide direct evidence in support of the underlying cognitive-behavioural theory of eating disorder maintenance. As reviewed in the preceding chapters, minimal research has directly evaluated assumptions that underpin the hypotheses derived from the cognitive-behavioural *theory* of eating disorder maintenance. Therefore, through a series of four distinct studies, this thesis examined the validity of this cognitive-behavioural model. The next section of this chapter will review the research questions outlined, how they were addressed in the four studies included in this thesis, what the relationship between findings of the four studies is, and how these findings relate to the broader literature. I will also focus on the implications of this program of research. Finally, I will discuss the limitations of this thesis and future directions for research, and will present an overall conclusion.

Is CBT an Efficacious Treatment for Eating Disorders?

The first study of this thesis aimed to examine whether CBT is an efficacious treatment for individuals with eating disorders. This was motivated by the fact that finding CBT to be more effective than no treatment provides foundational and indirect evidence in support of the validity of the underlying cognitive-behavioural theory (Lorenzo-Luaces et al., 2014). Additionally, demonstrating that CBT is more effective than other psychotherapies (based on different theoretical models) provides even stronger support for the cognitive-behavioural model, as it provides evidence that the observed clinical change is *likely* achieved through CBT's specific mechanisms of action, rather than through common therapeutic factors, such as the therapeutic alliance or the therapeutic environment (Lorenzo-Luaces et

al., 2014). To answer these questions, the first study employed meta-analytic procedures to test the short and long-term efficacy of CBT.

Combined, data from the 79 trials included in this meta-analysis demonstrated that CBT is an efficacious treatment for eating disorders (Study 1). Specifically, therapist-led and guided self-help CBT was significantly superior to control (i.e., wait-lists and care as usual) conditions, with moderate to large effect sizes, at post-treatment and follow-up on both behavioural and cognitive symptoms in BN and BED. When comparing CBT to active conditions (any other psychological intervention), CBT was also shown to be more efficacious in the treatment of BN and BED, but not AN. These findings are consistent with those of previous systematic reviews (Hay, 2013; Hay et al., 2009), and reinforces clinical practice guidelines (National Institute of Clinical Excellence, 2017) which recommend CBT as a first-line treatment for BN and BED. Subgroup analyses also showed that the superiority of CBT over active control conditions *only* occurred when studies delivered a CBT manual that was based on Fairburn's cognitive-behavioural maintenance model. Studies that delivered alternative CBT protocols (e.g., appetite-focused CBT, Beckian-based CBT) or modified Fairburn's treatment manual (e.g., removing key therapeutic strategies) showed no clear benefit over other active psychological comparisons. These findings therefore offer some indirect support for the specific cognitive-behavioural model of eating disorders and its hypothesized mechanisms of change outlined by Fairburn et al (2003).

Another important finding from Study 1 was that when any CBT-based protocol for any eating disorder presentation was compared to other *specific* psychological interventions, CBT was shown to outperform IPT and behavioural weight loss at post-treatment and follow-up. However, there was no evidence that CBT was more efficacious than behaviour therapy or non-specific supportive therapy for eating disorders. It is important to note that there were very few studies contributing to those head to head comparisons, so it may be that there was

insufficient power to detect significant differences between treatments. For instance, to have sufficient statistical power (.80) to detect a small effect (g= 0.30) between two active treatments, one would need at least 14 studies with an average sample size of 25 participants per condition (Borenstein et al., 2009). Some of the analyses in the present meta-analysis, for example, only consisted of three comparisons, so insufficient power was likely. Consequently, it is important to interpret these findings with this in mind and not overstate the conclusions of analysis that included a small number of studies.

Overall, the findings from this meta-analysis suggest that although some of CBT's effectiveness may be partly attributed to common factors, as suggested by previous reviews (Spielmans et al., 2013), it is more likely that most of CBTs success is a result of the specific CBT strategies and its purported mechanisms of change. However, although the results of Study 1 provide necessary evidence in support of this contention, this type of evidence is not sufficient. Indeed, rather than aggregating data from pre-post RCTs, what is required are studies that test whether changes in the purported mechanisms of action during the course of CBT account for or explain CBT's effectiveness (i.e., process-based research; Kazdin, 2007). As a result, the second study of this thesis sought to evaluate the evidence of studies that have tested mediators, moderators, and predictors of change during CBT for eating disorders.

Do We Know How, and For Whom, CBT Works?

Having found evidence supporting the efficacy of cognitive-behavioural *treatment,* the next study sought to synthesise the research that has examined mediators, moderators, and predictors of treatment outcome. The goal of this study was to therefore see whether there was *direct* empirical support for the underlying cognitive-behavioural model. Such direct support would come from research testing whether therapeutic change during CBT is explained by the mechanisms hypothesised by the cognitive-behavioural model (Kraemer et

al., 2002). It was argued that studying mediators, moderators, and predictors of outcome can answer this question and can thus test the assumptions that underpin this model (Kazdin, 2007). Additionally, focusing attention on the study of mediators, moderators, and predictors may also lead to improvements in CBT's effectiveness. For example, pinpointing mediators of change can make a treatment more powerful and more cost-effective, as active therapeutic elements known to target these mediators can be strengthened, and inactive therapeutic elements can be discarded (Murphy et al., 2009). Further, identifying moderators and predictors of change can help ensure that clients receive a particular psychological treatment that is effective for their given circumstances and presenting clinical profile (Kraemer et al., 2002).

Study 2 identified 65 studies that had tested predictors, moderators, and mediators of response to CBT. Overall, little consistency across the included studies emerged, particularly with respect to (a) the type of variables explored as mediators, moderators, and predictors, (b) the way in which outcomes were operationalized, and (c) the type of treatment modality delivered. There was, however, convincing evidence that the mediational mechanism of rapid response to treatment was consistently associated with better outcomes across a range of eating disorder diagnoses (i.e., BN, BED, and OSFED). This finding is consistent with two recent meta-analyses showing that an early response to a broad range of eating disorder treatments (not just CBT) is the most potent factor associated with successful outcomes (Linardon et al., 2016a; Vall & Wade, 2015). This finding also outlines how clinicians should aim to achieve early change in treatment, as it will likely signify continued symptom improvement. Study 2 also found preliminary evidence demonstrating that specifically reducing dietary restraint in the early stages of treatment mediated better short and long-term outcomes. This suggests that reducing dietary restraint could be a key mechanism of change underpinning CBT's success. This suggestion is consistent with the cognitive-behavioural

model's hypothesis that dietary restraint is an important maintaining mechanism of binge eating, which should therefore be an important target for treatment during the initial stages (Fairburn, 2008). Given that only associations between changes in dietary restraint and treatment outcome were observed, the causal nature of this hypothesised mechanism of action is not yet known.

Putative moderating variables were also synthesised from included studies. Few moderators had been tested thus far. Most explored moderators had only been examined in BED samples, and there was no evidence that any particular baseline variable was associated with a better or worse outcome for CBT relative to another psychological or pharmacological treatment. Broadly, this finding highlights that we do not yet have enough information to be able to match clients to a particular treatment based their psychological profile, and that the conditions under which CBT is most effective are not yet known (Kraemer, 2016).

Predictors of outcome were also synthesised. Although multiple potential predictors of outcome were tested across the identified studies, most predictors were shown to be statistically unrelated to outcomes. It was noted that this was likely a result of the different definitions of treatment outcome, and of the fact that several studies were not sufficiently powered to detect statistically significant relationships. These findings demonstrate that important variables that distinguish between treatment responders and non-responders have yet to be identified in the literature.

Overall, findings from this study offer important and unique contributions to the current literature. This was the first study to examine factors associated with outcome *specifically* to CBT. Previous reviews (e.g., Shapiro et al., 2007; Vall & Wade, 2015) have collated information about predictors and moderators of response across a range of distinct pharmacological and psychological interventions for eating disorders. Thus, what may have been highlighted as a robust predictor in previous reviews may not have been a robust

predictor when analysing studies that exclusively administered CBT, consequently limiting our ability to draw conclusions about the impact of specific variables on specific treatments. To highlight this with an example, Vall and Wade (2015) concluded that a higher BMI was one of the most robust predictors of better outcomes. This conclusion was based on studies that delivered a range of treatment types and formats to a range of eating disorder presentations. By contrast, Study 2 found some evidence that a higher BMI was better outcome for BN samples, but no evidence that BMI was related to outcomes for mixed eating disorder or BED samples. This suggests that BMI may be an important determinant of treatment outcome for some eating disorder subtypes but not others. There were also some consistencies with prior research. The finding that a rapid response to CBT was the factor most robustly related to better outcomes was also identified in previous reviews (Vall & Wade, 2015). Broadly, the present findings offer unique and novel insights toward understanding how, for whom, and under what conditions, CBT for eating disorders work.

Several broader implications emerged from the second study's findings. Most importantly, because there is only a reduced number of studies investigating mediating variables, CBTs mechanisms of action are not known. Thus, it is not conclusive whether therapeutic change during CBT is a result of theory-specific mechanisms and/or CBT-specific strategies, or a result of the factors that are common across therapies. While this systematic review could not provide a definitive answer to this, it did highlight the need for future research to start addressing this question (Murphy et al., 2009). Additionally, as moderators and predictors of change were large unexplored and unrelated to treatment outcome, respectively, it is also unclear who will or will not respond to this treatment. Critically, this review found that most moderators and predictors were examined because of their convenience (i.e., they are part of a routine baseline assessment, such as client demographics, clinical history) rather than their theoretical value. For instance, as part of the

rationale for devising CBT-Eb, Fairburn et al (2003) argued that many individuals who failed to respond to the original CBT-BN did so because of the presence of one or more additional maintaining mechanisms that were not adequately addressed during treatment (e.g., interpersonal problems, mood intolerance, low self-esteem, clinical perfectionism). Theoretically, the presence of such maintaining mechanisms should therefore be a powerful and consistent predictor or moderator of treatment failure. These variables, however, were not found to have been tested as predictors or moderators in previous research.

Overall, findings from the second study demonstrated that few studies have tested the underlying cognitive-behavioural model of eating disorder maintenance, suggesting that empirical evidence for this model is scarce. This is despite the fact that cognitive-behavioural *treatment* has received extensive support for its efficacy in treating eating disorders (Study 1). Consequently, and consistent with recommendations made for researchers developing or evaluating complex health interventions (Campbell et al., 2000), the third study aimed to evaluate the cognitive-behavioural model of eating disorder maintenance. In particular, this study evaluated the hypothesised cognitive-behavioural pathways in a cross-sectional study, with the intention of providing the necessary (albeit not sufficient) evidence for CBT's hypothesised mechanisms of change.

Can the Cognitive-Behavioural Pathways be validated?

The first two systematic reviews highlighted the absence of direct empirical support for the cognitive-behavioural model of eating disorder maintenance. Consequently, the third study evaluated this model and its proposed pathways using a cross-sectional design and SEM in a large community sample. The results of the study showed that the proposed cognitive-behavioural model and pathways were largely supported by the data. In particular,

shape and weight over-concern predicted dietary restraint, dietary restraint predicted binge eating, and binge eating predicted purging. The finding that these statistical associations were observed in the direction hypothesised by the cognitive-behavioural model provides the foundational and necessary (albeit not sufficient) evidence indicating that change during CBT *could* occur for the reasons specified by theory—that is, through modifying the core maintaining mechanisms (Fairburn et al., 1993b).

A subsequent aim of the third study was to explore the added contribution to the constructs of body checking, body avoidance, and dichotomous thinking within the context of the cognitive-behavioural model. Both body checking and avoidance are hypothesized to mediate the link between shape and weight concerns and dietary restraint (e.g., Shafran et al., 2004), and dichotomous thinking is hypothesized to mediate the link between dietary restraint and binge eating (Fairburn et al., 2003a). However, although statistical correlations between these variables and disordered eating have been observed (Byrne et al., 2008; Rosen et al., 1991; Shafran et al., 2004), the role of these variables within the cognitive-behavioural model had not been examined, and the mediational hypotheses mentioned above had not been tested before this study was conducted.

The results showed that body checking (but not avoidance) mediated the link between shape and weight concerns and dietary restraint, and dichotomous thinking mediated the link between dietary restraint and binge eating. These findings suggest that body checking and dichotomous thinking could be important factors that interrelate with cognitive-behavioural maintaining mechanisms and explain their link with eating disorder symptoms. Further, the statistical associations identified provided preliminary evidence that targeting these additional factors may also increase CBT's effectiveness. These variables warrant greater consideration as possible mediators of change during CBT.

Modelling these pathways cross-sectionally provides important foundational evidence in support of the validity of the cognitive-behavioural model of eating disorder maintenance. A next step in providing further support to this theoretical model comes from research examining the role of these maintaining mechanisms during a cognitive-behavioural *treatment* (Lampard & Sharbanee, 2015). The fundamental assumption of the cognitive-behavioural model is that if a treatment is capable of targeting the proposed eating disorder maintaining mechanisms, then it should be an effective treatment for reducing the core behavioural symptoms of eating disorders, including binge eating and purging (Fairburn et al., 1993). These purported maintaining mechanisms are therefore addressed via a range of different CBT strategies. To provide a preliminary test of this major assumption of CBT, the final study of this thesis aimed to explore the impact that changes in the proposed treatment strategies and maintaining mechanisms had on symptom improvement during CBT.

Does CBT Work because of the Reasons Specified by the Model?

An important conclusion from Study 2 was that it was unclear whether CBT for eating disorders is effective because of the reasons specified by its underlying model (i.e., through modifying the proposed maintaining mechanisms) or because of the reasons that occur across all psychotherapies (e.g., therapeutic alliance, empathic concern). This conclusion was based on the finding that minimal research has been devoted toward uncovering CBT's change mechanisms. Study 3 found evidence suggesting that some particular mechanisms (i.e., dietary restraint, body checking, dichotomous thinking, and shape and weight concerns) could account for CBT's effectiveness; however, the cross-sectional nature of those data precluded any inference regarding the direction of these relationships, and whether changes in these variables relate to changes in symptoms during treatment. In order to (a) offer incremental evidence in support of CBT's change mechanisms, (b) motivate research in the

area of identifying CBT's mechanisms of action, and (c) generate hypotheses for future research examining treatment mechanisms, the final study of this thesis used a SCED to explore the plausible mechanisms of change in participants exhibiting disordered eating who were treated with an eight week CBTgsh program.

Preliminary support for some of the CBT model's hypothesised mechanisms of change was identified in the final study. Consistent with Murphy and colleagues' hypothesis that the regular eating strategy is a crucial mechanism underpinning CBT's effectiveness, this study found that, for a number of participants, an adherence to regular eating principles in the early stages of treatment was associated with a concurrent decrease in dietary restraint and binge eating. In addition, when participants did not adhere to regular eating during the later stages of treatment, a concurrent *increase* in binge eating frequency was observed. Contrary to the cognitive-behavioural model of eating disorder maintenance, this study did not find evidence that targeting and reducing weight checking and avoidance, and weight and shape concerns were associated with behavioural symptom improvement, including dietary restriction and binge eating.

Findings from this study provided important foundational and preliminary evidence necessary to support a major hypothesis proposed by the cognitive-behavioural model. That is, Fairburn (2008) has argued that the regular eating strategy, which is implemented in the second week of CBT, is the most crucial element of CBT, because it is hypothesised to reduce binge eating via modifying two particular forms of dietary restraint: delayed eating and dietary restriction. Currently, there is very little direct support for this hypothesis, as research has only identified cross-sectional associations between the frequency of regular eating and levels of binge eating (Zendegui et al., 2014). Thus, the demonstration that an early adherence to regular eating was associated with a concurrent decrease in dietary restraint and binge eating provides evidence that this particular therapeutic strategy could be

crucial for CBT's success. It might also explain the rapid response to CBT observed in many individuals, and its association with a higher likelihood of recovery (Linardon et al., 2016a; MacDonald, McFarlane, Dionne, David, & Olmsted, 2017; MacDonald, Trottier, McFarlane, & Olmsted, 2015). While these findings suggest that the introduction of this "regular eating strategy" should be prioritized by clinicians treating disordered eating, it is important to note that these findings are preliminary. Indeed, it would be better to consider these results as a first step in encouraging further research that may more systematically explore this mechanism. It is crucial that the causal role of the regular eating strategy is examined in the future, ideally via a dismantling study, where this therapeutic strategy is removed in one group of participants but not the other, in which symptom improvements are then compared. Ultimately, it is hoped that these preliminary data will provide an impetus for future large sample trials examining the mechanisms of change for CBT.

Recommendations for Future Research

This program of research has highlighted the need for researchers to direct attention to questions about the process and mechanisms of therapeutic change during CBT for eating disorders. At present, CBT's change mechanisms have not been identified empirically, although some are clearly articulated in the theoretical model (as concluded in Study 2). Consequently, this section will provide three directions for future research to pinpoint CBT's change mechanisms.

The first recommendation for future research is to use theory as a guide for studying the mechanisms of change during CBT for eating disorders. Cognitive-behavioural *theory* provides hypotheses regarding when, how, and why change occurs, which then determines what, when, and why particular treatment strategies are implemented. Although using theory as a guide would seem to be an obvious recommendation, Study 2 demonstrated that most

variables used in *prediction/moderation* analyses seemed to have been selected for convenience rather than for their theoretical relevance. Basing the design of research around the testing of specific mediational hypotheses set *a priori* would then dictate what variables are measured, how they are operationalized, and when they are to be assessed (Murphy et al., 2009). This would increase the chance of identifying reliable factors that account for therapeutic change, and would therefore aid in the understanding of how and why CBT works.

The second recommendation for future research is to conceptualise CBT as a series of distinct procedural elements (rather than as a complete entity), with each element likely to contain its own mechanism of change (as suggested by Murphy et al., 2009). Viewing CBT in this manner would allow for identification of the active treatments components, that is, those that are effective in modifying a particular symptom or behaviour (Murphy et al., 2009). For example, the "regular eating" procedure of CBT was developed as a strategy to target binge eating. The hypothesised mechanism of change that underpins the "regular eating" procedures proposed effect on binge eating is through reducing dietary restraint (Fairburn, 2008). Currently, there is little *conclusive* evidence on whether CBT strategies like "regular eating" have a specific effect on binge eating and, if so, whether this effect is explained because of its effect on dietary restraint (as proposed by theory). Ultimately, studying the specific components of CBT, what effects they have, and how they achieve these effects, is crucial for improving the efficiency and effectiveness of this treatment (Murphy et al., 2009).

The third recommendation for future research is to draw from the experimental psychopathology (EPP) literature, which is the experimental study of mental health conditions (Jansen, 2016). EPP allows for testing of the *causal* role of certain maintaining mechanisms. The current evidence supporting the cognitive-behavioural pathways (see Chapter 7) is only correlational. Without an in-depth understanding of the causal status of the

proposed maintaining factors, it is not possible to know whether CBT is targeting the optimal treatment mechanisms (Jansen, 2016). One way to examine the causal status of cognitive-behavioural mechanisms is to conduct a dismantling RCT. In this design, the researcher isolates the specific target mechanism (e.g., weight concerns) by removing a key therapeutic strategy designed to address this mechanism (e.g., weekly weighing) in one group of participants but not for the other group. Any group differences at post-treatment on target mechanisms (e.g., weight concerns) and symptoms (e.g., binge eating or purging) are suggested to be *caused* by that therapeutic strategy (e.g., weekly weighing). Although this is one of the "gold-standard" approaches to study the mechanisms of change, this approach is expensive, time-consuming, and is sometimes not feasible for researchers. Consequently, it is important that other, less resource intensive methods are available to test the causal status of hypothesised cognitive-behavioural mechanisms. Experimental psychopathology (the experimental study of psychopathology) is one approach that can achieve this goal. Such approaches like EPP are also crucial for informing future dismantling designs, as they can provide the initial evidence that a particular mechanism warrants additional investigation in a RCT.

In EPP, variables that are predicted to be related to the presence of symptoms are manipulated in controlled laboratory settings (Jansen, 2016). The objective is to examine whether the manipulation of a hypothesized causal factor can induce some of the predicted symptoms observed in a particular psychological disorder (Zvolensky, Lejuez, Stuart, & Curtin, 2001). The manipulations are, however, of small magnitude and are done in healthy individuals (for ethical reasons), so the observed effects tend to be only mild imitations of what would otherwise be observed in clinical cases (Vervliet & Raes, 2013). The important goal is to examine the causal relation between a hypothesised maintaining mechanism and a target symptom. Consequently, EPP is not only a useful method for testing the causal status

of cognitive-behavioural maintaining mechanisms, but it can also assist the next generation of clinical research, by helping narrow down the search for mechanisms of change that may operate during treatment (Jansen, 2016). For these reasons, taking advantage of the EPP paradigm is another important avenue for future research on CBTs mechanisms.

For example, EPP could answer the question on whether weight checking *causes* elevated levels of weight concerns in a healthy population. Using a simple experimental design for illustrative purposes, a researcher could randomly assign (to prevent selection effects) healthy participants to one of two conditions: those that are weighed in front of an experimenter and those that are not weighed. The experimenter could then measure participant's level of weight concerns immediately after this manipulation. If there are group differences in weight concerns post-manipulation, such that those who were weighed expressed significantly highly levels of weight concern than those who were not, then it could be concluded that weight checking plays a causal role in weight concerns, at least in the short term. This finding would have important implications for clinical research. Knowing that a causal relationship between weight checking and weight concerns exists could then prompt clinical research to isolate the specific causal effect (via a dismantling study, for example) of the therapeutic strategies designed to target weight checking (e.g., weekly weighing). This approach would allow for an analysis of the "active ingredients" in CBT. In that regard, EPP is useful for providing the "groundwork" on the types of variables that warrant investigation as causal mechanisms in expensive clinical trials.

Limitations of this Thesis

There are several important limitations of this thesis. First, the use of meta-analytic procedures to quantify the effects of CBT for eating disorders may be an important limitation to this thesis that should be addressed. In particular, meta-analyses are often criticised for

combining studies that are too heterogeneous. Thus, computing a summary effect from a selected set of heterogonous studies may misrepresent or even simplify a complex relationship (Borenstein et al., 2009). For example, although the meta-analysis presented in Study 1 tried to account for this heterogeneity by conducting analyses separately across each diagnostic type (i.e., AN, BN, and BED trials) or treatment mode (i.e., therapist-led CBT, guided self-help), and also by conducting numerous subgroup analyses, we still found a large degree of statistical and methodological heterogeneity *within* these subgroups. This may be attributed to variability in factors such as treatment lengths, patient exclusion criteria, country of publication, etc.). There were not enough trials to try and account for or model this within-group heterogeneity. Consequently, the conclusions drawn from Study 1 may have been slightly different to conclusion drawn from another study that made efforts to *qualitatively* review all of the trials on CBT for eating disorders. Nevertheless, meta-analyses do have recognised strengths, including, for example, greater power to detect effects (see Chapter 4 for a full discussion), and many practice guidelines rely on meta-analytic findings to make clinical and policy recommendations (NICE, 2017).

Second, the meta-analysis reporting the efficacy of CBT was based on a large number of studies that were of poor quality, according to the Cochrane Risk of Bias Tool. Poor quality studies may overestimate the efficacy of particular psychological or pharmacological interventions (Higgins & Green, 2011). Because of this, experts usually recommend meta-analysts to perform a series of sensitivity analyses, which involves computing a separate summary effect from trials that are *only* of high quality. Such sensitivity analyses therefore allow one to compare the effect size observed from high quality trials to the effect size observed from all included trials. Although performing sensitivity analyses based on trial quality is recommended (Higgins & Green, 2011), Study 1 did not conduct sensitivity analyses for a number of reasons. First, the number of trials in many of the main analyses was

typically small (e.g., all but one comparison contained between 3-7 trials), so there were not enough trials to conduct sensitivity analyses based on study quality. Second, in the eating disorder literature, there is currently no evidence from recent meta-analyses to suggest that the effect sizes observed from poor quality RCTs differs significantly from the effect sizes observed from high quality RCTs (Grenon et al., 2017; Linardon, Fairburn, Fitzsimmons-Craft, Wilfley, & Brennan, 2017; Linardon & Wade, 2018; Traviss-Turner, West, & Hill, 2017). It is noted, however, that results from this meta-analysis are based on a number of low quality trials, and should therefore be interpreted bearing this in mind.

Third, the two systematic reviews in this thesis were not prospectively registered, which presents another limitation. Current guidelines for systematic reviews recommend that authors register their systematic review prior to conducting it (Higgins & Green, 2011). Registration is said to play an important part in helping ensure the integrity of the evidence base upon which decisions are made. A protocol of a systematic review written in advance should ensure that the review methods are transparent and reproducible, and that adherence to this pre-specified protocol should assist in avoiding potential biases (e.g., reporting biases, alterations of inclusion/exclusion criteria; Stewart, Moher & Shekelle, 2012). The fact that the present systematic reviews were not prospectively registered is a limitation to this thesis.

Fourth, the use of non-clinical samples to test the cognitive-behavioural model in Study 3 and 4 was a limitation. However, the transdiagnostic perspective stipulates that the purported cognitive-behavioural mechanisms operate to maintain eating disorder psychopathology, irrespective of diagnosis (Fairburn et al., 2003). Thus, it could be argued that the cognitive-behavioural model can be applied to anyone as long as disordered eating (e.g., binge eating) symptoms are present. It is therefore reasonable to speculate that the findings from Study 3 and 4, which are based on non-clinical samples, may be applicable to clinical cases. Indeed, some of the evidence gathered aligned well with the predictions of the

model. However, given the distinct nature, severity, and course of eating disorder cases relative to non-clinical cases (Fairburn & Harrison, 2003), it is important to acknowledge that the present findings may not necessarily generalise to individuals with eating disorders. A decision was made to recruit non-clinical samples because it made it possible to achieve the aims of this thesis. Specifically, in the third study, recruiting non-clinical participants made it possible to obtain a sufficiently large and adequately powered sample to test the original and expanded cognitive-behavioural model using SEM techniques, consistent with previous studies testing theories of eating disorders (for a review, see Pennesi & Wade, 2016). For the fourth study, individuals who exhibited symptoms of disordered eating but did not meet diagnostic criteria were recruited. We predicted that a "higher functioning" sample would (a) be more likely to adhere to the manualized protocols, (b) allow clinicians to deliver the manualized protocol as intended, without the need to adjust the intervention based on circumstances typical across clinical cases (e.g., severe depression, low motivation), (c) be less likely to dropout from treatment (Vall & Wade, 2015). For these reasons, the use of a "subthreshold" sample was more likely going to ensure that the CBT components were administered consistently across participants, allowing for a more accurate test of the impact of these treatment components. Although the current findings provide insights about the applicability of the cognitive-behavioural theory to "high risk" samples, replicating these findings in individuals with a clinical eating disorder is a necessary next step.

Fifth, the use of self-report questionnaires was another limitation of this thesis. Self-report questionnaires have the potential to introduce social desirability biases, which has been shown to be more pronounced when participants fill out these questionnaires in the presence of an experimenter who is known to review the responses (Van de Mortel, 2008). The issue of responding in a socially desirable fashion may have been evident in Study 4, where clinicians asked participants to report their level of adherence to the intervention and the

severity of their symptoms at the end of each guided session. In addition, another problem with self-report questionnaires is that the scores obtained from these questions do not always match the scores obtained from clinician-led interviews. For example, although high correlations between the interviewer-based EDE and the self-report EDE-Q scores have been observed, meta-analytic research has demonstrated that participants tend to obtain significantly higher scores on the EDE-Q than on the EDE (Berg, Peterson, Frazier, & Crow, 2011). It was suggested that this could be a result of participants overestimating their symptoms on self-report questionnaires. Also, because the EDE-Q is filled out independently, this discrepancy may be due to the fact that there is no opportunity for clinicians to clarify the meaning of certain items, i.e., objective versus subjective binge eating (Berg et al., 2011). Although the EDE is considered the preferred "gold-standard" for measuring disordered eating, the EDE-Q does have its advantages. That is, it is takes 20-30 minutes to complete, is easier to administer, and can be completed at a time and location of choice (making it applicable to Study 3). The choice of self-reported measures was also made because there are no semi-structured interviews to assess the other constructs studied in this thesis (e.g., body checking and avoidance).

Sixth, the cross-sectional nature of Study 3 is another limitation of this thesis. Although the statistical associations observed were consistent with the cognitive-behavioural model's hypotheses, the cross-sectional nature of this study precludes any inferences about the direction of these relationships. The identified statistical associations do, however, set up important hypotheses for future experimental or longitudinal research testing the temporal nature of these paths or the feedback loops specified in the model.

Seventh, the preliminary nature of Study 4 was also a limitation that needs to be considered. The small sample size of this study not only made it difficult to generalize these findings to a larger population, but it also meant that it was not possible to prevent the

observable baseline differences between those randomized to CBT and those randomized to HAES. Second, the interpretations of this study's findings were based on visual analysis of graphed data. Although visual analysis is the recommended approach to data analysis in SCEDs (Kazdin, 2011), visual analysis has been questioned on the grounds that it might be prone to researcher expectations and biases (Brossart et al., 2006). Third, this study did not employ a period where participants achieved a stable baseline. Future research would benefit from using a multiple baseline design for testing some of CBT's change mechanisms, in which participants are randomized to different baseline periods (e.g., two or three weeks) (Kazdin, 2011). A multiple baseline design is a more sophisticated design that allows for firmer conclusions regarding the impact of an intervention.

Conclusion

This thesis aimed to evaluate the cognitive-behavioural theory and treatment of eating disorders. A series of four studies were conducted to achieve this aim, including two systematic reviews and two empirical studies. While the first systematic review (Chapter 5) provided conclusive evidence that CBT is an efficacious treatment for a range of eating disorder presentations, the second systematic review (Chapter 6) could not identify what mechanisms were operating to achieve CBT's effectiveness. Consequently, the two empirical studies sought to gather converging evidence in support of the adequacy of the cognitive-behavioural theory of eating disorder maintenance. The third study (Chapter 8) validated the cognitive-behavioural model pathways in a cross-sectional study, while the fourth study (Chapter 10) found preliminary evidence to suggest that, consistent with the cognitive-behavioural model, the regular eating strategy could be a crucial mechanism that underpins CBT's effectiveness.

Overall, results from this thesis provide support for the cognitive-behavioural maintenance model of eating disorders, and suggests that CBT for eating disorders (a) works at least as well or better than other interventions, and (b) is *likely* to "work" because of the reasons specified by theory. However, this thesis does highlight the need for further clarification of CBT's casual change mechanisms, as the preliminary evidence presented here warrants more detailed investigation. Currently, the reasons for the structure of manualized CBT, including when and why certain therapeutic strategies are introduced, are based on theoretical hypotheses that are yet to be tested empirically. Ideally, this thesis will bring this matter to attention and prompt the next generation of research that seeks to study CBT's change mechanisms. This line of research should be a research priority, because it will be crucial for improving the efficacy, efficiency, and cost-effectiveness of CBT for eating disorders. Ultimately, given that CBT is the most empirically supported eating disorder treatment that is recommended by international guidelines (NICE, 2017), additional efforts toward augmenting CBTs active therapeutic components to make it even more potent is important for improving recovery rates and the quality of life in those with an eating disorder.

References

Aardoom, J. J., Dingemans, A. E., Spinhoven, P., van Ginkel, J. R., de Rooij, M., & van Furth, E. F. (2016). Web-based fully automated self-help with different levels of therapist support for individuals with eating disorder symptoms: a randomized controlled trial. *Journal of Medical Internet Research, 18*, e159. doi: 10.2196/jmir.5709

Aderka, I. M., Nickerson, A., Bøe, H. J., & Hofmann, S. G. (2012). Sudden gains during psychological treatments of anxiety and depression: A meta-analysis. *Journal of Consulting and Clinical Psychology, 80*, 93-101. doi: http://dx.doi.org/10.1037/a0026455

Agras, W. S., Crow, S. J., Halmi, K. A., Mitchell, J., Wilson, G., & Kraemer, H. C. (2000a). Outcome predictors for the cognitive behavior treatment of bulimia nervosa: Data from a multisite study. *The American Journal of Psychiatry, 157*, 1302-1308. doi: 10.1176/appi.ajp.157.8.1302

Agras, W. S., Fitzsimmons-Craft, E. E., & Wilfley, D. E. (2017). Evolution of cognitive-behavioral therapy for eating disorders. *Behaviour Research and Therapy, 88,* 26-36.

Agras, W. S., Rossiter, E. M., Arnow, B., Schneider, J. A., Telch, C. F., Raeburn, S. D., . . . Koran, L. M. (1992). Pharmacologic and cognitive-behavioral treatment for bulimia nervosa: A controlled comparison. *The American Journal of Psychiatry, 149,* 82-87. doi: 10.1176/ajp.149.1.82

Agras, W. S., Telch, C. F., Arnow, B., Eldredge, K., Wilfley, D. E., Raeburn, S. D., . . . Marnell, M. (1994). Weight loss, cognitive-behavioral, and desipramine treatments in binge eating disorder: An additive design. *Behavior Therapy, 25,* 225-238.

Agras, W. S., Walsh, B. T., Fairburn, C. G., Wilson, G. T., & Kraemer, H. C. (2000b). A multicenter comparison of cognitive-behavioral therapy and interpersonal psychotherapy for bulimia nervosa. *Archives of General Psychiatry, 57,* 459-466.

Andersson, G., & Cuijpers, P. (2009). Internet-based and other computerized psychological treatments for adult depression: a meta-analysis. *Cognitive Behaviour Therapy, 38,* 196-205.

Andersson, G., Cuijpers, P., Carlbring, P., Riper, H., & Hedman, E. (2014). Guided Internet-based vs. face-to-face cognitive behavior therapy for psychiatric and somatic disorders: a systematic review and meta-analysis. *World Psychiatry, 13,* 288-295.

Andrews, G., Cuijpers, P., Craske, M. G., McEvoy, P., & Titov, N. (2010). Computer therapy for the anxiety and depressive disorders is effective, acceptable and practical health care: a meta-analysis. *PLoS One, 5,* 131-196.

Arcelus, J., Haslam, M., Farrow, C., & Meyer, C. (2013). The role of interpersonal functioning in the maintenance of eating psychopathology: a systematic review and testable model. *Clin Psychol Rev., 33,* 156-167. doi: https://doi.org/10.1016/j.cpr.2012.10.009

Banasiak, S. J., Paxton, S. J., & Hay, P. (2005). Guided self-help for bulimia nervosa in primary care: A randomized controlled trial. *Psychological Medicine, 35*, 1283-1294.

Barlow, D. H., & Hersen, M. (1984). *Single case experimental designs*. USA: Pergamon Press.

Baron, R. M., & Kenny, D. A. (1986). The moderator–mediator variable distinction in social psychological research: Conceptual, strategic, and statistical considerations. *Journal of Personality and Social Psychology, 51*, 1173-1182.

Bartolucci, A. A., & Hillegass, W. B. (2010). Overview, strengths, and limitations of systematic reviews and meta-analyses *Evidence-based practice: Toward optimizing clinical outcomes* (pp. 17-33). Berlin: Springer.

Berg, K. C., Peterson, C. B., Frazier, P., & Crow, S. J. (2011). Convergence of scores on the interview and questionnaire versions of the Eating Disorder Examination: A meta-analytic review. *Psychological Assessment, 23*, 714-724. doi: http://dx.doi.org/10.1037/a0023246

Berg, K. C., Peterson, C. B., Frazier, P., & Crow, S. J. (2012). Psychometric evaluation of the eating disorder examination and eating disorder examination-questionnaire: A systematic review of the literature. *International Journal of Eating Disorders, 45*, 428-438.

Berkman, N. D., Lohr, K. N., & Bulik, C. M. (2007). Outcomes of eating disorders: A systematic review of the literature. *International Journal of Eating Disorders, 40*, 293-309. doi: 10.1002/eat.20369

Bhatnagar, K. A. C., Wisniewski, L., Solomon, M., & Heinberg, L. (2013). Effectiveness and feasibility of a cognitive-behavioral group intervention for body image disturbance in women with eating disorders. *Journal of Clinical Psychology, 69*, 1-13. doi: 10.1002/jclp.21909

Bland, J. M., & Altman, D. G. (2000). Statistics notes: the odds ratio. *BMJ: British Medical Journal, 320*(7247), 1468.

Bohn, K., Doll, H. A., Cooper, Z., O'Connor, M., Palmer, R. L., & Fairburn, C. G. (2008). The measurement of impairment due to eating disorder psychopathology. *Behaviour Research and Therapy, 46*, 1105-1110. doi: 10.1016/j.brat.2008.06.012

Bohon, C., Stice, E., & Burton, E. (2009). Maintenance factors for persistence of bulimic pathology: A prospective natural history study. *International Journal of Eating Disorders, 42*, 173-178.

Borenstein, M., Hedges, L. V., Higgins, J. P., & Rothstein, H. R. (2009). *Introduction to meta-analysis*. United Kingdom, UK: John Wiley & Sons.

Brossart, D. F., Parker, R. I., Olson, E. A., & Mahadevan, L. (2006). The relationship between visual analysis and five statistical analyses in a simple AB single-case research design. *Behavior Modification, 30*, 531-563.

Brown, T. A. (2015). *Confirmatory factor analysis for applied research*. New York: Guilford Publications.

Brownley, K. A., Berkman, N. D., Peat, C. M., Lohr, K. N., Cullen, K. E., Bann, C. M., & Bulik, C. M. (2016). Binge-Eating Disorder in AdultsA Systematic Review and Meta-analysisBinge-Eating Disorder: Treatment Effectiveness. *Annals of Internal Medicine, 165*, 409-420.

Byrne, S., Allen, K., Dove, E., Watt, F., & Nathan, P. (2008). The reliability and validity of the dichotomous thinking in eating disorders scale. *Eating behaviors, 9*, 154-162.

Byrne, S., Cooper, Z., & Fairburn, C. G. (2004). Psychological predictors of weight regain in obesity. *Behaviour Research and Therapy, 42*, 1341-1356.

Byrne, S., Fursland, A., Allen, K. L., & Watson, H. J. (2011). The effectiveness of enhanced cognitive behavioural therapy for eating disorders: An open trial. *Behaviour Research and Therapy, 49,* 219-226. doi: 10.1016/j.brat.2011.01.006

Byrne, S., & McLean. (2002). The cognitive-behavioral model of bulimia nervosa: A direct evaluation. *International Journal of Eating Disorders, 31,* 17-31.

Byrne, S., Wade, T., Hay, P., Touyz, S., Fairburn, C. G., Treasure, J., . . . Crosby, R. D. (2017). A randomised controlled trial of three psychological treatments for anorexia nervosa. *Psychological Medicine*, 1-11.

Byrne, S. M., & McLean, N. J. (2002). The cognitive-behavioral model of bulimia nervosa: A direct evaluation. *International Journal of Eating Disorders, 31,* 17-31.

Campbell, M., Fitzpatrick, R., Haines, A., Kinmonth, A. L., Sandercock, P., Spiegelhalter, D., & Tyrer, P. (2000). Framework for design and evaluation of complex interventions to improve health. *British Medical Journal, 321,* 694-696.

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

Carter, J. C., Olmsted, M. P., Kaplan, A. S., McCabe, R. E., Mills, J. S., & Aimé, A. (2003). Self-help for bulimia nervosa: A randomized controlled trial. *The American Journal of Psychiatry, 160,* 973-978.

Channon, S., de Silva, P., Hemsley, D., & Perkins, R. E. (1989). A controlled trial of cognitive-behavioural and behavioural treatment of anorexia nervosa. *Behaviour Research and Therapy, 27,* 529-535. doi: 10.1016/0005-7967(89)90087-9

Chen, E. Y., Brown, M. Z., Harned, M. S., & Linehan, M. M. (2009). A comparison of borderline personality disorder with and without eating disorders. *Psychiatry research, 170,* 86-90. doi: http://dx.doi.org/10.1016/j.psychres.2009.03.006

Chen, E. Y., Cacioppo, J., Fettich, K., Gallop, R., McCloskey, M. S., Olino, T., & Zeffiro, T. A. (2016). An adaptive randomized trial of dialectical behavior therapy and cognitive behavior therapy for binge-eating. *Psychological Medicine, 47*, 703-717. doi: 10.1017/S0033291716002543

Chen, E. Y., McCloskey, M. S., & Keenan, K. E. (2009). Subtyping dietary restraint and negative affect in a longitudinal community sample of girls. *International Journal of Eating Disorders, 42*, 275-283.

Chen, E. Y., McCloskey, M. S., Michelson, S., Gordon, K. H., & Coccaro, E. (2011). Characterizing eating disorders in a personality disorders sample. *Psychiatry research, 185*, 427-432.

Chen, E. Y., Touyz, S. W., Beumont, P. J., Fairburn, C. G., Griffiths, R., Butow, P., . . . Basten, C. (2003). Comparison of group and individual cognitive-behavioral therapy for patients with bulimia nervosa. *International Journal of Eating Disorders, 33*(3), 241-254.

Cooper, P. J., & Steere, J. (1995). A comparison of two psychological treatments for bulimia nervosa: Implications for models of maintenance. *Behaviour Research and Therapy, 33*, 875-885.

Crow, S., Pederson Mussell, M., Peterson, C., Knopke, A., & Mitchell, J. (1999). Prior treatment received by patients with bulimia nervosa. *International Journal of Eating Disorders, 25*, 39-44.

Cuijpers, P., Cristea, I., Weitz, E., Gentili, C., & Berking, M. (2016a). The effects of cognitive and behavioural therapies for anxiety disorders on depression: a meta-analysis. *Psychological Medicine*, 1-12.

Cuijpers, P., Donker, T., van Straten, A., Li, J., & Andersson, G. (2010). Is guided self-help as effective as face-to-face psychotherapy for depression and anxiety disorders? A

systematic review and meta-analysis of comparative outcome studies. *Psychological Medicine, 40*, 1943-1957.

Cuijpers, P., Donker, T., Weissman, M. M., Ravitz, P., & Cristea, I. A. (2016b). Interpersonal psychotherapy for mental health problems: a comprehensive meta-analysis. *American Journal of Psychiatry, 173*, 680-687.

Cuijpers, P., Driessen, E., Hollon, S. D., van Oppen, P., Barth, J., & Andersson, G. (2012). The efficacy of non-directive supportive therapy for adult depression: a meta-analysis. *Clinical Psychology Review, 32*, 280-291.

Cuijpers, P., Ebert, D. D., Acarturk, C., Andersson, G., & Cristea, I. A. (2016c). Personalized Psychotherapy for Adult Depression: A Meta-Analytic Review. *Behavior Therapy, 47*, 966-980.

Cuijpers, P., Hollon, S. D., van Straten, A., Bockting, C., Berking, M., & Andersson, G. (2013). Does cognitive behaviour therapy have an enduring effect that is superior to keeping patients on continuation pharmacotherapy? A meta-analysis. *BMJ, 3*, 2044-6055.

da Luz, F., Sainsbury, A., Mannan, H., Touyz, S., Mitchison, D., & Hay, P. (2017). Prevalence of obesity and comorbid eating disorder behaviors in South Australia from 1995 to 2015. *International Journal of Obesity, 41*, 1148-1153. doi: doi:10.1038/ijo.2017.79

Dakanalis, A., Carrà, G., Calogero, R., Zanetti, M. A., Gaudio, S., Caccialanza, R., . . . Clerici, M. (2015). Testing the cognitive-behavioural maintenance models across DSM-5 bulimic-type eating disorder diagnostic groups: a multi-centre study. *Eur Arch Psychiatry Clin Neurosci, 265*, 663-676.

Dakanalis, A., Clerici, M., Caslini, M., Gaudio, S., Serino, S., Riva, G., & Carrà, G. (2016). Predictors of initiation and persistence of recurrent binge eating and inappropriate

weight compensatory behaviors in college men. *International Journal of Eating Disorders, 49*, 581-590.

Dakanalis, A., Timko, C. A., Clerici, M., Zanetti, M. A., & Riva, G. (2014). Comprehensive examination of the trans-diagnostic cognitive behavioral model of eating disorders in males. *Eat. Behav, 15*, 63-67.

Dalle Grave, R., Calugi, S., Doll, H. A., & Fairburn, C. G. (2013). Enhanced cognitive behaviour therapy for adolescents with anorexia nervosa: An alternative to family therapy? *Behaviour Research and Therapy, 51*, R9-R12. doi: 10.1016/j.brat.2012.09.008

Decaluwé, V., & Braet, C. (2005). The cognitive behavioural model for eating disorders: A direct evaluation in children and adolescents with obesity. *Eat. Behav, 6*, 211-220. doi: http://dx.doi.org/10.1016/j.eatbeh.2005.01.006

Dekoitte Access Economics. (2012). *Paying the price: The economic and social impact of eating disorders in Australia*. New South Wales: The Butterfly Foundation. Retrieved from http://thebutterflyfoundation.org.au/wp-content/up.

Dobson, K. S., & Khatri, N. (2000). Cognitive therapy: Looking backward, looking forward. *Journal of Clinical Psychology, 56*, 907-923.

Egan, S. J., Piek, J. P., Dyck, M. J., & Rees, C. S. (2007). The role of dichotomous thinking and rigidity in perfectionism. *Behav Res Ther., 45*, 1813-1822. doi: http://dx.doi.org/10.1016/j.brat.2007.02.002

Eskildsen, A., Hougaard, E., & Rosenberg, N. K. (2010). Pre-treatment patient variables as predictors of drop-out and treatment outcome in cognitive behavioural therapy for social phobia: A systematic review. *Nordic Journal of Psychiatry, 64*, 94-105. doi: 10.3109/08039480903426929

Fairburn, C. G. (1995). *Overcoming bnge eating*. New York: Guilford Press.

Fairburn, C. G. (2013). *Overcoming binge eating*. London, UK: Guilford Press

Fairburn, C. G., Bailey-Straebler, S., Basden, S., Doll, H. A., Jones, R., Murphy, R., . . . Cooper, Z. (2015). A transdiagnostic comparison of enhanced cognitive behaviour therapy (CBT-E) and interpersonal psychotherapy in the treatment of eating disorders. *Behaviour Research and Therapy, 70*, 64-71.

Fairburn, C. G., & Beglin, S. (1994). Assessment of eating disorders: Interview or self-report questionnaire? *International Journal of Eating Disorders, 16*, 363-370.

Fairburn, C. G., Cooper, Z., Doll, H. A., O'Connor, M. E., Bohn, K., Hawker, D. M., . . . Palmer, R. L. (2009). Transdiagnostic cognitive-behavioral therapy for patients with eating disorders: A two-site trial with 60-week follow-up. *American Journal of Psychiatry, 166*, 311-319.

Fairburn, C. G., Cooper, Z., & Shafran, R. (2003a). Cognitive behaviour therapy for eating disorders: A "transdiagnostic" theory and treatment. *Behavior Research and Therapy, 41*, 509-528.

Fairburn, C. G., & Harrison, P. J. (2003). Eating disorders. *The Lancet, 361*, 407-416.

Fairburn, C. G., Jones, R., Peveler, R. C., Carr, S. J., Solomon, R. A., O'Connor, M. E., . . . Hope, R. A. (1991). Three psychological treatments for bulimia nervosa: A comparative trial. *Archives of General Psychiatry, 48*, 463-469.

Fairburn, C. G., Jones, R., Peveler, R. C., Hope, R. A., & O'Connor, M. (1993a). Psychotherapy and bulimia nervosa: Longer-term effects of interpersonal psychotherapy, behavior therapy, and cognitive behavior therapy. *Archives of General Psychiatry, 50*, 419-428. doi: 10.1001/archpsyc.1993.01820180009001

Fairburn, C. G., Kirk, J., O'Connor, M., & Cooper, P. J. (1986). A comparison of two psychological treatments for bulimia nervosa. *Behaviour Research and Therapy, 24*, 629-643.

Fairburn, C. G., Marcus, M. D., & Wilson, T. (1993b). Cognitive-behavioral therapy for binge eating and bulimia nervosa: A comprehensive treatment manual. In C. G. Fairburn & G. T. Wilson (Eds.), *Binge eating: Nature, assessment and treatment* (pp. 361-404). New York, NY: Guilford Press

Fairburn, C. G., Peveler, R., Jones, R., Hope, R., & Doll, H. (1993c). Predictors of 12-month outcome in bulimia nervosa and the influence of attitudes to shape and weight. *Journal of Consulting and Clinical Psychology, 61*, 696-698.

Fairburn, C. G., Stice, E., Cooper, Z., Doll, H. A., Norman, P. A., & O'Connor, M. E. (2003b). Understanding persistence in bulimia nervosa: A 5-year naturalistic study. *Journal of Consulting and Clinical Psychology, 71*, 103-109.

Freeman, C. P., Barry, F., Dunkeld-Turnbull, J., & Henderson, A. (1988). Controlled trial of psychotherapy for bulimia nervosa. *British Medical Journal (Clinical Research Ed.), 296*(6621), 521-525.

Freeman, C. P., Sinclair, F., Turnbull, J., & Annandale, A. (1985). Psychotherapy for bulimia: A controlled study. *Journal of Psychiatric Research, 19*, 473-478. doi: 10.1016/0022-3956(85)90056-1

Gallagher, M. W., Payne, L. A., White, K. S., Shear, K. M., Woods, S. W., Gorman, J. M., & Barlow, D. H. (2013). Mechanisms of change in cognitive behavioral therapy for panic disorder: The unique effects of self-efficacy and anxiety sensitivity. *Behaviour Research and Therapy, 51*, 767-777.

Garg, A. X., Hackam, D., & Tonelli, M. (2008). Systematic review and meta-analysis: when one study is just not enough. *Clinical Journal of the American Society of Nephrology, 3*, 253-260.

Garner, D. M., & Bemis, K. M. (1982). A cognitive-behavioral approach to anorexia nervosa. *Cognitive Therapy and Research, 6*, 123-150.

Garner, D. M., Rockert, W., Davis, R., Garner, M. V., Olmsted, M. P., & Eagle, M. (1993). Comparison of cognitive-behavioral and supportive-expressive therapy for bulimia nervosa. *American Journal of Psychiatry, 150*, 37-46.

Ge, L., Wang, J.-c., Li, J.-l., Liang, L., An, N., Shi, X.-t., . . . Tian, J.-h. (2014). The assessment of the quality of reporting of systematic reviews/meta-analyses in diagnostic tests published by authors in China. *PLoS One, 9*, e85908.

Ghaderi, A. (2001). Review of risk factors for eating disorders: Implications for primary prevention and cognitive behavioural therapy. *Scandinavian Journal of Behaviour Therapy, 30*, 57-74.

Ghaderi, A. (2006). Attrition and outcome in self-help treatment for bulimia nervosa and binge eating disorder: A constructive replication. *Eating behaviors, 7*(4), 300-308.

Ghaderi, A., & Andersson, G. (1999). Meta-analysis of CBT for bulimia nervosa: Investigating the effects using DSM-III-R and DSM-IV criteria. *Scandinavian Journal of Behaviour Therapy, 28*, 79-87. doi: 10.1080/028457199440034

Ghaderi, A., & Scott, B. (1999). Prevalence and psychological correlates of eating disorders among females aged 18–30 years in the general population. *Acta Psychiatrica Scandinavica, 99*, 261-266.

Ghaderi, A., & Scott, B. (2003). Pure and guided self-help for full and sub-threshold bulimia nervosa and binge eating disorder. *British Journal of Clinical Psychology, 42*, 257-269.

Ginzburg, D. M., Bohn, C., Höfling, V., Weck, F., Clark, D. M., & Stangier, U. (2012). Treatment specific competence predicts outcome in cognitive therapy for social anxiety disorder. *Behaviour Research and Therapy, 50*, 747-752. doi: http://dx.doi.org/10.1016/j.brat.2012.09.001

Goldbloom, D. S., Olmsted, M., Davis, R., Clewes, J., Heinmaa, M., Rockert, W., & Shaw, B. (1997). A randomized controlled trial of fluoxetine and cognitive behavioral therapy for bulimia nervosa: Short-term outcome. *Behaviour Research and Therapy, 35*, 803-811. doi: http://dx.doi.org/10.1016/S0005-7967(97)00041-7

Gormally, J., Black, S., Daston, S., & Rardin, D. (1982). The assessment of binge eating severity among obese persons. *Addictive Behaviors, 7*, 47-55.

Grenon, R., Schwartze, D., Hammond, N., Ivanova, I., McQuaid, N., Proulx, G., & Tasca, G. A. (2017). Group psychotherapy for eating disorders: A meta-analysis. *International Journal of Eating Disorders, 50*(9), 997-1013. doi: 10.1002/eat.22744

Griffiths, R. A., Hadzi-Pavlovic, D., & Channon-Little, L. (1994). A controlled evaluation of hypnobehavioural treatment for bulimia nervosa: Immediate pre-post treatment effects. *European Eating Disorders Review, 2*, 202-220. doi: 10.1002/erv.2400020405

Grilo, C. M., & Masheb, R. M. (2005). A randomized controlled comparison of guided self-help cognitive behavioral therapy and behavioral weight loss for binge eating disorder. *Behaviour Research and Therapy, 43*, 1509-1525.

Grilo, C. M., Masheb, R. M., Wilson, G. T., Gueorguieva, R., & White, M. A. (2011). Cognitive-behavioral therapy, behavioral weight loss, and sequential treatment for obese patients with binge-eating disorder: A randomized controlled trial. *Journal of Consulting and Clinical Psychology, 79*, 675-685.

Gu, J., Strauss, C., Bond, R., & Cavanagh, K. (2015). How do mindfulness-based cognitive therapy and mindfulness-based stress reduction improve mental health and wellbeing? A systematic review and meta-analysis of mediation studies. *Clinical Psychology Review, 37*, 1-12.

Halmi, K. A., Falk, J. R., & Schwartz, E. (1981). Binge-eating and vomiting: A survey of a college population. *Psychological Medicine, 11*, 697-706.

Hart, L. M., Granillo, M. T., Jorm, A. F., & Paxton, S. J. (2011). Unmet need for treatment in the eating disorders: a systematic review of eating disorder specific treatment seeking among community cases. *Clinical Psychology Review, 31*, 727-735.

Hay, P. J. (2003). Quality of life and bulimic eating disorder behaviors: Findings from a community-based sample. *International Journal of Eating Disorders, 33*, 434-442.

Hay, P. J. (2013). A systematic review of evidence for psychological treatments in eating disorders: 2005–2012. *International Journal of Eating Disorders, 46*, 462-469.

Hay, P. J., Bacaltchuk, J., Stefano, S., & Kashyap, P. (2009). Psychological treatments for bulimia nervosa and binging. *Cochrane Database of Systematic Reviews, 2009*, 1-170.

Hay, P. J., Chinn, D., Forbes, D., Madden, S., Newton, R., Sugenor, L., . . . Ward, W. (2014). Royal Australian and New Zealand college of psychiatrists clinical practice guidelines for the treatment of eating disorders. *Australian and New Zealand Journal of Psychiatry, 48*, 977-1008. doi: 10.1177/0004867414555814

Hay, P. J., Claudino, A., & Kaio, M. H. (2001). Antidepressants versus psychological treatments and their combination for bulimia nervosa. *Cochrane Database of Systematic Reviews*. doi: 10.1002/14651858.CD003385

Hay, P. J., Claudino, A. M., Touyz, S., & Abd Elbaky, G. (2015). Individual psychological therapy in the outpatient treatment of adults with anorexia nervosa. *The Cochrane Library*.

Hay, P. J., & Mond, J. (2005). How to'count the cost'and measure burden? A review of health-related quality of life in people with eating disorders. *Journal of Mental Health, 14*, 539-552. doi: 10.1080/09638230500400274

Hay, P. J., Mond, J., Buttner, P., & Darby, A. (2008). Eating disorder behaviors are increasing: findings from two sequential community surveys in South Australia. *PLoS One, 3*, e1541. doi: 10.1371/journal.pone.0001541

Hayes, A. M., Feldman, G. C., Beevers, C. G., Laurenceau, J.-P., Cardaciotto, L., & Lewis-Smith, J. (2007a). Discontinuities and cognitive changes in an exposure-based cognitive therapy for depression. *Journal of Consulting and Clinical Psychology, 75*, 409-431.

Hayes, A. M., Laurenceau, J.-P., Feldman, G., Strauss, J. L., & Cardaciotto, L. (2007b). Change is not always linear: The study of nonlinear and discontinuous patterns of change in psychotherapy. *Clinical Psychology Review, 27*, 715-723. doi: http://dx.doi.org/10.1016/j.cpr.2007.01.008

Herman, C. P., & Mack, D. (1975). Restrained and unrestrained eating. *Journal of Personality, 43*, 647-660.

Herpertz, S., Hagenah, U., Vocks, S., von Wietersheim, J., Cuntz, U., & Zeeck, A. (2011). The diagnosis and treatment of eating disorders. *Deutsches Ärzteblatt International, 108*, 678.

Higgins, J., & Green, S. (2011a). *Cochrane handbook for systematic reviews of interventions*. United Kingdom, UK: John Wiley & Sons.

Higgins, J., & Green, S. (2011b). *Cochrane handbook for systematic reviews of interventions* (Vol. 4): John Wiley & Sons.

Higgins, J., & Thompson, S. G. (2002). Quantifying heterogeneity in a meta-analysis. *Statistics in Medicine, 21*, 1539-1558.

Hilbert, A., Hoek, H., & Schmidt, R. (2017). Evidence-based clinical guidelines for eating disorders: international comparison. *Current Opinion in Psychiatry*.

Hoiles, K. J., Egan, S. J., & Kane, R. T. (2012). The validity of the transdiagnostic cognitive behavioural model of eating disorders in predicting dietary restraint. *Eat. Behav, 13*, 123-126.

Hu, L., & Bentler, P. M. (1999). Cutoff criteria for fit indexes in covariance structure analysis: Conventional criteria versus new alternatives. *Structural Equation Modeling: A Multidisciplinary Journal, 6*, 1-55.

Hunter, J. P., Saratzis, A., Sutton, A. J., Boucher, R. H., Sayers, R. D., & Bown, M. J. (2014). In meta-analyses of proportion studies, funnel plots were found to be an inaccurate method of assessing publication bias. *Journal of Clinical Epidemiology, 67*, 897-903.

Jacobi, C., Dahme, B., & Dittmann, R. (2002). Cognitive-behavioural, fluoxetine and combined treatment for bulimia nervosa: Short- and long-term results. *European Eating Disorders Review, 10*(3), 179-198. doi: 10.1002/erv.452

Jansen, A. (2016). Eating disorders need more experimental psychopathology. *Behaviour Research and Therapy, 86*, 2-10.

Jin, Y. h., Ma, E. t., Gao, W. j., Hua, W., & Dou, H. y. (2014). Reporting and methodological quality of systematic reviews or meta-analyses in nursing field in China. *International Journal of Nursing Practice, 20*, 70-78.

Kausman, R. (2004). *If not dieting, then what?* NSW, Australia: Allen & Unwin

Kaye, W. H., Bulik, C. M., Thornton, L., Barbarich, N., & Masters, K. (2004). Comorbidity of anxiety disorders with anorexia and bulimia nervosa. *American Journal of Psychiatry, 161*, 2215-2221.

Kazdin, A. E. (2007). Mediators and mechanisms of change in psychotherapy research. *Annual Review of Clinical Psychology, 3*, 1-27. doi: 10.1146/annurev.clinpsy.3.022806.091432

Kazdin, A. E. (2011). *Single-case research designs: Methods for clinical and applied settings*. UK: Oxford University Press.

Kazdin, A. E., & Nock, M. K. (2003). Delineating mechanisms of change in child and adolescent therapy: Methodological issues and research recommendations. *Journal of Child Psychology and Psychiatry, 44*, 1116-1129. doi: 10.1111/1469-7610.00195

Kenardy, J., Mensch, M., Bowen, K., Green, B., & Walton, J. (2002). Group therapy for binge eating in type 2 diabetes: a randomized trial. *Diabetic Medicine, 19*, 234-239.

Keski-Rahkonen, A., Hoek, H., Linna, M., Raevuori, A., Sihvola, E., Bulik, C. M., . . . Kaprio, J. (2009). Incidence and outcomes of bulimia nervosa: a nationwide population-based study. *Psychological Medicine, 39*, 823-831.

Knight, L. J., & Boland, F. J. (1989). Restrained eating: an experimental disentanglement of the disinhibiting variables of perceived calories and food type. *Journal of Abnormal Psychology, 98*, 412-420.

Knopp, J., Knowles, S., Bee, P., Lovell, K., & Bower, P. (2013). A systematic review of predictors and moderators of response to psychological therapies in OCD: Do we have enough empirical evidence to target treatment? *Clinical Psychology Review, 33*, 1067-1081. doi: 10.1016/j.cpr.2013.08.008

Kraemer, H. C. (2016). Messages for Clinicians: Moderators and Mediators of Treatment Outcome in Randomized Clinical Trials. *American Journal of Psychiatry, 173*, 672-679. doi: http://dx.doi.org/10.1176/appi.ajp.2016.15101333

Kraemer, H. C., Wilson, G. T., Fairburn, C. G., & Agras, W. S. (2002). Mediators and moderators of treatment effects in randomized clinical trials. *Archives of General Psychiatry, 59*, 877-883. doi: 10.1001/archpsyc.59.10.877.

Lampard, M. A., Byrne, S. M., McLean, N., & Fursland, A. (2011). An evaluation of the enhanced cognitive-behavioural model of bulimia nervosa. *Behaviour Research and Therapy, 49*, 529-535.

Lampard, M. A., & Sharbanee, J. M. (2015). The cognitive-behavioural theory and treatment of bulimia nervosa: An examination of treatment mechanisms and future directions. *Australian Psychologist, 50*, 6-13. doi: 10.1111/ap.12078

Lampard, M. A., Tasca, G., Balfour, L., & Bissada, H. (2013). An evaluation of the transdiagnostic cognitive-behavioural model of eating disorders. *Eur Eat Disord Rev, 21*, 99-107.

Latner, J. D., Mond, J. M., Vallance, J. K., Gleaves, D. H., & Buckett, G. (2012). Body checking and avoidance in women: Associations with mental and physical health-related quality of life. *Eating behaviors, 13*, 386-389.

Laurenceau, J. P., Hayes, A. M., & Feldman, G. C. (2007). Some methodological and statistical issues in the study of change processes in psychotherapy. *Clinical Psychology Review, 27*, 682-695. doi: 10.1016/j.cpr.2007.01.007

Lavender, J. M., Wonderlich, S. A., Crosby, R. D., Engel, S. G., Mitchell, J. E., Crow, S., . . . Le Grange, D. (2013). A naturalistic examination of body checking and dietary restriction in women with anorexia nervosa. *Behaviour Research and Therapy, 51*, 507-511.

Legenbauer, T., Martin, F., Blaschke, A., Schwenzfeier, A., Blechert, J., & Schnicker, K. (2017). Two sides of the same coin? A new instrument to assess body checking and avoidance behaviors in eating disorders. *Body Image, 21*, 39-46. doi: https://doi.org/10.1016/j.bodyim.2017.02.004

Lemmens, L. H., Müller, V. N., Arntz, A., & Huibers, M. J. (2016). Mechanisms of change in psychotherapy for depression: An empirical update and evaluation of research aimed at identifying psychological mediators. *Clinical Psychology Review, 50*, 95-107.

Linardon, J. (2016). Correlates of the over-evaluation of weight and shape in binge eating disorder and mixed eating disorder samples: A meta-analytic review. *Eating Disorders, 25*, 183-198. doi: 10.1080/10640266.2016.1260374

Linardon, J., & Brennan, L. (2017). The effects of cognitive-behavioral therapy for eating disorders on quality of life: A meta-analysis. *International Journal of Eating Disorders, 50*, 715–730. doi: 10.1002/eat.22719

Linardon, J., Brennan, L., & de la Piedad Garcia, X. (2016a). Rapid response to eating disorder treatment: A systematic review and meta-analysis. *International Journal of Eating Disorders, 49*, 905-919.

Linardon, J., de la Piedad Garcia, X., & Brennan, L. (2016b). Predictors, moderators and mediators of treatment outcome following manualised cognitive-behavioural therapy for eating disorders: A systematic review. *European Eating Disorders Review, 25*, 3-12.

Linardon, J., Fairburn, C. G., Fitzsimmons-Craft, E. E., Wilfley, D. E., & Brennan, L. (2017). The empirical status of the third-wave behaviour therapies for the treatment of eating disorders: A systematic review. *Clinical Psychology Review*.

Linardon, J., & Mitchell, S. (2017). Rigid dietary control, flexible dietary control, and intuitive eating: Evidence for their differential relationship to disordered eating and body image concerns. *Eating behaviors*, *26*, 16-22. doi: http://dx.doi.org/10.1016/j.eatbeh.2017.01.008

Linardon, J., Wade, T., De la Piedad Garcia, X., & Brennan, L. (in press). The efficacy of cognitive-behavioral therapy for eating disorders: A systematic review and meta-analysis *Journal of Consulting and Clinical Psychology*. doi: http://dx.doi.org/10.1037/ccp0000245.supp

Linardon, J., & Wade, T. D. (2018). How many individuals achieve symptom abstinence following psychological treatments for bulimia nervosa? A meta-analytic review. *International Journal of Eating Disorders*.

Lipsey, M. W., & Wilson, D. (2001). *Practical meta-analysis*. United Kingdom, UK: Sage Publications.

Lorenzo-Luaces, L., German, R. E., & DeRubeis, R. J. (2014). It's complicated: The relation between cognitive change procedures, cognitive change, and symptom change in cognitive therapy for depression. *Clinical Psychology Review*, *41*, 3-15.

Loucas, C. E., Fairburn, C. G., Whittington, C., Pennant, M. E., Stockton, S., & Kendall, T. (2014). E-therapy in the treatment and prevention of eating disorders: A systematic review and meta-analysis. *Behaviour Research and Therapy, 63,* 122-131. doi: 10.1016/j.brat.2014.09.011

Lundervold, D. A., & Belwood, M. F. (2000). The best kept secret in counseling: Single-case (N= 1) experimental designs. *Journal of Counseling and Development:, 78*, 92-102. doi: 10.1002/j.1556-6676.2000.tb02565.x

Lyman, G. H., & Kuderer, N. M. (2005). The strengths and limitations of meta-analyses based on aggregate data. *BMC Medical Research Methodology, 5*, 14-21.

Lynch, F. L., Striegel-Moore, R. H., Dickerson, J. F., Perrin, N., DeBar, L., Wilson, G. T., & Kraemer, H. C. (2010). Cost-effectiveness of guided self-help treatment for recurrent binge eating. *Journal of Consulting and Clinical Psychology, 78*, 322-333.

MacDonald, D. E., McFarlane, T., Dionne, M., David, L., & Olmsted, M. (2017). Rapid response to intensive treatment for bulimia nervosa and purging disorder: A randomized controlled trial of a CBT intervention to facilitate early behavior change. *Journal of Consulting and Clinical Psychology, 85*, 896-908.

MacDonald, D. E., Trottier, K., McFarlane, T., & Olmsted, M. P. (2015). Empirically defining rapid response to intensive treatment to maximize prognostic utility for bulimia nervosa and purging disorder. *Behavior Research and Therapy, 68*, 48-53. doi: 10.1016/j.brat.2015.03.007

McIntosh, V. V. W., Jordan, J., Carter, F. A., Luty, S. E., McKenzie, J. M., Bulik, C. M., . . . Joyce, P. R. (2005). Three psychotherapies for anorexia nervosa: A randomized, controlled trial. *The American Journal of Psychiatry, 162*, 741-747.

McIntosh, V. V. W., Jordan, J., Carter, J. D., Frampton, C. M. A., McKenzie, J. M., Latner, J. D., & Joyce, P. R. (2016). Psychotherapy for transdiagnostic binge eating: A randomized controlled trial of cognitive-behavioural therapy, appetite-focused cognitive-behavioural therapy, and schema therapy. *Psychiatry research, 240*, 412-420.

Messer, S. B., & Wampold, B. E. (2002). Let's face facts: Common factors are more potent than specific therapy ingredients. *Clinical Psychology: Science and Practice, 9*, 21-25.

Mitchell, J. E., Fletcher, L., Hanson, K., Mussell, M. P., Seim, H., Crosby, R., & Al-Banna, M. (2001). The relative efficacy of fluoxetine and manual-based self-help in the treatment of outpatients with bulimia nervosa. *Journal of Clinical Psychopharmacology, 21*(3), 298-304.

Moher, D., Cook, D. J., Eastwood, S., Olkin, I., Rennie, D., & Stroup, D. F. (2000). Improving the quality of reports of meta-analyses of randomised controlled trials: the QUOROM statement. *Revista Española de Salud Pública, 74*, 00-00.

Moher, D., Liberati, A., Tetzlaff, J., & Altman, D. G. (2009). Preferred reporting items for systematic reviews and meta-analyses: The PRISMA statement. *Annals of Internal Medicine, 151*, 264-269.

Mond, J., Hay, P. J., Rodgers, B., & Owen, C. (2006). Eating disorder examination questionnaire (EDE-Q): norms for young adult women. *Behaviour Research and Therapy, 44*, 53-62. doi: 10.1016/j.brat.2004.12.003

Mountford, V., Haase, A., & Waller, G. (2006). Body checking in the eating disorders: Associations between cognitions and behaviors. *Int J Eat Disord, 39*, 708-715.

Munsch, S., Biedert, E., Meyer, A., Michael, T., Schlup, B., Tuch, A., & Margraf, J. (2007). A randomized comparison of cognitive behavioral therapy and behavioral weight loss treatment for overweight individuals with binge eating disorder. *International Journal of Eating Disorders, 40*, 102-113.

Murphy, R., Cooper, Z., Hollon, S. D., & Fairburn, C. G. (2009). How do psychological treatments work? Investigating mediators of change. *Behaviour Research and Therapy, 47*, 1-5. doi: 10.1016/j.brat.2008.10.001

Muthén, L. K., & Muthén, B. O. (2010). *Mplus user's guide: Statistical analysis with latent variables*. Los Angeles: Muthén & Muthén.

National Institute of Clinical Excellence. (2017). *Eating disorders: Recognition and treatment*. Available at: www.nice.org.uk/guidance/ng69.

Nauta, H., Hospers, H., Kok, G., & Jansen, A. (2000). A comparison between a cognitive and a behavioral treatment for obese binge eaters and obese non-binge eaters. *Behavior Therapy, 31*, 441-461.

Nilsen, T. S., Eisemann, M., & Kvernmo, S. (2013). Predictors and moderators of outcome in child and adolescent anxiety and depression: A systematic review of psychological treatment studies. *European Child & Adolescent Psychiatry, 22*, 69-87. doi: 10.1007/s00787-012-0316-3

Olmsted, M. P., Kaplan, A. S., & Rockert, W. (2005). Defining remission and relapse in bulimia nervosa. *International Journal of Eating Disorders, 38*, 1-6.

Olmsted, M. P., MacDonald, D. E., McFarlane, T., Trottier, K., & Colton, P. (2015). Predictors of rapid relapse in bulimia nervosa. *International Journal of Eating Disorders, 48*, 337-340.

Palascha, A., van Kleef, E., & van Trijp, H. C. (2015). How does thinking in Black and White terms relate to eating behavior and weight regain? *Journal of Health Psychology, 20*, 638-648.

Pennesi, J., & Wade, T. D. (2016). A systematic review of the existing models of disordered eating: Do they inform the development of effective interventions? *Clinical Psychology Review, 43*, 175-192. doi: https://doi.org/10.1016/j.cpr.2015.12.004

Peterson, C. B., Mitchell, J. E., Crow, S. J., Crosby, R. D., & Wonderlich, S. A. (2009). The efficacy of self-help group treatment and therapist-led group treatment for binge eating disorder. *The American Journal of Psychiatry, 166*, 1347-1354. doi: 10.1176/appi.ajp.2009.09030345

Peterson, C. B., Mitchell, J. E., Engbloom, S., Nugent, S., Mussell, M. P., & Miller, J. P. (1998). Group cognitive-behavioral treatment of binge eating disorder: A comparison of therapist-led versus self-help formats. *International Journal of Eating Disorders, 24*, 125-136.

Petticrew, M. (2003). Why certain systematic reviews reach uncertain conclusions. *BMJ, 326*, 756-758.

Pike, K. M., Loeb, K., & Vitousek, K. (2001). *Cognitive-behavioral therapy for anorexia nervosa and bulimia nervosa*. UK: Wiley and Sons.

Pike, K. M., Walsh, B. T., Vitousek, K., Wilson, G. T., & Bauer, J. (2003). Cognitive behavior therapy in the posthospitalization treatment of anorexia nervosa. *American Journal of Psychiatry, 160*, 2046-2049.

Polnay, A., James, V., Hodges, L., Murray, G., Munro, C., & Lawrie, S. (2014). Group therapy for people with bulimia nervosa: systematic review and meta-analysis. *Psychological Medicine, 44*, 2241-2254.

Porter, E., & Chambless, D. L. (2015). A systematic review of predictors and moderators of improvement in cognitive-behavioral therapy for panic disorder and agoraphobia. *Clinical Psychology Review, 42*, 179-192.

Poulsen, S., Lunn, S., Daniel, S. I., Folke, S., Mathiesen, B. B., Katznelson, H., & Fairburn, C. G. (2014). A randomized controlled trial of psychoanalytic psychotherapy or cognitive-behavioral therapy for bulimia nervosa. *American Journal of Psychiatry, 171*, 109-116.

Preacher, K. J., & Hayes, A. F. (2008). Asymptotic and resampling strategies for assessing and comparing indirect effects in multiple mediator models. *Behavior Research Methods, 40*, 879-891.

Reas, D. L., Grilo, C. M., Masheb, R. M., & Wilson, G. T. (2005). Body checking and avoidance in overweight patients with binge eating disorder. *International Journal of Eating Disorders, 37*, 342-346.

Reas, D. L., Whisenhunt, B. L., Netemeyer, R., & Williamson, D. A. (2002). Development of the body checking questionnaire: A self-report measure of body checking behaviors. *International Journal of Eating Disorders, 31*, 324-333.

Renaud, J., Brent, D. A., Baugher, M., Birmaher, B., Kolko, D. J., & Bridge, J. (1998). Rapid response to psychosocial treatment for adolescent depression: a two-year follow-up. *Journal of the American Academy of Child & Adolescent Psychiatry, 37*, 1184-1190.

Robinson, P., & Serfaty, M. (2008). Getting better byte by byte: a pilot randomised controlled trial of email therapy for bulimia nervosa and binge eating disorder. *European Eating Disorders Review, 16*, 84-93.

Rohde, P., Stice, E., & Gau, J. M. (2016). Predicting persistence of eating disorder compensatory weight control behaviors. *International Journal of Eating Disorders, 50*, 561–568.

Rosen, J. C., & Ramirez, E. (1998). A comparison of eating disorders and body dysmorphic disorder on body image and psychological adjustment. *Journal of Psychosomatic Research, 44*, 441-449.

Rosen, J. C., Srebnik, D., Saltzberg, E., & Wendt, S. (1991). Development of a body image avoidance questionnaire. *J. Consult. Clin. Psychol., 3*, 32-37.

Rosenthal, R. (1991). *Meta-analytic procedures for social research*. London, United Kingdom: Sage Publications.

Sacks, H. S., Berrier, J., Reitman, D., Ancona-Berk, V., & Chalmers, T. C. (1987). Meta-analyses of randomized controlled trials. *New England Journal of Medicine, 316*, 450-455.

Satorra, A., & Bentler, P. M. (2001). A scaled difference chi-square test statistic for moment structure analysis. *Psychometrika, 66*, 507-514.

Schneider, R. L., Arch, J. J., & Wolitzky-Taylor, K. B. (2015). The state of personalized treatment for anxiety disorders: A systematic review of treatment moderators. *Clinical Psychology Review, 38*, 39-54. doi: 10.1016/j.cpr.2015.02.004

Schnitzler, C. E., von Ranson, K. M., & Wallace, L. M. (2012). Adding thin-ideal internalization and impulsiveness to the cognitive–behavioral model of bulimic symptoms. *Eat. Behav, 13*, 219-225.

Serfaty, M. A., Turkington, D., Heap, M., Ledsham, L., & Jolley, E. (1999). Cognitive therapy versus dietary counselling in the outpatient treatment of anorexia nervosa: Effects of the treatment phase. *European Eating Disorders Review, 7*, 334-350.

Shafran, R., Clark, D., Fairburn, C., Arntz, A., Barlow, D., Ehlers, A., . . . Ost, L. (2009). Mind the gap: Improving the dissemination of CBT. *Behaviour Research and Therapy, 47,* 902-909.

Shafran, R., Fairburn, C. G., Robinson, P., & Lask, B. (2004). Body checking and its avoidance in eating disorders. *Int J Eat Disord, 35,* 93-101.

Shapiro, J. R., Berkman, N. D., Brownley, K. A., Sedway, J. A., Lohr, K. N., & Bulik, C. M. (2007). Bulimia nervosa treatment: A systematic review of randomized controlled trials. *International Journal of Eating Disorders, 40,* 321-336. doi: 10.1002/eat.20372

Smits, J. A., Julian, K., Rosenfield, D., & Powers, M. B. (2012). Threat reappraisal as a mediator of symptom change in cognitive-behavioral treatment of anxiety disorders: A systematic review. *Journal of Consulting and Clinical Psychology, 80*(4), 624.

Spangler, D. L., Baldwin, S. A., & Agras, W. S. (2004). An examination of the mechanisms of action in cognitive behavioral therapy for bulimia nervosa. *Behavior Therapy, 35,* 537-560. doi: 10.1016/S0005-7894(04)80031-5

Spielmans, G. I., Benish, S. G., Marin, C., Bowman, W. M., Menster, M., & Wheeler, A. J. (2013). Specificity of psychological treatments for bulimia nervosa and binge eating disorder? A meta-analysis of direct comparisons. *Clinical Psychology Review, 33,* 460-469.

Spitzer, R. L., Devlin, M., Walsh, B. T., Hasin, D., Wing, R., Marcus, M., . . . Agras, S. (1992). Binge eating disorder: a multisite field trial of the diagnostic criteria. *International Journal of Eating Disorders, 11,* 191-203.

Steinhausen, H. C., & Weber, S. (2009). The outcome of bulimia nervosa: Findings from one-quarter century of research. *The American Journal of Psychiatry, 166,* 1331-1341. doi: 10.1176/appi.ajp.2009.09040582

Stice, E. (2002). Risk and maintenance factors for eating pathology: a meta-analytic review. *Psychol Bull., 128*, 825-848. doi: http://dx.doi.org/10.1037/0033-2909.128.5.825

Stice, E., & Agras, W. S. (1998). Predicting onset and cessation of bulimic behaviors during adolescence: A longitudinal grouping analysis. *Behavior Therapy, 29*, 257-276.

Stice, E., Nemeroff, C., & Shaw, H. E. (1996). Test of the dual pathway model of bulimia nervosa: Evidence for dietary restraint and affect regulation mechanisms. *Journal of Social and Clinical Psychology, 15*, 340-363.

Striegel-Moore, R. H., Wilson, G. T., DeBar, L., Perrin, N., Lynch, F., Rosselli, F., & Kraemer, H. C. (2010). Cognitive behavioral guided self-help for the treatment of recurrent binge eating. *Journal of Consulting and Clinical Psychology, 78*, 312-321.

Sundgot-Borgen, J., Rosenvinge, J. H., Bahr, R., & Schneider, L. S. (2002). The effect of exercise, cognitive therapy, and nutritional counseling in treating bulimia nervosa. *Medicine and Science in Sports and Exercise*, *34*, 190-195.

Tang, T. Z., & DeRubeis, R. J. (1999). Sudden gains and critical sessions in cognitive-behavioral therapy for depression. *Journal of Consulting and Clinical Psychology, 67*, 894-904.

Tatham, M., Turner, H., Mountford, V. A., Tritt, A., Dyas, R., & Waller, G. (2015). Development, psychometric properties and preliminary clinical validation of a brief, session-by-session measure of eating disorder cognitions and behaviors: The ED-15. *International Journal of Eating Disorders*, *48*, 1005–1015.

Telch, C. F., Agras, W. S., & Linehan, M. M. (2001). Dialectical behavior therapy for binge eating disorder. *J. Consult. Clin. Psychol.*, *69*(6), 1061-1065. doi: =

Telch, C. F., Agras, W. S., Rossiter, E. M., Wilfley, D., & Kenardy, J. (1990). Group cognitive-behavioral treatment for the nonpurging bulimic: An initial evaluation. *Journal of Consulting and Clinical Psychology, 58*, 629-635.

Thackwray, D. E., Smith, M. C., Bodfish, J. W., & Meyers, A. W. (1993). A comparison of behavioral and cognitive-behavioral interventions for bulimia nervosa. *Journal of Consulting and Clinical Psychology, 61*(4), 639-645. doi: 10.1037/0022-006X.61.4.639

Timmerman, G. M. (1999). Binge eating scale: further assessment of validity and reliability. *J Appl Biobehav Res., 4*, 1-12. doi: 10.1111/j.1751-9861.1999.tb00051.x

Tolin, D. F. (2010). Is cognitive–behavioral therapy more effective than other therapies?: A meta-analytic review. *Clinical Psychology Review, 30*, 710-720.

Touyz, S., Le Grange, D., Lacey, H., Hay, P., Smith, R., Maguire, S., . . . Crosby, R. D. (2013). Treating severe and enduring anorexia nervosa: a randomized controlled trial. *Psychological Medicine, 43*, 2501-2511.

Touyz, S., Polivy, J., & Hay, P. (2008). *Eating disorders: Psychological Therapies and Behavioural Medicine Evidence-Based Practice*. Australia.

Traviss-Turner, G. D., West, R. M., & Hill, A. J. (2017). Guided Self-help for Eating Disorders: A Systematic Review and Metaregression. *European Eating Disorders Review, 25*, 148-164. doi: 10.1002/erv.2507

Tunis, A. S., McInnes, M. D., Hanna, R., & Esmail, K. (2013). Association of study quality with completeness of reporting: have completeness of reporting and quality of systematic reviews and meta-analyses in major radiology journals changed since publication of the PRISMA statement? *Radiology, 269*, 413-426.

Vall, E., & Wade, T. D. (2015). Predictors of treatment outcome in individuals with eating disorders: A systematic review and meta-analysis. *International Journal of Eating Disorders, 48*, 946–971. doi: 10.1002/eat.22411

Van de Mortel, T. F. (2008). Faking it: social desirability response bias in self-report research. *Australian Journal of Advanced Nursing, 25*, 40-48.

van der Velden, A. M., Kuyken, W., Wattar, U., Crane, C., Pallesen, K. J., Dahlgaard, J., . . . Piet, J. (2015). A Systematic Review of Mechanisms of Change in Mindfulness-Based Cognitive Therapy in the Treatment of Recurrent Major Depressive Disorder. *Clinical Psychology Review, 37*, 26-39.

Vervliet, B., & Raes, F. (2013). Criteria of validity in experimental psychopathology: application to models of anxiety and depression. *Psychological Medicine, 43*, 2241-2244.

Von Ranson, K. M., Wallace, L. M., & Stevenson, A. (2013). Psychotherapies provided for eating disorders by community clinicians: infrequent use of evidence-based treatment. *Psychotherapy Research, 23*, 333-343.

Wade, T., Zhu, G., & Martin, N. (2011). Undue influence of weight and shape: is it distinct from body dissatisfaction and concern about weight and shape? *Psychological Medicine, 41*, 819-828.

Wagner, B., Nagl, M., Dölemeyer, R., Klinitzke, G., Steinig, J., Hilbert, A., & Kersting, A. (2016). Randomized controlled trial of an internet-based cognitive-behavioral treatment program for binge-eating disorder. *Behavior Therapy, 47*, 500-514.

Walsh, B. T., Wilson, G. T., Loeb, K. L., Devlin, M. J., Pike, K. M., Roose, S. P., . . . Waternaux, C. (1997). Medication and psychotherapy in the treatment of bulimia nervosa. *The American Journal of Psychiatry, 154*, 523-531.

Watson, H., & Bulik, C. M. (2013). Update on the treatment of anorexia nervosa: review of clinical trials, practice guidelines and emerging interventions. *Psychological Medicine, 43*, 2477-2500.

Welch, E., Birgegård, A., Parling, T., & Ghaderi, A. (2011). Eating disorder examination questionnaire and clinical impairment assessment questionnaire: general population and clinical norms for young adult women in Sweden. *Behav Res Ther., 49*(2), 85-91.

Wilfley, D. E., Agras, W. S., Telch, C. F., Rossiter, E. M., Schneider, J. A., Cole, A. G., . . . Raeburn, S. D. (1993). Group cognitive-behavioral therapy and group interpersonal psychotherapy for the nonpurging bulimic individual: a controlled comparison. *Journal of Consulting and Clinical Psychology, 61*, 296-305.

Wilfley, D. E., Welch, R. R., Stein, R. I., Spurrell, E. B., Cohen, L. R., Saelens, B. E., . . . Matt, G. E. (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, S. E., Watts, T. K. O., & Wade, T. D. (2012). A review of the definitions of outcome used in the treatment of bulimia nervosa. *Clinical Psychology Review, 32*, 292-300. doi: 10.1016/j.cpr.2012.01.006

Wilson, G. T., Fairburn, C. C., Agras, W. S., Walsh, B. T., & Kraemer, H. (2002). Cognitive-behavioral therapy for bulimia nervosa: Time course and mechanisms of change. *Journal of Consulting and Clinical Psychology, 70*, 267.

Wilson, G. T., Wilfley, D. E., Agras, W. S., & Bryson, S. (2010). Psychological treatments of binge eating disorder. *Archives of General Psychiatry, 67*, 94-101. doi: 10.1001/archgenpsychiatry.2009.170.

Wilson, G. T., & Zandberg, L. (2012). Cognitive–behavioral guided self-help for eating disorders: Effectiveness and scalability. *Clinical Psychology Review, 32*, 343-357.

Wolf, E. M., & Crowther, J. H. (1992). An evaluation of behavioral and cognitive-behavioral group interventions for the treatment of bulimia nervosa in women. *International Journal of Eating Disorders, 11*, 3-15.

Wonderlich, S., Peterson, C., Crosby, R., Smith, T., Klein, M., Mitchell, J. E., & Crow, S. J. (2014). A randomized controlled comparison of integrative cognitive-affective

therapy (ICAT) and enhanced cognitive-behavioral therapy (CBT-E) for bulimia nervosa. *Psychological Medicine, 44*, 543-553.

Zendegui, E. A., West, J. A., & Zandberg, L. J. (2014). Binge eating frequency and regular eating adherence: The role of eating pattern in cognitive behavioral guided self-help. *Eating behaviors, 15*, 241-243. doi: 10.1016/j.eatbeh.2014.03.002

Zipfel, S., Wild, B., Groß, G., Friederich, H.-C., Teufel, M., Schellberg, D., . . . Herpertz, S. (2014). Focal psychodynamic therapy, cognitive behaviour therapy, and optimised treatment as usual in outpatients with anorexia nervosa (ANTOP study): Randomised controlled trial. *The Lancet, 383*, 127-137.

Zvolensky, M. J., Lejuez, C., Stuart, G. L., & Curtin, J. J. (2001). Experimental psychopathology in psychological science. *Review of General Psychology, 5*, 371-381.

Appendix A: Proof of publication or acceptance to journal for Study 1

Date:	07/18/2017
To:	"Jake Linardon" jake.linardon@acu.edu.au
cc:	tracey.wade@flinders.edu.au, xochitl.delapiedadgarcia@acu.edu.au, leah.brennan@acu.edu.au
From:	"Journal of Consulting and Clinical Psychology" jvaccaro@apa.org
Subject:	Your Submission CCP-2017-0640R1

CCP-2017-0640R1
The efficacy of cognitive-behavioral therapy for eating disorders: A systematic review and meta-analysis.
Journal of Consulting and Clinical Psychology

Dear Mr Linardon,

I have now had the opportunity to review your revised manuscript "The efficacy of cognitive-behavioral therapy for eating disorders: A systematic review and meta-analysis.." It is with great pleasure that I am able to inform you that I am accepting your article for publication in a future volume of *Journal of Consulting and Clinical Psychology*. I want to personally thank you for being so responsive to reviewer comments.

APA has a number of required publication forms, regarding copyright, conflict of interest, and other matters. You will receive an email shortly from DocuSign, requesting electronic signatures for publication forms. These forms must be signed by all authors prior to your manuscript entering production. If you

have questions about this process, please contact manuscript coordinator, Jenna Vaccaro, at jvaccaro@apa.org

Also, please note that APA journal articles will now be published online in the PsycARTICLES database prior to appearing in print issues. Once your manuscript has been sent to the APA Journals production office (pending the receipt of your publication forms), you will receive an e-mail notification within 2 to 4 weeks that a typeset proof of your article is available for you to review. You will be asked to submit any corrections you may have and answers to any queries from the production office within 48 hours. All corrections will be incorporated by production staff. Assuming you submit all the necessary paperwork and review and correct your proofs at the time you receive them, your article could by published Online First in as little as 30 days. Once you have submitted corrections to your article and it has been released as Online First, you will not have the option of making additional corrections prior to the publication of the final version. Articles appearing Online First are considered published, so any textual changes that may need to be made to an article once it has appeared online will require a formal correction notice/errata. The only difference between the Online First version of your article and the one that appears in print will be the addition of volume, issue, and page numbers."

Thank you for allowing us to review your work. I look forward to seeing this paper in print.

Best Regards,

Eric Stice
Associate Editor
Journal of Consulting and Clinical Psychology

Appendix B: Research Output from Candidature

Peer-Reviewed Academic Publications

Linardon, J., Brennan, L., & de la Piedad Garcia, X. (2016). Rapid response to eating disorder treatment: A systematic review and meta-analysis. *International Journal of Eating Disorders, 49,* 905-919: doi: 10.1002/eat.22595 [SCImago = Q1, ISI Impact Factor = 3.567]

Linardon, J., de la PieDad Garcia, X., & Brennan, L. (2017). Predictors, moderators and mediators of treatment outcome following manualized cognitive-behavioural therapy for eating disorders: A systematic review. *European Eating Disorders Review. 25*, 3-12. doi: 10.1002/erv.2492. [SCImago = Q1, ISI Impact Factor = 3.391]

Linardon, J., Wade, T., de la Piedad Garcia, X., & Brennan, L (in press): Psychotherapy for bulimia nervosa on symptoms of depression: A meta-analysis of randomized controlled trials. *International journal of eating disorders:* doi: 10.1002/eat.22763 [SCImago = Q1, ISI Impact Factor = 3.567]

Linardon, J., Wade, T., de la Piedad Garcia, X., & Brennan, L (in press). The efficacy of cognitive-behavioural therapy for eating disorders: A systematic review and meta-analysis. *Journal of Consulting and Clinical Psychology* [SCImago = Q1, ISI Impact Factor = 4.593]

Linardon, J., & Brennan, L. (2017). The effects of cognitive-behavioural therapy for eating disorders on quality of life: A meta-analysis. *International Journal of Eating Disorders, 50*, 715-730. Doi: 10.1002/eat.22719. [SCImago = Q1, ISI Impact Factor = 3.567]

Linardon, J., Mitchell, S (2017). Flexible control, rigid control and intuitive eating. Evidence for their differential relationship to disordered eating and body image concerns. *Eating Behaviors 26,* 16-22. (SCImago Q1; ISI Impact factor = 2.258)

Linardon, J. (2017). Correlates of the over-evaluation of weight and shape in binge eating disorder and mixed eating disorder samples: A meta-analytic review. *Eating Disorders, 25*, 183-198. [SCImago = Q2; ISI Impact Factor = 1.484]

Brennan, L Teede, H, Skouteris, H, **Linardon, J**, Hill, B & Moran, L (in press). Lifestyle and Behavioural Management of Polycystic Ovary Syndrome. *Journal of Women's Health* [SCImago = Q1, ISI Impact Factor = 2.032]

Brennan, L., Mitchell, S., & **Linardon, J.** (2016) Feeding and Eating Disorders. Abnormal psychology in context: The Australian and New Zealand Handbook. Cambridge University Press.

Research Papers Under Review/Submitted for Publication

Linardon, J, Fairburn, CG., Wilfley, D., Fitzimmons-Craft, E., & Brennan, L: The empirical status of the third-wave behaviour therapies for the treatment of eating disorders: A systematic review and meta-analysis.

Linardon J., Fitzimmons-Craft, E., & Wilfley D. Dropout from interpersonal psychotherapy for mental health disorders: A meta-analysis of randomized controlled trials.

Linardon, J, Phillapou, Castle, D., Newton, R., Harrison, P., Cistullo, L., Griffiths, S., A, Hindle, & Brennan, L. Testing the relative contributions of over-evaluation, fear of weight gain, and feeling fat on psychopathology in anorexia nervosa and bulimia nervosa:

Linardon, J, Phillapou, Castle, D., Newton, R., Harrison, P., Cistullo, L., Griffiths, S., A, Hindle, & Brennan, L: The relative associations of shape and weight over-evaluation, preoccupation, and dissatisfaction on measures of eating disorder and general psychopathology: An extension study in individuals with anorexia nervosa:

Linardon, J. Meta-analysis on the efficacy of cognitive-behavioural therapy for reducing the core eating disorder maintaining mechanisms: Implications for mechanisms of therapeutic change.

Linardon, J., Hindle, A, & Brennan, L: Dropout from cognitive-behavioural therapy for eating disorders: A meta-analysis of randomized controlled trials.

Linardon, J., Braithwaite, R, Cousins, R., & Brennan, L. The relationship between symptoms of social anxiety and disordered eating: The mediating role of appearance-based rejection sensitivity.

Conference Presentations

Linardon, J., Wade, T., de la Piedad Garcia, X., & Brennan, L (August 2017): The empirical status of cognitive-behavioural therapy for eating disorders: Talk

presented at the Australian New Zealand Academy for Eating Disorders conference, Sydney, Australia.

Brennan, L & **Linardon J** (August 2017): Are third-wave behaviour therapies empirically supported eating disorder treatments? Talk presented at the Australian New Zealand Academy for Eating Disorders conference, Sydney, Australia.

Kotsikas, J., Freijah., I., **Linardon, J** & de la Piedad Garcia, X (August 2017): Psychosocial correlates of body checking and body avoidance: A systematic review and meta-analysis. Poster presented at the Australian New Zealand Academy for Eating Disorders conference, Sydney, Australia.

Kotsikas, J., Freijah., I., **Linardon, J** & de la Piedad Garcia, X (August 2017): The psychological effects of weight checking: An experimental analysis. Poster presented at the Australian New Zealand Academy for Eating Disorders conference, Sydney, Australia.

Linardon, J (September 2016)**:** The cognitive-behavioural theory and treatment of eating disorders Talk presented at Australian Catholic University conference.

Linardon, J (September 2015): A systematic review of predictors, moderators and mediators of treatment outcome following CBT for eating disorders. Talk presented at Australian Catholic University conference

Linardon, J (August 2015): Cogntive-behavioural treatment for eating disorders: Three minute thesis competition at Australian Catholic University.

Appendix C: Supplementary material for Study 1.

Table 1
Previous Meta-Analyses of Cognitive-Behavioural Therapy for Eating Disorders

Study	ED	Comparisons	Key Findings
Lewandowski et al. (1997)	BN	1. TL CBT vs any comparison at EOT (k=13) 2. Pre-post symptom change (k=17)	CBT was significantly more efficacious than comparisons on *behavioural* (i.e., any behavioural symptom; r=.64) and *cognitive* outcomes (i.e., any eating disorder inventory; r=.64). Large pre=post improvements in *behavioural* (r=.74) and *cognitive* symptoms (r=.69) were observed.
Ghaderi & Andersson (1999)	BN	1. TL CBT vs any comparison at EOT (k=5) 2. Pre-post symptom change (k=7)	CBT was more efficacious than comparisons at reducing *binge eating* (d=0.47) and *purging* (d=0.58) Large pre-post improvements in *binge eating* (d=1.32) and *purging* (d=1.50) were observed.
Whittal et al. (1999)	BN	Pre-post symptom change (k=24)	Large pre-post improvements in *binge eating* (d=1.28) and *purging* (d=1.22) and *eating attitudes* (i.e., an aggregate of restraint and shape and weight concerns; d=1.35) were observed.
Thompson-Brenner et al. (2003)	BN	Individual/group CBT vs inactive control (k=5)	CBT was significantly more efficacious than inactive controls at reducing *binge eating* (d=0.52) and *purging* (d=0.79) at post-treatment.
Hay et al. (2001)	BN	TL CBT vs antidepressants (k=5)	No difference in *remission rates* (RR=1.26) was found.
Hay et al. (2009)	BN, BED OSF ED	1. TL CBT vs wait-list (k=12) 2. TL CBT vs any psychotherapy(k=15) 3. TL CBT vs a component of CBT (BT; k= 4)	CBT was significantly superior to wait-lists at producing *remission* from binge eating (RR= 0.69) and reducing *bulimic frequencies* (d=0.94) at post-treatment. CBT was significantly superior to active comparisons at reducing *bulimic frequencies* (d=0.21) at post-treatment. No significant difference between CBT and active comparisons on *remission rates* (RR=0.87) was observed. *Remission* rates were higher for CBT over behaviour therapy (RR=0.67).
Vocks et al. (2010)	BED	1. TL CBT vs wait-list (k= 7) 2. CBTgsh vs wait-list (k=4)	CBT was significantly superior to wait-lists on remission rates (OR=6.83) and on reducing *binge eating frequencies* (d=0.82), *eating concerns* (d=0.98) and *weight concerns* (d=0.53). No significant differences on shape concerns (d=0.35) and *dietary restraint* (d=0.32) were found.

Table 1
Previous Meta-Analyses of Cognitive-Behavioural Therapy for Eating Disorders

Study	ED	Comparisons	Key Findings
			CBTgsh was superior to wait-lists on producing *remission rates* (OR= 25.77) and on reducing *binge eating frequencies* (d=0.84), *dietary restraint* (0.68), *shape concerns* (d=0.66), *weight concerns* (d=0.85), and *eating concerns* (d=0.85).
Spielmans et al. (2013)	BN, BED	1. CBT (TL or GSH) vs bona-fide non CBT (k= 9) 2. CBT vs BT (k=12)	CBT was significantly more efficacious than bona-fide non-CBTs at EOT (d= 0.27); however, this difference was not significant at follow-up (d= 0.19) There was no significant difference between CBT and BT at EOT (d= 0.05) or follow-up (0.17).
Loucas et al. (2014)	BN, BED OSFED	1. E-therapy CBT for BN vs wait-list (k=3) 2. E-therapy CBT for BN vs bibliotherapy (k=2) 3. E-therapy CBT for BED vs wait-list (k=2)	E-CBT for BN was more efficacious than wait-lists at EOTon *binge eating* (d=-0.44), *vomiting* (d=0.43), and *cessation of bulimic behaviours* (RR=1.94). No EOT differences were observed for *global ED symptoms, weight concern, shape concern and dietary restraint.* At follow-up, E-CBT for BN was more efficacious on all outcomes (d's >0.50) No differences at EOT (d's >0.03) or follow-up (d's >0.04) between E-CBT for BN to bibliotherapy. No differences at EOT (d's >0.07) or follow-up (d >0.08) between E-CBT for BED and wait-lists.
Polnay et al. (2014)	BN	Group CBT vs no treatment (k=5)	CBT outperformed wait-lists on bulimic frequencies at post-treatment (d=0.56).
Hay et al. (2015)	AN	1. TL CBT vs TAU (k=2) 2. TL CBT vs IPT or FPDT (k=2)	No EOT and follow-up differences in *body weight* (MD= 0.91 and 0.02), *recovery* (RR=0.97 and 1.20), and *eating disorder symptom scores* (d= 0.05 and -0.23) between CBT and TAU. No EOT and follow-up differences in *weight* (MD= 0.41 and -0.19), *recovery* (RR= 0.80 and 0.54), and *eating disorder symptoms* (d= -0.33 and 0.07) between CBT and IPT/FPDT.
Brownley et al. (2016)	BED	TL CBT vs wait-list (k=3)	CBT was more efficacious for producing abstinence than wait-lists at post-treatment (RR= 4.95).
Cuijpers et al. (2016)	BN, BED	TL CBT vs TL IPT (k=6)	CBT was significantly more efficacious than IPT for reducing EOT *behavioural symptoms* (d=0.20).

Note: BED= binge eating disorder; BN= Bulimia nervosa; OSFED; other specified feeding or eating disorder; AN= anorexia nervosa; k= number of studies analysed for that particular comparison; TAU= treatment as usual; IPT= interpersonal psychotherapy; FPDT= Focal psychodynamic therapy; EOT= end of treatment; TL= therapist-led; GSH= guided self-help.

Table 2
Characteristics of Included Studies in the Meta-Analysis

Study	Sample	Cognitive-behavioural condition			Comparison (n)	Behavioural outcome	Cognitive outcome	Risk of bias
		CBT format (n)	CBT mode	CBT protocol				
Agras et al (1994)	BN	Therapist-led (n=23)	Individual	CBT-BN variant	Desipramine (n=12)	Binge episodes Purge episodes	Diet preoccupation	? ? + -
Agras et al (1989)	BN	Therapist-led (n=17)	Individual	CBT-BN variant	Wait-list (n=18) Non-directive therapy (n=16)	Purge episodes	Diet factor	+ ? SR -
Agras et al (1995)	BED	Therapist-led (n=31)	Individual	Other (Based on a modified version for the manual developed for the Telch 2001 study).	Wait-list (n=11)	Binge episodes	Cognitive restraint	? ? SR -
Agras et al (2000)	BN	Therapist-led (n=65)	Individual	CBT-BN	IPT (n=64)	Remission from b/p Binge episodes	EDE global	+ + + -
Agras et al (1994)	BED	Therapist-led (n=36)	Group	Other (Based on a modified version for the manual developed for the Telch 2001 study).	BWL (n=37)	Binge episodes	Cognitive restraint	+ ? SR +
Allen & Craighead, 1999	BED	Therapist-led (n=11)	Group	Other (Appetite-Awareness-Training)	Wait-list (n=11)	Binge episodes	-	? ? SR -
(Ball & Mitchell, 2004)	AN (adoles cents)	Therapist-led (n=9)	Individual	Other (Treatment manual developed by Garner & Bemis, 1982)	BFT (n=9)	-	EDE global	? ? ? -

Table 2
Characteristics of Included Studies in the Meta-Analysis

Study	Sample	Cognitive-behavioural condition			Comparison (n)	Behavioural outcome	Cognitive outcome	Risk of bias
		CBT format (n)	CBT mode	CBT protocol				
(Banasiak, Paxton, & Hay, 2005)	BN	GSH (n=54)	Individual	CBT-BN variant	Wait-list (n=55)	Binge episodes Purge episodes Remission from b/p	EDE global	+ + + +
(Byrne et al., 2017)	AN	Therapist-led (n=39)	Individual	CBT-E	MANTRA N=41 SSCM (N=40)	-	EDE global	+ + + +
Carrard et al., 2011)	BED	GSH (n=37)	Internet	Other (Online program developed in the SALUT project. 11 modules)	Wait-list (n=37)	Binge episodes	EDE global	+ - SR +
(Carter & Fairburn, 1998)	BED	GSH (n=34) PSH (n=35)	Individual Individual	CBT-BN variant CBT-BN variant	Wait-list (n=24)	Binge episodes Remission from b	EDE global	+ + + +
(Carter et al., 2003)	BN	PSH (n=28)	Individual	CBT-BN variant	Supportive GSH (n=28) Wait-list (n=29)	Binge episodes Purge episodes	Weight concern Shape concern Dietary restraint Eating concern	+ + + +
(Chen et al., 2016)	Trans (most BED)	Therapist-led (n=31)	Individual	CBT-BN variant	DBT (n=26)	Binge episodes Purge episodes Remission from b Remission from p	EDE global	+ ? + +
(Cooper & Steere, 1995)	BN	Therapist-led (n=13)	Individual	CBT-BN variant	BT (n=14)	Binge episodes Purge episodes Remission from b Remission from p	Dietary restraint Weight concern Shape concern Eating concern	? ? + -

Table 2
Characteristics of Included Studies in the Meta-Analysis

Study	Sample	Cognitive-behavioural condition			Comparison (n)	Behavioural outcome	Cognitive outcome	Risk of bias
		CBT format (n)	CBT mode	CBT protocol				
(Davis, McVey, Heinmaa, Rockert, & Kennedy, 1999)	BN	Therapist-led (n=37)	Individual	CBT-BN variant	Wait-list (n=19)	Binge episodes Purge episodes Remission from b/p	EDE global	? ? + -
Durand & King, 2003)	BN	GSH (n=34)	Individual	CBT-BN variant	TAU (n=34)	Binge episodes Purge episodes	EDE global	+ + - -
DeBar et al., 2011)	BED	GSH (n=81)	Individual	CBT-BN variant	TAU (n=79)	Remission from b	Weight concern Shape concern Dietary restraint Eating concern	+ ? SR +
DeBar et al., 2013)	BED	GSH (n=13)	Individual	Other (Treatment program adapted for adolescents an was developed for the study)	TAU (n=13)	Remission from b Binge episodes	Weight concern Shape concern Dietary restraint Eating concern	? ? + +
Dingemans, Spinhoven, & van Furth, 2007)	BED	Therapist-led (n=30)	Group	Other (Based on the manual described by Telch, 2002).	Wait-list (n=22)	Remission from b Binge episodes	EDE global	? ? + +
(Eldredge et al., 1997)	BED	Therapist-led (n=36)	Group	CBT-BN variant	Wait-list (n=10)	Binge episodes	Cognitive restraint	? ? SR -
(Fairburn et al., 2015)	Trans (BN most)	Therapist-led (n=58)	Individual	CBT-E	IPT (n=60)	Remission from b/p	EDE global	+ ? + -

Table 2
Characteristics of Included Studies in the Meta-Analysis

Study	Sample	Cognitive-behavioural condition			Comparison (n)	Behavioural outcome	Cognitive outcome	Risk of bias
		CBT format (n)	CBT mode	CBT protocol				
(Fairburn et al., 1991)	BN	Therapist-led (n=21)	Individual	CBT-BN	IPT (n=22) BT (n=19)	Remission from b Binge episodes Purge episodes	Weight concern Shape concern Dietary restraint Eating concern	? ? + -
(Freeman, Barry, Dunkeld-Turnbull, & Henderson, 1988)	BN	Therapist-led (n=32)	Individual	Other (not described)	BT (n=30) Supportive therapy (n=30) Wait-list (n=20)	Binge episodes Purge episodes	-	+ ? SR +
Garner et al., 1993)	BN	Therapist-led (n=25)	Individual	CBT-BN	Supportive expressive therapy (n=25)	Binge episodes Purge episodes	Weight Concern Shape concern Drive for thinness Body dissatisfaction	+ - ? -
(Goldbloom et al., 1997)	BN	Therapist-led (n=14)	Individual	CBT-BN	Fluoxetine (n=12)	Binge episodes Purge episodes Remission from b/p	Weight Concern Shape concern Dietary restraint Eating concern	? ? ? -
Gorin, Le Grange, & Stone, 2003)	BED	Group (63)	Therapist-led	Other (based on a manual developed By Telch and colleagues)	Wait-list (31)	Abstinence Binge frequency	-	+ ? ? +
(Griffiths, Hadzi-Pavlovic, &	BN	Therapist-led (n=20)	Individual	CBT-BN variant	BT (n=21) Wait-list (n=22)	Binge episodes Purge episodes Remission from b/p	Drive for thinness	? ? SR -

Table 2
Characteristics of Included Studies in the Meta-Analysis

Study	Sample	Cognitive-behavioural condition			Comparison (n)	Behavioural outcome	Cognitive outcome	Risk of bias
		CBT format (n)	CBT mode	CBT protocol				
Channon-Little, 1994)							Body dissatisfaction	
Grilo & Masheb, 2005)	BED	GSH (n=37)	Individual	CBT-BN variant	BWL (n=38) Self-monitoring (n=15)	Binge episodes Remission from b	Weight Concern Shape concern Dietary restraint Eating concern	+ + SR +
(Grilo et al., 2014)	BED	PSH (n=23)	Individual	CBT-BN variant	Sibutramine (n=20)	Binge episodes Remission from b	EDE global	+ + + -
(Grilo, Masheb, & Wilson, 2005)	BED	Therapist-led (n=28)	Individual	CBT-BN	Fluoxetine (n=27)	Binge episodes Remission from b	EDE global	+ + SR +
Grilo, Masheb, Wilson, Gueorguieva, & White, 2011)	BED	Therapist-led (n=45)	Group	CBT-BN	BWL (n=45)	Binge episodes Remission from b	EDE global	+ ? ? +
Grilo, White, Gueorguieva, Barnes, & Masheb, 2013)	BED	PSH (n=24)	Individual	CBT-BN variant	TAU (n=24)	Binge episodes Remission from b	EDE global	+ ? + +
Hsu et al., 2001)	BN	Individual (24)	Therapist-led	Other (based on a manual developed by Hsu)	Support group (24)	Abstenence	-	+ ? SR +
Jacobi, Dahme, & Dittmann, 2002)	BN	Therapist-led (n=19)	Individual	CBT-BN variant	Fluoxetine (n=16)	Binge episodes Purge episodes Remission from b/p	Drive for thinness Body dissatisfaction	? ? SR +

Table 2
Characteristics of Included Studies in the Meta-Analysis

Study	Sample	Cognitive-behavioural condition			Comparison (n)	Behavioural outcome	Cognitive outcome	Risk of bias
		CBT format (n)	CBT mode	CBT protocol				
Kenardy, Mensch, Bowen, Green, & Walton, 2002)	BED	Group (n=17)	Therapist-led	Other (manual developed by Telch and colleagues)	Non-prescriptive therapy (n=17)	Binge frequency	Drive for thinness Body dissatisfaction	+ ? SR -
(Kelly & Carter, 2015)	BED	GSH (n=13)	Individual	CBT-BN variant	Compassion therapy (n=15) Wait-list (n=13)	Binge episodes	EDE global	+ ? SR +
Kirkley, Schneider, Agras, & Bachman, 1985)	BN	Therapist-led (n=13)	Group	Other (unclear)	Non-directive supportive therapy (n=9)	Binge episodes Purge episodes	-	+ ? SR -
Kristeller, Wolever, & Sheets, 2014)	BED	CBT psychoeducation (n=24)	-	-	Mindfulness-based therapy (n=35) Wait-list (n=24)	Binge episodes Remission from b	Cognitive restraint	? ? ? +
(Lavender et al., 2012)	BN	Therapist-led (n=21)	Group	CBT-BN variant	Emotion social mind training (n=23)	Binge episodes Purge episodes Remission from b/p	EDE global	+ ? + +
(Le Grange, Lock, Agras, Bryson, & Jo, 2015)	BN (Adolescents)	Therapist-led (n=58)	Individual	CBT-BN variant	FBT-BN (n=51)	Remission from b/p Binge episodes Purge episodes	EDE global	+ + + +
Lee & Rush, 1986)	BN	Therapist-led (n=15)	Group	Other	Wait-list (n=15)	Binge episodes Purge episodes	-	+ ? SR +

Table 2
Characteristics of Included Studies in the Meta-Analysis

Study	Sample	Cognitive-behavioural condition			Comparison (n)	Behavioural outcome	Cognitive outcome	Risk of bias
		CBT format (n)	CBT mode	CBT protocol				
				(unpublished treatment manual developed by Kumetz-lee)				
Leitenberg, Rosen, Gross, Nudelman, & Vara, 1988)	BN	Therapist-led (n=22)	Group	CBT-BN variant	Exposure response prevention (n=11) Exposure response prevention 2 (n=12) Wait-list (n=12)	Purge episodes		? ? SR -
(Ljotsson et al., 2007)	Trans (most BED)	GSH (n=33)	Individual	CBT-BN variant	Wait-list (n=34)	Binge episodes Purge episodes	EDE global	+ ? ? -
(Lock et al., 2013)	AN	Therapist-led (n=23)	Individual	Other (CBT-AN)	Cognitive remediation therapy (n=23)	-	EDE global	+ ? + +
(McIntosh et al., 2005)	AN	Therapist-led (n=19)	Individual	Other (CBT-AN)	IPT (n=21) Non-specific supportive management (n=16)	-	Weight concern Shape concern Eating concern Dietary restraint Drive for thinness Body dissatisfaction	? ? ? +
(McIntosh et al., 2016)	Trans (most BN)	Therapist-led (n=38) Therapist-led (n=36)	Individual Individual	CBT-BN variant Other (Appetite-focused CBT)	Schema therapy (n=38)	Binge episodes Purge episodes	EDE global	+ + + +

Table 2
Characteristics of Included Studies in the Meta-Analysis

Study	Sample	Cognitive-behavioural condition			Comparison (n)	Behavioural outcome	Cognitive outcome	Risk of bias
		CBT format (n)	CBT mode	CBT protocol				
Munsch et al (2007)	BED	Therapist-led	Group	CBT-BN variant	BWL	Remission from b Binge episodes	Weight concern Eating concern Shape concern Dietary restraint	+ ? - -
Nauta, Hospers, Kok, & Jansen, 2000)	BED	Individual (21)	Therapist-led	Other (based on Beckian CT that included self-monitoring)	Behavior therapy (16)	Binge frequency	Shape and weight concern Eating concern Restraint	? ? SR +
(Peterson, Mitchell, Crow, Crosby, & Wonderlich, 2009)	BED	Therapist- led (n=60)	Group	Other (Treatment manual devised by Mitchell et al., 2009)	Wait-list (n=69)	Remission from b Binge episodes	EDE global	+ + + +
		Therapist assisted (n=63)	Group	Other				
		GSH (n=67)	Group	Other				
(Peterson et al., 1998)	BED	Therapist- led (n=16)	Group	Other (Treatment manual devised by Mitchell et al., 1990)	Wait-list (n=11)	Binge episodes	-	? ? SR +
		Therapist assisted (n=19)	Group	Other				
		GSH (n=15)	Group	Other				

Table 2
Characteristics of Included Studies in the Meta-Analysis

Study	Sample	Cognitive-behavioural condition			Comparison (n)	Behavioural outcome	Cognitive outcome	Risk of bias
		CBT format (n)	CBT mode	CBT protocol				
Pike, Walsh, Vitousek, Wilson, & Bauer, 2003)	AN	Therapist-led	Individual	Other (Garner et al)	Nutritional counselling	-	EDE global less than 1 SD of general pop	+ ? ? +
(Poulsen et al., 2014)	BN	Therapist-led (n=36)	Individual	CBT-E	Psychoanalysis (n=36)	Binge episodes Purge episodes	EDE global	+ + + +
Ricca et al., 2001)	BED	Therapist-led (n=20)	Individual	CBT-BN variant	Fluoxetine (n=21) Fluvoxamine (n=22)	Binge episodes	EDE global	? ? SR +
(Sánchez-Ortiz et al., 2011)	BN	GSH (n=31)	Online	Other (treatment manual developed by Williams, 1998)	Wait-list (n=36)	Binge episodes Purge episodes	EDE global	+ + + -
(Schmidt et al., 2007)	BN	GSH (n=44)	Individual	Other (manual developed by Schmidt & Treasure)	FBT (n=41)	Remission from b/p	Shape/weight concerns Dietary restraint Eating concern	+ + + +
(Schlup, Munsch, Meyer, Margraf, & Wilhelm, 2009)	BED	Therapist-led (n=18)	Group	Other (treatment manual by Munsch, 2007)	Wait-list (n=18)	Remission from b Binge episodes	Weight concern Shape concern Dietary restraint Eating concern	+ ? SR +
(Schmidt et al., 2008)	BN	GSH (n=49)	Internet	Other (overcoming bulimia, CD-ROM based treatment)	Wait-list (n=48)	Binge episodes Purge episodes	EDE global	+ + + +
Sundgot-Borgen,	BN	Therapist-led (n=14)	Individual	Other (not specified)	Nutritional counselling (n=17)	Binge episodes Purge episodes	-	? ? SR -

Table 2
Characteristics of Included Studies in the Meta-Analysis

Study	Sample	Cognitive-behavioural condition			Comparison (n)	Behavioural outcome	Cognitive outcome	Risk of bias
		CBT format (n)	CBT mode	CBT protocol				
Rosenvinge, Bahr, & Schneider, 2002)					Physical activity (n=12)			
Shapiro et al., 2007)	BED	Therapist-led (n=22)	Group	Other (treatment program developed for the purposes of the study)	Wait-list (n=22)	Binge episodes Remission from b	-	+ ? SR -
		GSH (n=22)	Internet	Other				
(Steele & Wade, 2008)	BN	GSH (n=15)	Individual	CBT-BN variant	Mindfulness-based CT (n=15)	Binge episodes Purge episodes	EDE global	+ - + +
					CBT for perfectionism (n=17)			
(Stefini et al., 2017)	BN	Therapist-led (n=39)	Individual	CBT-BN variant	Psychodynamic therapy (n=42)	Binge episodes Purge episodes	EDE global	+ + + +
Striegel-Moore et al., 2010)	Trans (BED)	GSH (n=59)	Individual	CBT-BN variant	TAU (n=64)	Remission from b	Weight concern Shape concern Dietary restraint Eating concern	+ ? + +
(Tantillo & Sanftner, 2003)	Trans (BN)	Therapist-led (n=7)	Group	Other (treatment program developed for the purposes of the study)	Relational therapy (n=8)	Binge episodes Purge episodes	-	? ? SR -

Table 2
Characteristics of Included Studies in the Meta-Analysis

Study	Sample	Cognitive-behavioural condition			Comparison (n)	Behavioural outcome	Cognitive outcome	Risk of bias
		CBT format (n)	CBT mode	CBT protocol				
(Tasca et al., 2006)	BED	Therapist-led (n=37)	Group	Other (treatment manual from Wilfley, 1996)	Psychodynamic interpersonal therapy (n=37) Wait-list (n=33)	Binge episodes	Cognitive restraint	? ? SR -
Telch, Agras, Rossiter, Wilfley, & Kenardy, 1990)	BED	Therapist-led (n=18)	Group	CBT-BN variant	Wait-list (n=22)	Binge episodes Remission from b	Diet factor	+ ? SR -
(ter Huurne et al., 2015)	Trans (Most BED)	GSH (n=108)	Internet	Other (treatment program developed for the purposes of the study)	Wait-list (n=106)	-	EDE global	+ ? SR +
(Thackwray, Smith, Bodfish, & Meyers, 1993)	BN	Therapist-led (n=13)	Individual	CBT-BN variant	BT (n=13) Non-specific self-monitoring treatment (n=13)	Binge episodes Purge episodes Remission from B/P	Drive for thinness	? ? SR -
(Touyz et al., 2013)	AN	Therapist-led (n=31)	Individual	Other (CBT-AN)	Specialist supportive clinical management (n=32)	-	EDE global	+ + + +
Traviss, Heywood-Everett, & Hill, 2011)	Trans (BN)	GSH (n=37)	Individual	Other (treatment program developed for the purposes of the study)	Wait-list (n=31)	Remission from b Binge episodes Purge episodes	EDE global	+ ? + +

Table 2
Characteristics of Included Studies in the Meta-Analysis

Study	Sample	Cognitive-behavioural condition			Comparison (n)	Behavioural outcome	Cognitive outcome	Risk of bias
		CBT format (n)	CBT mode	CBT protocol				
Treasure et al., 1994)	BN	Therapist-led (n=21) PSH (n=41)	Individual	Other (Treatment manual by Schmidt & Treasure) Other	Wait-list (n=19)	Remission from b Remission from p	Dietary restraint Shape concern Weight concern Eating concern	? ? ? +
(Wagner et al., 2016)	BED	GSH (n=69)	Internet	Other (mixture of a variety of different CBT approaches)	Wait-list (n=70)	Binge episodes	EDE global	+ ? SR +
(Walsh et al., 1997)	BN	Therapist-led (n=25)	Individual	CBT-BN variant	Supportive expressive therapy (n=22) Fluoxetine (n=28)	Binge episodes Purge episodes Remission from b/p	EDE global	? ? + +
Walsh, Fairburn, Mickley, Sysko, & Parides, 2004)	BN	GSH (n=25)	Individual	CBT-BN variant	Fluoxetine (n=20) Wait-list (n=22)	Binge episodes Purge episodes Remission from b/p	Dietary restraint	? ? ? +
Wilfley et al., 1993)	BED	Therapist-led (n=18)	Group	Other (Manual developed by Telch 1990)	IPT (n=18) Wait-list (n=20)	Binge episodes Remission from b/p	Cognitive restraint	? ? + +
Wilfley et al., 2002)	BED	Therapist-led (n=78)	Group	Other (Manual developed by Telch 1990)	IPT (n=80)	Remission from b	Dietary restraint Shape concern Weight concern Eating concern	? ? + +

Table 2
Characteristics of Included Studies in the Meta-Analysis

Study	Sample	Cognitive-behavioural condition			Comparison (n)	Behavioural outcome	Cognitive outcome	Risk of bias
		CBT format (n)	CBT mode	CBT protocol				
Wilson et al (2010)	BED	GSH (n=66)	Individual	CBT-BN variant	IPT (n=75) BWL (n=64)	Binge episodes	EDE global	+ ? + +
Wolf & Crowther, 1992)	BN	Therapist-led (n=15)	Group	Other (not clear)	BT (n=15) Wait-list (n=11)	Binge episodes Purge episodes	Drive for thinness Body dissatisfaction	? ? + +
Wonderlich et al (2014)	BN	Therapist-led (n=36)	Individual	CBT-E	Integrative cognitive affective therapy (n=36)	Binge episodes Purge episodes Remission from b/p	EDE global	+ ? + +
Zipfel et al (2014)	AN	Therapist-led (n=80)	Individual	CBT-E	Focal psychodynamic therapy (n=80) Optimized specialised care (n=82)	-	EDI total	+ + + +

a) In the last column a positive sign (low risk), a "?" (unclear), or a negative sign (high risk) is given for the four items of risk of bias: allocation sequence; concealment of allocation to
blinding of assessors; and intention-to treat analyses. For Blinding of assessor we reported "SR" when only self-report outcome measures were used; TAU= treatment as usual' BT= beh
therapy; GSH= guided self-help; trans=transdiagnostic; BED= binge eating disorder; BN= bulimia nervosa; AN= anorexia nervosa; PSH= pure self-help; IPT= interpersonal psychothera
eating disorder examination; BWL= behavioural weight loss.

Appendix D: Supplementary material for study 2

Mediators, moderators and predictors of follow-up

Mediators (Table 1)

Bulimia nervosa: Only early symptom change was explored as a mediator of follow-up outcome for BN in at least two studies. Early behavioural symptom change was assessed as a mediator of follow-up treatment outcome in two studies (Marrone, Mitchell, Crosby, Wonderlich, & Jollie-Trottier, 2009; Thompson-Brenner, Shingleton, Sauer-Zavala, Richards, & Pratt, 2015). Both studies found that a 25% reduction in binge eating and purging was associated higher rates of binge/purge remission.

Binge eating disorder. Only early symptom change was explored as a mediator of follow-up outcome for BED in at least two studies. Only one study found early behavioural symptom change (65% reduction in binge eating by week 4) to be associated with greater rates of binge eating remission at follow-up (Hilbert, Hildebrandt, Agras, Wilfley, & Wilson, 2015). This was not replicated in two other studies (Fischer, Meyer, Dremmel, Schlup, & Munsch, 2014; Grilo, White, Wilson, Gueorguieva, & Masheb, 2012). Two of these studies (Grilo et al., 2012; Hilbert et al., 2015) reported on cognitive outcomes, and found no effect for early behavioural symptom change.

Mixed sample. No mediators of follow-up outcome were reported in at least two studies of underweight or normal weight mixed samples.

Moderators (Table 2)

Binge eating disorder. Demographic variables were the only variables tested as moderators of follow-up treatment outcome in two or more studies. Client age, gender, ethnicity, and education did not moderate follow-up treatment outcome in a trial that

administered psychological comparisons (Wilson, Wilfley, Agras, & Bryson, 2010) and a trial that administered a pharmacological comparison (Grilo et al., 2014).

Bulimia nervosa. No moderators of follow-up treatment outcome were observed in two or more studies.

Mixed sample. No moderators of follow-up treatment outcome were observed in two or more studies of underweight or normal weight mixed samples.

Predictors (Table 3)

Bulimia nervosa. Several predictors explored in two or more studies of BN were identified. Baseline bulimic frequencies were tested in three studies of CBT-BN follow-up treatment outcome. While two studies found no relationship between baseline frequencies and follow-up outcome (Baell & Wertheim, 1992; Ghaderi, 2006), Fahy and Russell (1993) found that a higher pre-treatment vomiting frequency predicted poorer follow-up rates of remission from bingeing/purging and also higher levels of eating disorder cognitions. Additionally, weight and shape concern and dietary restraint were tested as predictors of follow-up and no relationships emerged (Fahy & Russell, 1993; Ghaderi, 2006). Depression scores were tested as a predictor of follow-up in two studies, and while Ghaderi (2006) found no evidence of prediction following CBT-BN, Fahy & Russell (1993) found higher depression scores to predict poorer rates of binge eating and purge remission at follow-up. Finally, self-esteem was tested in two studies, and while Ghaderi (2006) found no evidence of prediction, Baell and Wertheim (1992) found a lower self-esteem to predict higher levels of eating disorder cognitions at follow-up.

Mixed sample. The only variable to be tested in two or more studies at follow-up was baseline depression scores. Two studies tested baseline depression, and while Vaz, Conceicao, and Machado (2014) found no relationship between depression scores and binge eating/purging remission rates at follow-up, Castellini et al. (2012) found higher depression

scores to predict less change in OBE at follow-up. These two studies sampled individuals of normal weight.

Binge eating disorder. No predictors of follow-up tested in two or more studies were observed for BED.

Table 1:

Mediators of follow-up in bulimia nervosa and binge eating disorder

Sample			**Study Design**		**Follow up behavioural outcome**			**Follow-up cognitive outcome**			
BN	**Mediator Variable**	**Studies**	**RCT**	**Other**	**CBT-BN**	**CBT-E**	**CBTgsh**	**CBT-BN**	**CBT-E**	**CBTgsh**	**Mean Quality rating % (range)**
	Early symptom change										
	Behavioural symptoms	2	1	1	+	+					92% (83-100%)
	Cognitive symptoms	0									
BED	Early symptom change										
	Behavioural symptoms	3	2	1	0		0 +			0 0	53% (28-56%)
	Cognitive symptoms										

Note + = greater change in mediator is associated with better outcome;

Table 2

Moderators of follow-up in binge eating disorder

			Comparison Treatment		**Follow-up Behavioural Outcomes**			**Follow-up Cognitive Outcomes**			
BED	**Moderator**	**Studies**	**Psychological**	**Pharmacology**	**CBT-BN**	**CBT-E**	**CBTgsh**	**CBT-BN**	**CBT-E**	**CBTgsh**	**Mean Quality rating % (range)**
	Age	2	1	1			0 0			0	57% (43-71%)
	Gender	2	1	1			0 0			0	57% (43-71%)
	Ethnicity	2	1	1			0 0			0	57% (43-71%)
	Education	2	1	1			0 0			0	57% (43-71%)

Table 3
Predictors of follow-up in bulimia nervosa and mixed transdiagnostic samples.

Sample			**Study Design**		**Follow-up behavioural outcome**			**Follow-up cognitive outcome**			
BN	**Predictor Variable**	**Studies**	**RCT**	**Other**	**CBT-BN**	**CBT-E**	**CBTgsh**	**CBT-BN**	**CBT-E**	**CBTgsh**	**Mean Quality rating % (range)**
	Higher baseline bulimic frequencies	3	1	2	0 —			0 —			51% (25-71%)
	Weight concern	2	1	1	0 0			0			41% (25-57%)
	Shape concern	2	1	1	0 0			0			41% (25-57%)
	Dietary restraint	2	1	1	0 0			0			41% (25-57%)
	Higher depression scores	2	1	1	— 0			0			41% (25-57%)
	Lower self-esteem	2	1	1	0			—			64% (57-71%)
Mixed											
	Higher depression scores	2	0	2	—		0				

Predictors of Secondary Outcomes

Diagnostic-related outcomes. Predictors of diagnostic outcomes were tested in five included studies (Castellini et al., 2011; Castellini et al., 2012; Fioravanti et al., 2014; La Mela, Maglietta, Lucarelli, Mori, & Sassaroli, 2013; Ricca et al., 2010). Diagnostic outcomes include: Recovery (not meeting diagnostic criteria for an eating disorder), diagnostic cross-over, relapse, and treatment resistance. All studies used a mixed sample, with the exception of Fioravanti et al. (2014) who used a BN sample.

Age (Castellini et al., 2012; La Mela et al., 2013; Ricca et al., 2010) and gender (Castellini et al., 2011; Castellini et al., 2012; Ricca et al., 2010) were tested in three studies. No relationships with outcome were reported. Overweight during childhood was tested in two studies, and while Castellini et al. (2011) found no relationship to outcome, Ricca et al. (2010) found higher rates of treatment resistance in participants who were overweight during childhood.

Shape concern was tested in three studies, and while La Mela et al. (2013) and Ricca et al. (2010) found no relationship to outcome, Castellini et al. (2012) found a higher shape concern to predict lower recovery rates and cross-over from BN to AN. Weight and eating concern were unrelated to outcome in these three studies. One study found higher binge eating severity scores to be related to lower recovery rates (Ricca et al., 2010), although this was not replicated (Castellini et al., 2011). Finally, out of three studies that tested baseline bulimic frequencies, only one found higher baseline frequencies to predict lower rates of recovery (Castellini et al., 2012). Finally, comorbid personality disorders were unrelated to outcome in three studies (Castellini et al., 2011; La Mela et al., 2013; Ricca et al., 2010).

Weight loss outcomes. Seven studies explored predictors and moderators of weight loss in the eating disorders (Dalle Grave, Calugi, & Marchesini, 2012; Grilo et al., 2014; Grilo, Masheb, Wilson, & Crosby, 2012; Masheb & Grilo, 2008a; Ricca et al., 2010; Schlup,

Meyer, & Munsch, 2010; Striegel-Moore et al., 2010). There were no variables identified in two or more of these studies that were significantly related to weight loss following CBT.

Psychosocial outcomes. Psychosocial outcomes include sexual functioning improvement, interpersonal problems, depression and anxiety scores, and quality of life. Nine studies tested predictors of psychosocial outcomes (Castellini et al., 2013; Chui, Safer, Bryson, Agras, & Wilson, 2007; Dalle Grave, Calugi, & Marchesini, 2009; Dalle Grave et al., 2012; Fahy, Eisler, & Russell, 1993; Grilo, Masheb, et al., 2012; Masheb & Grilo, 2008a, 2008b; Watson, Allen, Fursland, Byrne, & Nathan, 2012), and little consistency emerged. Age was tested in three studies and only Grilo et al (2014) found an older age to predict faster reductions in depression scores at post-treatment. Comorbid personality disorder predicted higher depression scores in only one (Fahy et al., 1993) study and baseline bulimic frequencies as well as EDE global scores were consistently unrelated to outcome. See table 4.

Individual Mediators, Moderators, and Predictors Tested

Variable (Sample)	Study Design		Behavioural outcomes			Cognitive outcomes		
Mediators	**RCT**	**Other**	**CBT-BN**	**CBT-E**	**CBTgsh**	**CBT-BN**	**CBT-E**	**CBTgsh**
Sudden gains during treatment (mixed)		1					+	
Meal and snack consumption (BN)	1		+					
Client engagement in treatment (BN)	1		+			0		
Change in self-efficacy (BN)	1		+					
Change in self-esteem (BN)	1		0					
Change in interpersonal problems (BN)	1		0					
Changes in suitability of treatment ratings(BN)	1		0					
Adherence to regular eating (mixed)		1			+			
Moderators								
Emotion dysregulation (BN)	1		0			0		
Structural analysis social behaviour (BN)	1		0			0		
Assessment of personality pathology (BN)	1		0			0		
Transdiagnostic maintaining mechanisms(mix)	1						—	
Current substance abuse (BED)	1				0			
Quality of life scores (BED)	1				0			
EDE global scores × self-esteem (BED)	1		—				—	
DIBR affective/interpersonal problem (BN)	1						0	
Eating disorder diagnosis (mixed)	1							
Predictors								
Current depression (BN)		1	—					
Social adjustment (BN)		1	—					
Bulimic thoughts score (BN)		1	0					
General self-efficacy(BN)		1	0					
Interpersonal problems (BN)		1	0					
Impulsivity (BN)		1	0					
EDI ineffectiveness (BN)		1				0		
Knowledge of CBT principles (BED)	1				+			
Perceived effectiveness of CBT (BED)	1				0			
Obsessive-compulsive disorder (mixed)		1	0					
Amount of exercise (mixed)		1					—	
Childhood body shape (BN)		1		0				
Family history of overweight (BN)		1		0				
Body weight difference (BN)		1		0				
Prior psychiatric admission (BN)		1	—					
Eating attitude test scores (BN)		1	—					
Obsessionality (BN)		1	—					
Emotional eating (BED)		1	0					
Social support (mixed)	1		0					
OCD scores (BED)		1						0
Dissociative experiences (BED)		1			—			
NEO-PI scores (BED)		1				0		
SCL-90 subscales (BED)		1				0		
Amphetamine use (BED)	1		—					
Number of diet attempts (BED)	1		—					
Dysfunctional core beliefs (BN)		1	0					
Therapist type (BN)	1		0					
Treatment Adherence (BN)	1		0					
Amenorrhea presence (AN)	1				0		0	
Alcohol intake (Mixed)	1						0	

Note: + = variable associated with better outcome; - = variable associated with poorer outcome.

Appendix E: Participant information letter for Study 2

PARTICIPANT INFORMATION LETTER

PROJECT TITLE: Exploring disordered eating maintaining factors.
PRINCIPAL INVESTIGATOR: A/Prof Leah Brennan
Co-Investigator: Dr Xochitl De la Piedad Garcia
STUDENT RESEARCHER: Mr Jake Linardon
STUDENT'S DEGREE: Doctor of Philosophy

Dear Participant,

You are invited to participate in the research project described below.

What is the project about?
This research project investigates the psychological factors that are associated with disordered eating (e.g., dieting, excessive exercise, fasting, binging, and vomiting). For example, the way we evaluate our body is thought to impact on when, how much, and what we eat. However, it is unclear on whether it is the behaviours associated with body-image (e.g., looking in the mirror frequently) or the cognitions (e.g., the way we evaluate ourselves) that influence eating behaviour. It is the current project's aim to clarify these relationships.

Who is undertaking the project?
This project is being conducted by A/Prof Leah Brennan (Primary investigator), Dr Xochitl de la Piedad Garcia (Co-investigator) and Mr Jake Linardon (Student researcher). This project will form the basis of Mr Jake Linardon's PhD thesis.

Are there any risks associated with participating in this project?
As you will complete several standardised self-report questionnaires, higher scores within a certain range on these questionnaires might be indicative of a possible, undiagnosed eating disorder. Participants who score highly on these questionnaires will be made aware of this after completion of the survey which may have the potential to cause slight emotional and psychological discomfort. To minimise discomfort associated with answering these items, we have only included standardised self-report questionnaires that are commonly used for research purposes. In addition, a PDF list of referral options will be provided should you feel concerned in any way. All responses will be kept under complete confidentiality.

Who can participate?
Eligible participants for this survey are male and females who are over the age of 18 years.

What will I be asked to do?
Participants will be asked to complete an online survey including several short standard measures such as questionnaires about your eating behaviours, body-image and wellbeing. A link will be provided to you, as the participant, which will take you to a secure experiment website that will allow you to complete the survey.

If you are willing to take part in this research, having read this information letter, you should follow the link provided at the bottom of the page. At the end of the survey, you may be asked if you are interested in being notified about opportunities to participate in future research on a similar topic. If you are interested, you will be asked to provide your name, contact number, or email address. This information will not be stored with your survey responses. Your personal details will remain completely confidential.

How much time will the project take?
Completion of measures will take approximately 40 minutes.

What are the benefits of the research project?
This research project will contribute to greater understanding of the psychological factors associated with disordered eating attitudes and behaviours. In addition to this, another benefit of this project would be becoming aware of the *possible* presence of an eating disorder so that, if need be, early interventions and professional help can be set up appropriately. In addition, after completion of the survey, you will have a chance to enter a draw to win an iPad mini. If you express interest, you will be asked to provide your email address at the end of the survey. Your email address will in no way be linked to your questionnaire responses. You will be notified of the outcome after completion of this study.
Can I withdraw from the study?
Participation in this study is completely voluntary. You are not under any obligation to participate and if you do agree to participate, your information will not be linked to your responses.

Will anyone else know the results of the project?
The findings of this study will be prepared for presentation at conferences, publication in peer-reviewed journals and inclusion in the student researcher's thesis. Only non-identifiable group data will be reported. The confidentiality of results is assured as no individual responses will be identified, and only aggregated results will be presented.

Will I be able to find out the results of the project?
Aggregated findings will be available on the School of Psychology research website.

Who do I contact if I have questions about the project?
If you have any questions about the project that cannot be addressed by the student researcher, please contact the Principal Investigator, A/Prof Leah Brennan via email Leah.Brennan@acu.edu.au.

What if I have a complaint or any concerns?
The study has been reviewed by the Human Research Ethics Committee at Australian Catholic University If you have any complaints or concerns about the conduct of the project, you may write to the Manager of the Human Research Ethics Committee or Deputy Vice Chancellor (Research).

Manager, Ethics
c/o Office of the Deputy Vice Chancellor (Research)
Australian Catholic University
North Sydney Campus
PO Box 968
NORTH SYDNEY, NSW 2059
Ph.: 029739 2519
Fax: 02 9739 2870
Email: res.ethics@acu.edu.au

Any complaint or concern will be treated in confidence and fully investigated. You will be informed of the outcome.

I want to participate! How do I sign up?
If you agree to participate, you should click on the link provided to complete the measures.

Yours sincerely,

A/Prof Leah Brennan
Principle Investigator

Appendix F: Participant information letter for Study 3

PARTICIPANT INFORMATION LETTER

PROJECT TITLE: Pathways to Calm and Healthy Eating
PRINCIPAL INVESTIGATOR: Associate Professor Leah Brennan
INVESTIGATOR: Doctor Xochitl De la Piedad Garcia
INVESTIGATOR: Mr Jake Linardon

Dear Participant,

You are invited to participate in the research study described below, and this information letter is for you to keep. Please read all the information in full before making a decision.

What is the project about?
The aim of this study is to evaluate and compare two guided self-help treatment programs for body image and eating behavior problems. The treatments aim to provide you with the strategies to improve your problem eating behaviors, which will therefore improve your overall physical and mental health. We want to evaluate these two interventions and compare what effects it has on eating behavior as well as psychological and psycho-social wellbeing.

You have been provided with this information letter because you have indicated an interest in participating in this research study and believe that you fit the eligibility criteria.

Who is undertaking the project?
This study is being conducted by Associate Professor Leah Brennan, Doctor Xochitl De la Piedad Garcia and Mr Jake Linardon from the School of Psychology at Australian Catholic University Melbourne.

Are there any risks associated with participating in this project?
Assessment and treatment involves increased awareness of unhelpful behaviours and implementation of strategies aimed at changing established ways of behaving. This may increase discomfort in the short term but is aimed at reducing discomfort in the longer term.

We will attempt to minimise any discomfort you may experience by thoroughly explaining all procedures to you, using trained and experienced psychologists or supervised psychology interns to conduct interviews and psychological treatment, and by using established and widely-used questionnaires and interventions.

In the unlikely event that you experience significant distress during the study, you can withdraw from the study and we can refer you to a qualified counsellor or physician who is independent of the research team.

During assessment or intervention, if it appears you may be at risk or possibly have a psychological condition warranting treatment, we will inform you and you will be referred to your GP for additional care. A flyer listing relevant support services (including free services) is also attached. If you choose to engage in any additional counselling this will be at your own expense.

What will I be asked to do?

If you decide to participate in this study there are a number of steps involved:

Step 1: Initial Assessment

You will be required to complete the assessment process. There are a number of reasons for this. Firstly, it allows us to see if the program is appropriate for you; it helps us determine how best to meet your needs; and finally, it provides details about your health and behaviour so we can see if the program benefits you.

(a) *Initial interview:* You will be asked to attend an interview that will take approximately 1½ hours. This will take place at the Melbourne Psychology and Counselling Clinic at ACU. During this interview we will discuss your current eating and activity habits, and past and present physical and mental health. We will also talk about your eating behaviour concerns and general wellbeing, as well as anything else you think may be relevant. There will also be opportunity for you to ask any questions about the intervention. This interview will be video-taped for research, training and supervision purposes. Your eligibility to participate in the treatment will be assessed based on this interview. If you are eligible to continue to participation, you will be asked to complete a set of questionnaires.

(b) *Questionnaires:* You will also be asked to complete self-report questionnaires that will take approximately 90 minutes to complete. These questionnaires are designed to measure factors related to eating habits and behaviours, such as quality of life, disordered eating, body image, self-esteem, depression, anxiety and stress, perfectionism, cognitive distortions, and mood tolerance. Demographic questions will also be included in the booklet.

Step 2. Group Allocation

After you have returned the questionnaires you will be randomly allocated to one of the two guidance groups.

Step 3. Treatment

One treatment group will be based on the self-help manual *Overcoming Binge Eating* (2nd edition) by Christopher Fairburn who is the leading researcher in psychological treatment for eating and body image-related problems. This manual consists of two sections: First, the manual will provide you with information on unhelpful eating behaviours and their associated physical and psychological problems. The second section consists of a cognitive-behaviour therapy-based treatment program designed to provide you with strategies to improve unhelpful thoughts and eating behaviour.

The other treatment group will be based on the self-help manual *If not dieting, then what?* by Rick Kausmann. Rick is the Australian pioneer of the person-centred approach to the treatment of eating and weight-related problems. This manual informs the reader on the nature of dieting, eating and weight-related problems, and also provides helpful techniques on how to combat these problems.

Throughout treatment you will be required to read the self-help manual, undertake a number of tasks and activities outlined in the manual, and attend eight 25-50 minute guidance sessions designed to help educate you about the program, guide you through the program, keep you motivated and on track, and answer any questions you have.

For the first two weeks you will be asked to read the information section of the manual. Over the remaining 8 weeks you will undertake the treatment section of the manual. The 25-50 minute guidance sessions will start after you have read the information section of the manual, and will be conducted weekly. Guidance sessions will be held at the Melbourne Psychology and Counselling Clinic at ACU.

Prior to starting each guidance session, the facilitator will ask you to complete a brief questionnaire designed to assess your eating behavior over the past week.. This will take no longer than 5 minutes. After the treatment program has ended, you will be asked to complete the same battery of self-report questionnaires that you completed prior to treatments onset. This will take around 60 minutes. You will

be mailed out and asked to complete these self-report questionnaires again 6-months and 24-months after treatment.

All guidance sessions will be videotaped for research, training and supervision purposes. The video focus will be on the treatment guide and not you. If you are captured at all it will be from behind and your face will not be identified in the video. Voices may be audible but only first names will be identified in the video.

How much time will the project take?

The initial assessment should take no longer than 2 hours in total. The treatment phase will take up to 10 weeks (2 weeks to read the information section of the manual, then 8 weeks to complete the treatment program including 8 x 25-30 minute guidance sessions). You will be asked to complete self-report questionnaires (90 minutes) before and after the program and again 6-months and 24-months after you finish your treatment.

What are the benefits of the research project?

There are a number of ways in which you may benefit from participating in this study:

- You will receive empirically-based information and strategies to assist you to make positive changes to your eating habits, thoughts and behaviours.
- Your physical health and psychosocial wellbeing may also improve.

There is a need to evaluate guided self-help programs designed to improve eating behaviours so that it can be promoted to the community as a cost-effective, efficient means of psychological treatment. The results of this study will provide information on the differential effect of receiving two different guided self-help treatments as well as information on who is likely to benefit most from these treatments.

This research does not involve payment of any kind.

Can I withdraw from the study?

Being in this study is voluntary and you are under no obligation to consent to participate. If you do decide to take part and later change your mind, you are free to withdraw from the study. To do that, please let the researchers know about your wish to withdraw at any time before or during the study. If you do withdraw throughout the study, we will ask to be able to continue to use any data that you have already provided. You can also expect any questions you have about the study to be answered at any time.

Will anyone else know the results of the project?

All the information you give to us will be treated in the strictest confidence and used only for research purposes. Your name will not be used and all participants will be identified by a code number. All treatment videos and information you give us will be kept in a locked filing cabinet in the School of Psychology at ACU for 7 years and only people involved in the study will have access to the information. Data will be entered into an electronic database on a secure server at Australian Catholic University. It will be stored in an encrypted form and only the research team will have access to the data. Following completion of the study, data will be stored in a de-identified form in a locked filing cabinet in the School of Psychology at ACU.

A report of the study may be submitted for publication, but individual participants will not be identifiable in such a report. Your non-identifiable data may be used for other research purposes in the future; however, only the researchers will have access to your original data. Results of this study may be reported in peer reviewed journals, conference presentations, reports and students theses. In all cases only group results will be reported in reports, publications and presentations. It will not be possible for anyone to identify you or your responses. The video material may be used for research,

training and supervision purposes. The video focus will be on the treatment guide; therefore, if participants are captured at all it will be from behind. Voices may be audible but only first names will be identified in the video.

Will I be able to find out the results of the project?
If you would like to be informed of the aggregate research findings at completion of the study, please send an email requesting the results to Associate Professor Leah Brennan at Leah.Brennan@acu.edu.au.

Who do I contact if I have questions about the project?
If you have any questions or would like more information, please call or email Associate Professor Leah Brennan on 9953 3662 or Leah.Brennan@acu.edu.au.
What if I have a complaint or any concerns?

The study has been reviewed by the Human Research Ethics Committee at Australian Catholic University (approval number 2014 ***). If you have any complaints or concerns about the conduct of the project, you may write to Manager, Ethics c/o Office of the Deputy Vice Chancellor (Research).
Manager, Ethics
c/o Office of the Deputy Vice Chancellor (Research)
Australian Catholic University
North Sydney Campus
PO Box 968
NORTH SYDNEY, NSW, 2059
Ph: 02 9739 2519
Fax: 02 9739 2870
Email: res.ethics@acu.edu.au

Any complaint or concern will be treated in confidence and fully investigated. You will be informed of the outcome.

I want to participate! How do I sign up?
To participate in the study, please sign both consent forms, and return the form marked "Copy for the Researcher". The other copy is for you to keep for your records. Please include your contact details where indicated so that the researchers can contact you on receipt of your consent form. Please mail your consent form in the attached self-addressed envelope to:

Associate Professor Leah Brennan
School of Psychology
Australian Catholic University
Melbourne Campus (St Patrick's)
Locked Bag 4115
Fitzroy MDC

Appendix G: Consent form for Study 3

CONSENT FORM
Copy for Participant to Keep

TITLE OF PROJECT: Pathways to Calm and Healthy Eating
PRINCIPAL INVESTIGATOR: Associate Professor Leah Brennan
INVESTIGATOR: Dr Xochitl De la Piedad Garcia
INVESTIGATOR: Mr Jake Linardon

I ..... *(the participant)* have read and understood the information provided in the Participant Information Letter. Any questions I have asked have been answered to my satisfaction.

I agree to participate in this research study involving participating in a guided self-help intervention that will consist of reading and activities related to the self-help book "*Overcoming Binge Eating" (2nd Edition)* or "*If not dieting, then what"?,* and eight, 30-50 minute face-to-face guidance sessions conducted weekly at the Melbourne Psychology and Counselling Clinic at ACU. I give my permission for these sessions to be video-taped for research, training and supervision purposes.

In addition,
I agree to be contacted for a brief phone interview to assess my eligibility for this study.
I agree to complete questionnaires asking me about my weight, eating and activity, physical and mental health, and general well-being (approximately 90 minutes) prior to commencing the study, at completion of the study and at 6-month and 24-month follow-ups.
I agree to undertake a treatment assessment interview (approximately 60 to 90 minutes) prior to me commencing the study.
I agree to complete a brief questionnaire designed to assess eating behaviour at the beginning of each guided session (approximately 3-5 minutes)
I understand that my participation is voluntary, that I can choose not to participate in part or all of the project, and that I can withdraw at any stage of the project without being penalised or disadvantaged in any way.

I agree that research data collected for the study may be used in reports or published findings, or may be used in future research projects, and understand that this information will not contain names or identifying characteristics, and that I will not be able to be identified in any way.

NAME OF PARTICIPANT: ……

Phone number: …… Email address: ……..

Postal Address
……
…….

SIGNATURE: ..... DATE:

.....

SIGNATURE OF PRINCIPAL INVESTIGATOR/INVESTIGATOR: ……....

DATE: ……

Appendix H: Statement of contribution

Publication 1:

The Efficacy of Cognitive-Behavioural Therapy for Eating Disorders: A Systematic Review and Meta-Analysis.

Status: Accepted for publication in Journal of Consulting and Clinical Psychology.

Statement of Contribution of Others

I acknowledge that my contribution to the above paper is 60%.

……..

Jake Linardon

I acknowledge that my contribution to the above paper is about 20%.

A/Prof Leah Brennan

I acknowledge that my contribution to the above paper is about 20%.

Dr Xochitl de la Piedad Garcia

Publication 2:

Predictors, Moderators, and Mediators of Treatment Outcome Following Manualised Cognitive-Behavioural Therapy for Eating Disorders: A Systematic Review.

Status: Accepted for publication in the European Eating Disorders Review

Statement of Contribution of Others

I acknowledge that my contribution to the above paper is 60%.

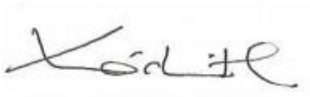
… ……..

Jake Linardon

I acknowledge that my contribution to the above paper is about 20%.

A/Prof Leah Brennan

I acknowledge that my contribution to the above paper is about 20%.



Dr Xochitl de la Piedad Garcia

Article 3:

Evaluating an Expanded Cognitive-Behavioural Model of Bulimia Nervosa: The Role of Body Checking, Body Avoidance, and Dichotomous Thinking

Status: Under Review in Eating Behaviors

Statement of Contribution of Others

I acknowledge that my contribution to the above paper is 60%.

……..

Jake Linardon

I acknowledge that my contribution to the above paper is about 20%.

A/Prof Leah Brennan

I acknowledge that my contribution to the above paper is about 20%.

Dr Xochitl de la Piedad Garcia

Your manuscript EATBEH_2017_65_R2 has been sent for review

EB

Eating Behaviors <EviseSupport@elsevier.com>

Mon 7/3, 12:29 AM

Jake Linardon

Reply all

Inbox

This message was sent automatically. Please do not reply.

Reference: EATBEH_2017_65_R2
Title: Evaluating an expanded cognitive-behavioural model of bulimia nervosa: The role of body checking, body avoidance, and dichotomous thinking
Journal: Eating Behaviors

Dear Mr. Linardon,

I am currently identifying and contacting reviewers who are acknowledged experts in the field. Since peer review is a voluntary service it can take time to find reviewers who are both qualified and available. While reviewers are being contacted, the status of your manuscript will appear in EVISE® as 'Reviewer Invited'.

Once a reviewer agrees to review your manuscript, the status will change to 'Under Review'. When I have received the required number of expert reviews, the status will change to 'Ready for Decision' while I evaluate the reviews before making a decision on your manuscript.

To track the status of your manuscript, please log into EVISE® and go to 'My Submissions' via: http://www.evise.com/evise/faces/pages/navigation/NavController.jspx?JRNL_ACR=EATBEH

Kind regards,

Eating Behaviors

Article 4:

Exploring mechanisms of change during a cognitive-behavioural guided self-help intervention for disordered eating: A single case experimental design.

Status: In preparation

Statement of Contribution of Others

I acknowledge that my contribution to the above paper is 60%.

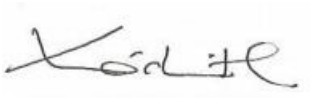
……..

Jake Linardon

I acknowledge that my contribution to the above paper is about 20%.

A/Prof Leah Brennan

I acknowledge that my contribution to the above paper is about 20%.



Dr Xochitl de la Piedad Garcia

Appendix I: Ethics approval for Study 3 and Study 4

AUSTRALIAN CATHOLIC UNIVERSITY

Human Research Ethics Committee
Approval Form

Principal Investigator/Supervisor: Dr Leah Brennan

Co-Investigators: Dr Xochitl De la Piedad Garcia

Student Researcher: Mr Jake Linardon (HDR student)

Ethics approval has been granted for the following project:
An evaluation of the cognitive-behavioural maintenance model of disordered eating

for the period: 31/12/2016

Human Research Ethics Committee (HREC) Register Number: 2015-46E

This is to certify that the above application has been reviewed by the Australian Catholic University Human Research Ethics Committee (ACU HREC). The application has been approved for the period given above.

Researchers are responsible for ensuring that all conditions of approval are adhered to, that they seek prior approval for any modifications and that they notify the HREC of any incidents or unexpected issues impacting on participants that arise in the course of their research. Researchers are also responsible for ensuring that they adhere to the requirements of the *National Statement on Ethical Conduct in Human Research,* the *Australian Code for the Responsible Conduct of Research* and the University's *Code of Conduct*.

Any queries relating to this application should be directed to the Research Ethics Manager (resethics.manager@acu.edu.au).

Kind regards

Date 6/09/2017
Research Ethics Manager

Research Ethics | Office of the Deputy Vice-Chancellor (Research)
Australian Catholic University
T: +61 2 9739 2646
E: Res.Ethics@acu.edu.au
W: ACU Research Ethics

AUSTRALIAN CATHOLIC UNIVERSITY

Human Research Ethics Committee
Approval Form

Principal Investigator/Supervisor: Dr Leah Brennan

Co-Investigators: Dr Xochitl De la Piedad Garcia

Student Researcher: Mr Jake Linardon (HDR student)

Ethics approval has been granted for the following project:
The efficacy of Cognitive-Behavioural and Health at Every Size guided self-help interventions for disordered eating.

for the period: 31/12/2017

Human Research Ethics Committee (HREC) Register Number: 2015-58H

This is to certify that the above application has been reviewed by the Australian Catholic University Human Research Ethics Committee (ACU HREC). The application has been approved for the period given above.

Researchers are responsible for ensuring that all conditions of approval are adhered to, that they seek prior approval for any modifications and that they notify the HREC of any incidents or unexpected issues impacting on participants that arise in the course of their research. Researchers are also responsible for ensuring that they adhere to the requirements of the *National Statement on Ethical Conduct in Human Research,* the *Australian Code for the Responsible Conduct of Research* and the University's *Code of Conduct*.

Any queries relating to this application should be directed to the Research Ethics Manager (resethics.manager@acu.edu.au).

Kind regards

Date 6/09/2017

Research Ethics | Office of the Deputy Vice-Chancellor (Research)
Australian Catholic University
T: +61 2 9739 2646
E: Res.Ethics@acu.edu.au
W: ACU Research Ethics

Appendix J: Copies of Assessment Instruments Used

EATING QUESTIONNAIRE

Instructions: The following questions are concerned with the past four weeks (28 days) only. Please read each question carefully. Please answer all the questions. Thank you.

Questions 1 to 12: Please circle the appropriate number on the right. Remember that the questions only refer to the past four weeks (28 days) only.

.

	On how many of the past 28 days …..	No days	1-5 days	6-12 days	13-15 days	16-22 days	23-27 days	Every day
1	Have you been deliberately trying to limit the amount of food you eat to influence your shape or weight (whether or not you have succeeded)?	0	1	2	3	4	5	6
2	Have you gone for long periods of time (8 waking hours or more) without eating anything at all in order to influence your shape or weight?	0	1	2	3	4	5	6
3	Have you tried to exclude from your diet any foods that you like in order to influence your shape or weight (whether or not you have succeeded)?	0	1	2	3	4	5	6
4	Have you tried to follow definite rules regarding your eating (for example, a calorie limit) in order to influence your shape or weight (whether or not you have succeeded)?	0	1	2	3	4	5	6
5	Have you had a definite desire to have an empty stomach with the aim of influencing your shape or weight?	0	1	2	3	4	5	6
6	Have you had a definite desire to have a totally flat stomach?	0	1	2	3	4	5	6
7	Has thinking about food, eating or calories made it very difficult to concentrate on things you are interested in (for example, working, following a conversation, or reading)?	0	1	2	3	4	5	6
8	Has thinking about shape or weight made it very difficult to concentrate on things you are interested in (for example, working, following a conversation, or reading)?	0	1	2	3	4	5	6
9	Have you had a definite fear of losing control over eating?	0	1	2	3	4	5	6
10	Have you had a definite fear that you might gain weight?	0	1	2	3	4	5	6
11	Have you felt fat?	0	1	2	3	4	5	6
12	Have you had a strong desire to lose weight?	0	1	2	3	4	5	6

Questions 13-18: Please fill in the appropriate number in the boxes on the right. Remember that the questions only refer to the past four weeks (28 days).

Over the past four weeks (28 days) ……

13 Over the past 28 days, how many times have you eaten what other people would regard as an unusually large amount of food (given the circumstances)?	……
14 ….. On how many of these times did you have a sense of having lost control over your eating (at the time that you were eating)?	……
15 Over the past 28 days, on how many **DAYS** have such episodes of overeating occurred (i.e., you have eaten an unusually large amount of food and have had a sense of loss of control at the time)?	……
16 Over the past 28 days, how many times have you made yourself sick (vomit) as a means of controlling your shape or weight?	……
17 Over the past 28 days, how many times have you taken laxatives as a means of controlling your shape or weight?	……
18 Over the past 28 days, how many times have you exercised in a "driven" or "compulsive" way as a means of controlling your weight, shape or amount of fat, or to burn off calories?	……

Questions 19 to 21: Please circle the appropriate number. Please note that for these questions the term "binge eating" means eating what others would regard as an unusually large amount of food for the circumstances, accompanied by a sense of having lost control over eating.

19 Over the past 28 days, on how many days have you eaten in secret (ie, furtively)? ….. Do not count episodes of binge eating	No days	1-5 days	6-12 days	13-15 days	16-22 days	23-27 days	Every day
	0	1	2	3	4	5	6
20 On what proportion of the times that you have eaten have you felt guilty (felt that you've done wrong) because of its effect on your shape or weight? ….. Do not count episodes of binge eating	None of the times	A few of the times	Less than half	Half of the times	More than half	Most of the time	Every time
	0	1	2	3	4	5	6
21 Over the past 28 days, how concerned have you been about other people seeing you eat? ….. Do not count episodes of binge eating	Not at all		Slightly		Moderately		Markedly
	0	1	2	3	4	5	6

Questions 22 to 28: Please circle the appropriate number on the right. Remember that the questions only refer to the past four weeks (28 days).

	Over the past 28 days …..	**Not at all**		**Slightly**		**Moderate -ly**		**Markedly**
22	Has your weight influenced how you think about (judge) yourself as a person?	0	1	2	3	4	5	6
23	Has your shape influenced how you think about (judge) yourself as a person?	0	1	2	3	4	5	6
24	How much would it have upset you if you had been asked to weigh yourself once a week (no more, or less, often) for the next four weeks?	0	1	2	3	4	5	6
25	How dissatisfied have you been with your weight?	0	1	2	3	4	5	6
26	How dissatisfied have you been with your shape?	0	1	2	3	4	5	6
27	How uncomfortable have you felt seeing your body (for example, seeing your shape in the mirror, in a shop window reflection, while undressing or taking a bath or shower)?	0	1	2	3	4	5	6
28	How uncomfortable have you felt about others seeing your shape or figure (for example, in communal changing rooms, when swimming, or wearing tight clothes)?	0	1	2	3	4	5	6

What is your weight at present? (Please give your best estimate.) .....

What is your height? (Please give your best estimate.) .....

If female: Over the past three-to-four months have you missed any menstrual periods? .....

If so, how many? .....

Have you been taking the "pill"? .....

THANK YOU

APPENDIX

Body Checking Questionnaire

Circle the number which best describes how often you engage in these behaviors at the present time.

1 = never
2 = rarely
3 = sometimes
4 = often
5 = very often

Table Appendix *(Continued)*

1. I check to see if my thighs spread when I'm sitting down.	1	2	3	4	5
2. I pinch my stomach to measure fatness.	1	2	3	4	5
3. I have special clothes which I try on to make sure they still fit.	1	2	3	4	5
4. I check the diameter of my wrist to make sure it's the same size as before.	1	2	3	4	5
5.1 check my reflection in glass doors or car windows to see how I look.	1	2	3	4	5
6. I pinch my upper arms to measure fatness.	1	2	3	4	5
7. I touch underneath my chin to make sure I don't have a "double chir."	1	2	3	4	5
8. I look at others to see how my body size compares to their body size.	1	2	3	4	5
9. I rub (or touch) my thighs while sitting to check for fatness.	1	2	3	4	5
10. I check the diameter of my legs to make they're the same size as before.	1	2	3	4	5
11. I ask others about their weight or clothing size so I can compare my own weight/size.	1	2	3	4	5
12. I check to see how my bottom looks in the mirror.	1	2	3	4	5
13.1 practice sitting and standing in various positions to see how I would look in each position.	1	2	3	4	5
14. I check to see if my thighs rub together.	1	2	3	4	5
15. I try to elicit comments from others about how fat I am.	1	2	3	4	5
16. I check to see if my fat jiggles.	1	2	3	4	5
17. I suck in my gut to see what it is like when my stomach is completely flat.	1	2	3	4	5
18. I check to mare sure my rings fit the same way as before,	1	2	3	4	5
19. I look to see if I have cellulite on my thighs when I am sitting.	1	2	3	4	5
20. I lie down on the floor to see if I can feel my bones touch the floor.	1	2	3	4	5
21. I pull my clothes as tightly as possible around myself to see how I look.	1	2	3	4	5
22. I compare myself to models on TV or in magazines.	1	2	3	4	5
23. I pinch my cheeks to measure fatness.	1	2	3	4	5

Note: The BCQ is in questionnaire format and can therefore be administered in either individual or group settings. Completion time for the measure is approximately 5-10 minutes. To score the total BCQ, simply sum all the items. To calculate the overall appearance scale, sum the following items: 3, 5, 8, 11,12, 13, 15, 17, 21, 22 . To calculate the specific body parts scale, sum the following items: 1, 2, 6, 9,10, 14, 16, 19. To calculate the idiosyncratic checking scale, sum the following items: 4, 7, 18, 20,23.

Body Image Avoidance Questionnaire

Circle the number which best describes how often you engage in these behaviors at the present time.

	Always	Usually	Often	Sometimes	Rarely	Never
1. I wear baggy clothes	5	4	3	2	1	0
2. I wear clothes I do not like	5	4	3	2	1	0
3. I wear darker color clothing	5	4	3	2	1	0
4. I wear a special set of clothing, e.g., my "fat clothes"	5	4	3	2	1	0
5. I restrict the amount of food I eat	5	4	3	2	1	0
6. I only eat fruits, vegetables and other low calorie foods	5	4	3	2	1	0
7. I fast for a day or longer	5	4	3	2	1	0
8. I do not go out socially if I will be "checked out"	5	4	3	2	1	0
9. I do not go out socially if the people I am with will discuss weight	5	4	3	2	1	0
10. I do not go out socially if the people I am with are thinner than me	5	4	3	2	1	0
11. I do not go out socially if it involves eating	5	4	3	2	1	0
12. I weigh myself	5	4	3	2	1	0
13. I am inactive	5	4	3	2	1	0
14. I look at myself in the mirror	5	4	3	2	1	0
15. I avoid physical intimacy	5	4	3	2	1	0
16. I wear clothes that will divert attention from my weight	5	4	3	2	1	0
17. I avoid going clothes shopping	5	4	3	2	1	0
18. I don't wear "revealing" clothes (e.g., bathingsuits, tank tops, or shorts)	5	4	3	2	1	0
19. I get dressed up or made up	5	4	3	2	1	0

Appendix A. Dichotomous Thinking in Eating Disorders Scale-11 (DTEDS-11)

PLEASE READ EACH OF THE FOLLOWING STATEMENTS AND DECIDE HOW TRUE IT IS OF YOUR THINKING OVER THE PAST MONTH. If it is not true of you at all, circle 1; if it is slightly true of you, circle 2; if it is fairly true of you, circle 3; and if it is very true of you, circle 4.

	Not at all true of me	Slightly true of me	Fairly true of me	Very true of me
1. (1) I think of food as either "good" or "bad"	1	2	3	4
2. (3) I think of things in "black and white" terms	1	2	3	4
3. (6) I think of myself as either good or bad	1	2	3	4
4. (7) I view my attempts to diet as either successes or failures	1	2	3	4
5. (8) I think of myself as either in control or out of control	1	2	3	4
6. (9) When dieting, if I eat something that I had planned not to, I think that I have failed	1	2	3	4
7. (10) I think of myself as either clever or stupid	1	2	3	4
8. (11) When dieting, I view my eating as having been either good or bad	1	2	3	4
9. (12) I either get on very well with people or not at all	1	2	3	4
10. (14) I think of myself as either ugly or good-looking	1	2	3	4
11. (16) I think of myself as doing things either very well or very badly	1	2	3	4

Note: Numbers in parentheses indicate item number in the original scale. Eating subscale = items 1 (1), 4 (7), 6 (9), 8 (11); General subscale = items 2 (3), 3 (6), 5 (8), 7 (10), 9 (12), 10 (14), 11 (16).

Eating habits checklist

Instructions. Below are groups of numbered statements. Read all of the statements in each group and mark on this sheet the one that best describes the way you feel about the problems you have controlling your eating behavior.

#1

(0) 1. I don't feel self-conscious about my weight or body size when I'm with others.
(0) 2. I feel concerned about how I look to others, but it normally does not make me feel disappointed with myself.
(1) 3. I do get self-conscious about my appearance and weight which makes me feel disappointed in myself.
(3) 4. I feel very self-conscious about my weight and frequently, I feel intense shame and disgust for myself. I try to avoid social contacts because of my self-consciousness.

#2

(0) 1. I don't have any difficulty eating slowly in the proper manner.
(1) 2. Although I seem to "gobble down" foods, I don't end up feeling stuffed because of eating too much.
(2) 3. At times, I tend to eat quickly and then, I feel uncomfortably full afterwards.
(3) 4. I have the habit of bolting down my food, without really chewing it. When this happens I usually feel uncomfortably stuffed because I've eaten too much.

#3

(0) 1. I feel capable to control my eating urges when I want to.
(1) 2. I feel like I have failed to control my eating more than the average person.
(3) 3. I feel utterly helpless when it comes to feeling in control of my eating urges.
(3) 4. Because I feel so helpless about controlling my eating I have become very desperate about trying to get in control.

#4

(0) 1. I don't have the habit of eating when I'm bored.
(0) 2. I sometimes eat when I'm bored, but often I'm able to "get busy" and get my mind off food.
(0) 3. I have a regular habit of eating when I'm bored, but occasionally, I can use some other activity to get my mind off eating.
(2) 4. I have a strong habit of eating when I'm bored. Nothing seems to help me break the habit.

#5

(0) 1. I'm usually physically hungry when I eat something.
(1) 2. Occasionally, I eat something on impulse even though I really am not hungry.
(2) 3. I have the regular habit of eating foods, that I might not really enjoy, to satisfy a hungry feeling even though physically, I don't need the food.
(3) 4. Even though I'm not physically hungry, I get a hungry feeling in my mouth that only seems to be satisfied when I eat a food, like a sandwich, that fills my mouth. Sometimes, when I eat the food to satisfy my mouth hunger, I then spit the food out so I won't gain weight.

#6

(0) 1. I don't feel any guilt or self-hate after I overeat.
(1) 2. After I overeat, occasionally I feel guilt or self-hate.
(3) 3. Almost all the time I experience strong guilt or self-hate after I overeat.

#7

(0) 1. I don't lose total control of my eating when dieting even after periods when I overeat.
(2) 2. Sometimes when I eat a "forbidden food" on a diet, I feel like I "blew it" and eat even more.
(3) 3. Frequently, I have the habit of saying to myself, "I've blown it now, why not go all the way" when I overeat on a diet. When that happens I eat even more.
(3) 4. I have a regular habit of starting strict diets for myself, but I break the diets by going on an eating binge. My life seems to be either a "feast" or "famine."

#8

(0) 1. I rarely eat so much food that I feel uncomfortably stuffed afterwards.
(1) 2. Usually about once a month, I eat such a quantity of food, I end up feeling very stuffed.
(2) 3. I have regular periods during the month when I eat large amounts of food, either at mealtime or at snacks.
(3) 4. I eat so much food that I regularly feel quite uncomfortable after eating and sometimes a bit nauseous.

#9

(0) 1. My level of calorie intake does not go up very high or go down very low on a regular basis.
(1) 2. Sometimes after I overeat, I will try to reduce my caloric intake to almost nothing to compensate for the excess calories I've eaten.
(2) 3. I have a regular habit of overeating during the night. It seems that my routine is not to be hungry in the morning but overeat in the evening.
(3) 4. In my adult years, I have had week-long periods where I practically starve myself. This follows periods when I overeat. It seems I live a life of either "feast or famine."

#10

(0) 1. I usually am able to stop eating when I want to. I know when "enough is enough."
(1) 2. Every so often, I experience a compulsion to eat which I can't seem to control.
(2) 3. Frequently, I experience strong urges to eat which I seem unable to control, but at other times I can control my eating urges.
(3) 4. I feel incapable of controlling urges to eat. I have a fear of not being able to stop eating voluntarily.

#11

(0) 1. I don't have any problem stopping eating when I feel full.
(1) 2. I usually can stop eating when I feel full but occasionally overeat leaving me feeling uncomfortably stuffed.

(2) 3. I have a problem stopping eating once I start and usually I feel uncomfortably stuffed after I eat a meal.
(3) 4. Because I have a problem not being able to stop eating when I want, I sometimes have to induce vomiting to relieve my stuffed feeling.

#12
(0) 1. I seem to eat just as much when I'm with others (family, social gatherings) as when I'm by myself.
(1) 2. Sometimes, when I'm with other persons, I don't eat as much as I want to eat because I'm self-conscious about my eating.
(2) 3. Frequently, I eat only a small amount of food when others are present, because I'm very embarrassed about my eating.
(3) 4. I feel so ashamed about overeating that I pick times to overeat when I know no one will see me. I feel like a "closet eater."

#13
(0) 1. I eat three meals a day with only an occasional between meal snack.
(0) 2. I eat 3 meals a day, but I also normally snack between meals.
(2) 3. When I am snacking heavily, I get in the habit of skipping regular meals.
(3) 4. There are regular periods when I seem to be continually eating, with no planned meals.

#14
(0) 1. I don't think much about trying to control unwanted eating urges.
(1) 2. At least some of the time, I feel my thoughts are pre-occupied with trying to control my eating urges.
(2) 3. I feel that frequently I spend much time thinking about how much I ate or about trying not to eat anymore.
(3) 4. It seems to me that most of my waking hours are pre-occupied by thoughts about eating *or* not eating. I feel like I'm constantly struggling not to eat.

#15
(0) 1. I don't think about food a great deal.
(1) 2. I have strong cravings for food but they last only for brief periods of time.
(2) 3. I have days when I can't seem to think about anything else but food.
(3) 4. Most of my days seem to be pre-occupied with thoughts about food. I feel like I live to eat.

#16
(0) 1. I usually know whether or not I'm physically hungry. I take the right portion of food to satisy me.
(1) 2. Occasionally, I feel uncertain about knowing whether or not I'm physically hungry. At these times it's hard to know how much food I should take to satisfy me.
(2) 3. Even though I might know how many calories I should eat, I don't have any idea what is a "normal" amount of food for me.

INSTRUCTIONS

Please place an 'X' in the column which best describes how your eating habits, exercising or feelings about your eating, shape or weight have affected your life over the past four weeks (28 days). Thank you.

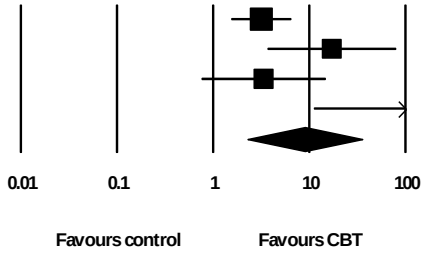
	Over the past 28 days, to what extent have your …eating habits …exercising or feelings about your eating, shape or weight …	Not at all	A little	Quite a bit	A lot
1	... made it difficult to concentrate?				
2	... made you feel critical of yourself?				
3	... stopped you going out with others?				
4	... affected your work performance (if applicable)?				
5	... made you forgetful?				
6	... affected your ability to make everyday decisions?				
7	... interfered with meals with family or friends?				
8	... made you upset?				
9	... made you feel ashamed of yourself?				
10	... made it difficult to eat out with others?				
11	... made you feel guilty?				
12	... interfered with you doing things you used to enjoy?				
13	... made you absent-minded?				
14	... made you feel a failure?				
15	... interfered with your relationships with others?				
16	... made you worry?				

Appendix K: Forest Plots from Meta-Analysis

Remission rates

Therapist-led CBT for BN versus control at post tx

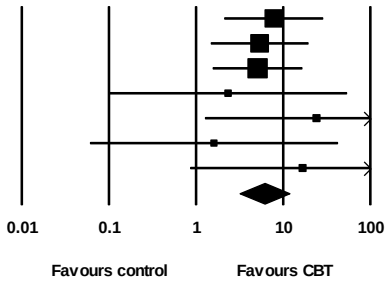
Study name	Comparison	Time point	Outcome	Odds ratio	Lower limit	Upper limit	p-Value
Davis a	WL	Blank	BN	3.170	1.529	6.569	0.002
Griffiths WL	WL	Blank	BN	17.154	3.641	80.818	0.000
Treasure TL	WL	Blank	BN	3.353	0.751	14.976	0.113
Telch	WL	Blank	BN	199.234	11.070	3585.597	0.000
				8.893	2.252	35.123	0.002



Meta Analysis

Therapist-led CBT for BED versus control at post tx

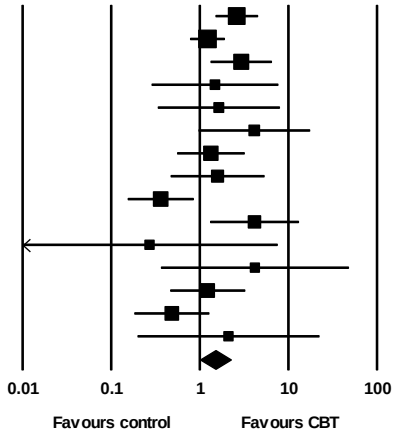
Study name	Comparison	Time point	Outcome	Odds ratio	Lower limit	Upper limit	p-Value
Dingemans	WL	Blank	BED	7.779	2.095	28.881	0.002
Gorin S CBT	WL	Blank	BED	5.367	1.468	19.622	0.011
Peterson 09 TL	WL	Blank	BED	5.079	1.544	16.711	0.007
Peterson 98 TL	WL	Blank	BED	2.333	0.100	54.421	0.598
Schlup 09	WL	Blank	BED	24.124	1.257	462.852	0.035
Shapiro TL	WL	Blank	BED	1.605	0.060	42.719	0.777
Wilfley 93 WL	WL	Blank	BED	16.693	0.852	327.188	0.064
				6.064	3.137	11.721	0.000



Meta Analysis

Therapist-led CBT for BN versus active comparison at post tx

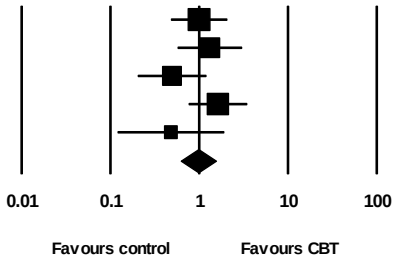
Study name	Comparison	Time point	Outcome	Odds ratio	Lower limit	Upper limit	p-Value
Agras 2000	AC	IPT	BN	2.615	1.497	4.570	0.001
Cooper a	AC	BT	BN	1.221	0.772	1.931	0.394
Fairburn 15	AC	IPT	BN	2.937	1.315	6.559	0.009
Fairburn 91 BT	AC	BT	BN	1.485	0.284	7.756	0.639
Faribum 91 IPT	AC	IPT	BN	1.641	0.334	8.067	0.542
Garner	AC	Blank	BN	4.123	0.960	17.710	0.057
Griffiths 94 BT	AC	BT	BN	1.334	0.551	3.234	0.523
Hsu	AC	Blank	BN	1.588	0.465	5.419	0.460
Le Grange	AC	Blank	BN	0.363	0.153	0.861	0.021
Poulsen	AC	Blank	BN	4.145	1.301	13.210	0.016
Thackway BT	AC	BT	BN	0.271	0.010	7.604	0.443
Thackway NS	AC	Blank	BN	4.191	0.362	48.535	0.252
Walsh 97 SET	AC	Blank	BN	1.227	0.460	3.274	0.682
Wonderlich	AC	Blank	BN	0.484	0.181	1.291	0.147
Lavender	AC	Blank	BN	2.107	0.197	22.571	0.538
				1.493	1.001	2.226	0.049



Meta Analysis

Therapist-led CBT for BED versus active comparison at post tx

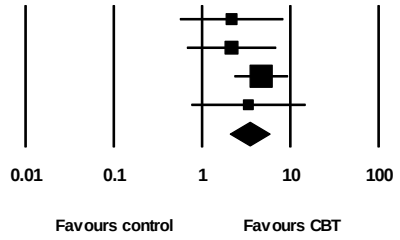
Study name	Comparison	Time point	Outcome	Odds ratio	Lower limit	Upper limit	p-Value
Chen 16	AC	Blank	BED	0.998	0.478	2.084	0.996
Grilo 11	AC	BWL	BED	1.317	0.567	3.059	0.521
Munsch	AC	BWL	BED	0.495	0.202	1.212	0.124
Wilfley 02	AC	IPT	BED	1.626	0.757	3.492	0.213
Wilfley 93 IPT	AC	IPT	BED	0.481	0.120	1.929	0.301
				0.971	0.616	1.532	0.900



Meta Analysis

Self-help CBT for BN versus control at post tx

Study name	Comparison	Time point	Outcome	Odds ratio	Lower limit	Upper limit	p-Value
Treasure PSH	WL	Blank	BN	2.162	0.558	8.376	0.265
Sanchez	WL	Blank	BN	2.158	0.670	6.949	0.197
Banasiak	WL	Blank	BN	4.673	2.303	9.480	0.000
Travis	WL	Blank	BN	3.353	0.751	14.976	0.113
				3.446	2.052	5.787	0.000

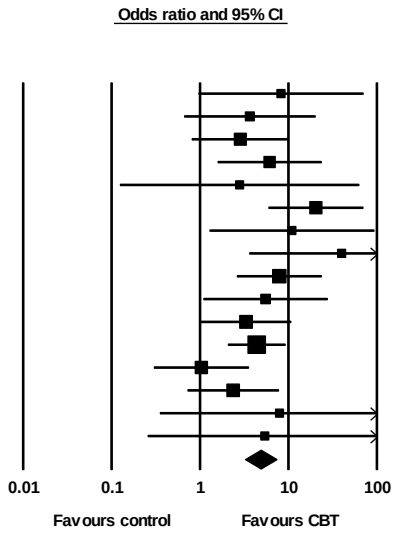


Meta Analysis

Self-help CBT for BED versus control at post tx

Study name	Comparison	Time point	Outcome	Odds ratio	Lower limit	Upper limit	p-Value
Carter 98 PSH	WL	Blank	BED	8.244	0.956	71.077	0.055
Grilo 13	WL	Blank	BED	3.664	0.658	20.404	0.138
Kristella WL	WL	Blank	BED	2.858	0.801	10.205	0.106
Carrard	WL	Blank	BED	6.134	1.572	23.935	0.009
Shapiro GSH	WL	Blank	BED	2.807	0.124	63.645	0.517
Wagner	WL	Blank	BED	20.454	5.894	70.981	0.000
Carter 98 GSH	WL	Blank	BED	10.960	1.271	94.494	0.029
Debar	WL	Blank	BED	40.000	3.579	447.034	0.003
DeBar	WL	Blank	BED	7.893	2.594	24.014	0.000
Grilo 05 WL	WL	Blank	BED	5.521	1.088	28.029	0.039
Ljottson	WL	Blank	BED	3.317	1.015	10.834	0.047
Striegel-Moore	WL	Blank	BED	4.377	2.053	9.334	0.000
Peterson 09 GSH	WL	Blank	BED	1.037	0.299	3.599	0.954
Peterson 09 TA	WL	Blank	BED	2.375	0.717	7.871	0.157
Peterson 98 GSH	WL	Blank	BED	7.936	0.350	179.942	0.193
Peterson 98 TA	WL	Blank	BED	5.402	0.254	114.906	0.280
				4.826	3.203	7.272	0.000

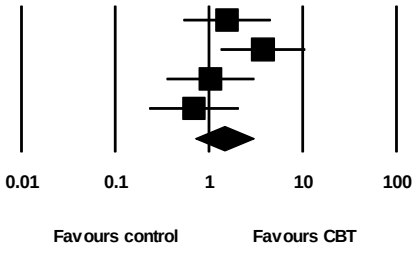
Statistics for each study



Meta Analysis

Self-help CBT for BED versus active comparison at post tx

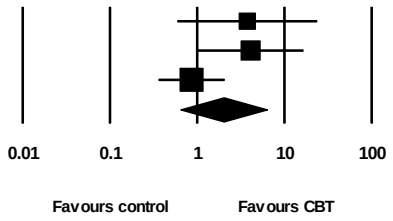
Study name	Comparison	Time point	Outcome	Odds ratio	Lower limit	Upper limit	p-Value
Kristella Mind	AC	Blank	BED	1.560	0.529	4.596	0.420
Grilo 05 BWL	AC	BWL	BED	3.766	1.324	10.709	0.013
Wilson 10 BWL	AC	BWL	BED	1.039	0.351	3.072	0.945
Wilson 10 IPT	AC	IPT	BED	0.692	0.229	2.090	0.514
				1.454	0.710	2.977	0.306



Meta Analysis

Therapist-led CBT for BN versus pharmacotherapy at post tx

Study name	Comparison	Time point	Outcome	Odds ratio	Lower limit	Upper limit	p-Value
Goldbloom 97	MED	Blank	BN	3.752	0.589	23.913	0.162
Jacobi	MED	Blank	BN	4.101	1.011	16.640	0.048
Walsh 97 Med	MED	Blank	BN	0.863	0.359	2.077	0.743
				1.999	0.637	6.274	0.235

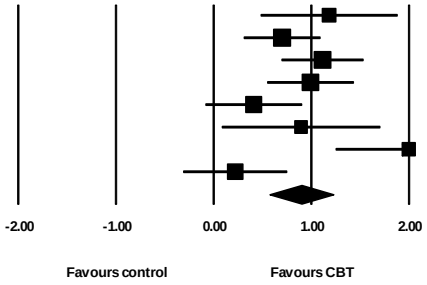


Meta Analysis

Binge/purge frequency outcomes

Therapist-led CBT for BN versus control on binge/purge frequency at post tx

Studyname	Comparison	Outcome	Hedges's g	Standard error	Variance	Lower limit	Upper limit	Z-Value	p-Value
Agras 89	WL	BN	1.182	0.360	0.129	0.478	1.887	3.288	0.001
Davis	WL	BN	0.701	0.202	0.041	0.305	1.097	3.468	0.001
Freeman 88	WL	BN	1.115	0.216	0.047	0.692	1.538	5.169	0.000
Griffiths	WL	BN	0.992	0.228	0.052	0.546	1.438	4.358	0.000
Lee 86	WL	BN	0.411	0.254	0.064	-0.087	0.908	1.617	0.106
Leitenberg	WL	BN	0.895	0.415	0.172	0.081	1.709	2.156	0.031
Telch	WL	BN	1.998	0.383	0.147	1.246	2.749	5.212	0.000
Wolf	WL	BN	0.221	0.273	0.075	-0.314	0.756	0.809	0.419
			0.895	0.166	0.028	0.569	1.222	5.379	0.000

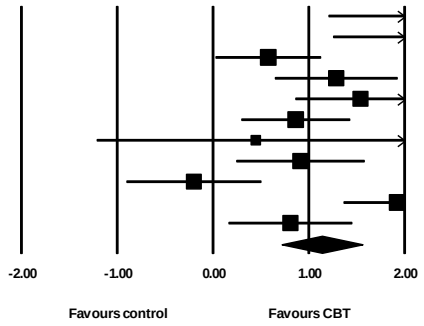


Meta Analysis

Therapist-led CBT for BED versus control on binge/purge frequency at post tx

Study name	Comparison	Outcome	Hedges's g	Standard error	Variance	Lower limit	Upper limit	Z-Value	p-Value
Agras 95	WL	BED	2.002	0.408	0.167	1.203	2.802	4.906	0.000
Allen 94	WL	BED	2.369	0.571	0.326	1.251	3.488	4.151	0.000
Dingemans	WL	BED	0.577	0.283	0.080	0.023	1.131	2.042	0.041
Gorin	WL	BED	1.286	0.329	0.108	0.643	1.930	3.916	0.000
Gorin S CBT	WL	BED	1.540	0.348	0.121	0.857	2.222	4.420	0.000
Peterson 09 TL	WL	BED	0.864	0.291	0.085	0.293	1.435	2.966	0.003
Peterson 98 TL	WL	BED	0.447	0.849	0.720	-1.216	2.110	0.527	0.598
Schlup	WL	BED	0.912	0.343	0.118	0.240	1.585	2.658	0.008
Shapiro TL	WL	BED	-0.200	0.361	0.130	-0.907	0.507	-0.554	0.580
Tasca	WL	BED	1.922	0.287	0.082	1.360	2.485	6.703	0.000
Wilfley 93	WL	BED	0.808	0.331	0.110	0.159	1.456	2.441	0.015
			1.135	0.215	0.046	0.713	1.556	5.276	0.000

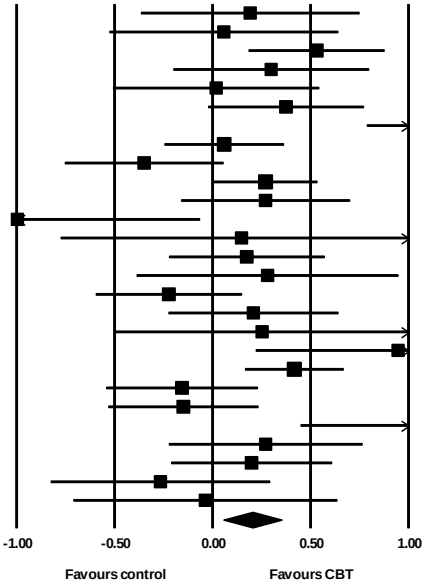
Statistics for each study



Meta Analysis

Therapist-led CBT for BN versus active comparison on binge/purge frequency at post tx

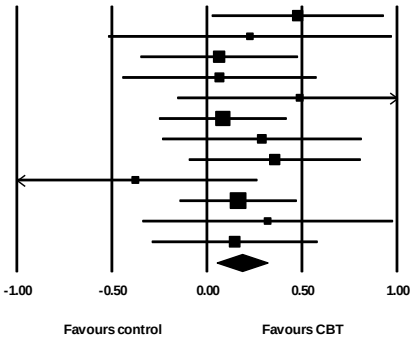
Study name	Comparison	Outcome	Hedges's g	Standard error	Variance	Lower limit	Upper limit	Z-Value	p-Value
Steele Mind	Blank	BN	0.192	0.285	0.081	-0.367	0.752	0.675	0.500
Steele Perf	Blank	BN	0.058	0.299	0.089	-0.528	0.643	0.194	0.847
Agras 2000	IPT	BN	0.531	0.178	0.032	0.182	0.880	2.983	0.003
Fairburn 91	IPT	BN	0.300	0.256	0.065	-0.201	0.800	1.172	0.241
Fairburn 91b	BT	BN	0.018	0.269	0.072	-0.509	0.546	0.069	0.945
Garner	Blank	BN	0.375	0.204	0.042	-0.024	0.774	1.841	0.066
Poulsen	Blank	BN	1.140	0.181	0.033	0.786	1.495	6.301	0.000
Wonderlich	Blank	BN	0.059	0.156	0.024	-0.247	0.366	0.380	0.704
Freeman	BT	BN	-0.349	0.207	0.043	-0.755	0.058	-1.682	0.092
Cooper	BT	BN	0.271	0.136	0.018	0.004	0.537	1.993	0.046
Griffiths	BT	BN	0.271	0.221	0.049	-0.162	0.703	1.227	0.220
Thachrway	BT	BN	-0.995	0.476	0.227	-1.928	-0.062	-2.091	0.037
Thackrway NS	Blank	BN	0.148	0.472	0.223	-0.777	1.073	0.314	0.754
Walsh 97	Blank	BN	0.175	0.204	0.041	-0.224	0.574	0.860	0.390
Agras	Blank	BN	0.281	0.342	0.117	-0.389	0.950	0.822	0.411
Le Grange	Blank	BN	-0.223	0.191	0.037	-0.598	0.152	-1.165	0.244
Stefani	Blank	BN	0.209	0.222	0.049	-0.227	0.644	0.939	0.348
Sundgot PA	Blank	BN	0.253	0.383	0.146	-0.497	1.003	0.662	0.508
Sundgot NC	Blank	BN	0.947	0.372	0.138	0.219	1.675	2.550	0.011
Fairburn 15	IPT	BN	0.417	0.129	0.017	0.164	0.670	3.231	0.001
Mcintosh CBT-A	Blank	BN	-0.156	0.198	0.039	-0.544	0.233	-0.786	0.432
Mcintosh CBT-BN	Blank	BN	-0.149	0.196	0.039	-0.534	0.236	-0.759	0.448
Kirkly	Blank	BN	1.066	0.316	0.100	0.447	1.684	3.378	0.001
Wolf	BT	BN	0.271	0.253	0.064	-0.224	0.767	1.073	0.283
Lavender	Blank	BN	0.199	0.210	0.044	-0.213	0.611	0.949	0.343
Leitenberg	BT	BN	-0.266	0.286	0.082	-0.827	0.295	-0.928	0.353
Tantillino	Blank	BN	-0.037	0.344	0.119	-0.712	0.638	-0.107	0.915
			0.202	0.076	0.006	0.054	0.351	2.675	0.007



Meta Analysis

Therapist-led CBT for BED versus active comparison on binge/purge frequency at post tx

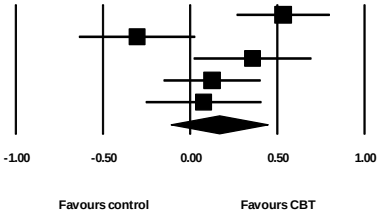
Study name	Comparison	Outcome	Hedges's g	Standard error	Variance	Lower limit	Upper limit	Z-Value	p-Value
Grilo 05	BWL	BED	0.478	0.232	0.054	0.024	0.932	2.064	0.039
Kelly	Blank	BED	0.227	0.382	0.146	-0.521	0.974	0.594	0.553
Wilson 10	BWL	BED	0.064	0.212	0.045	-0.352	0.481	0.304	0.761
Kristella	Blank	BED	0.066	0.261	0.068	-0.446	0.579	0.253	0.800
Nauta	Blank	BED	0.489	0.330	0.109	-0.157	1.135	1.482	0.138
Chen	Blank	BED	0.084	0.172	0.030	-0.253	0.421	0.489	0.625
Grilo 11	BWL	BED	0.290	0.269	0.072	-0.237	0.816	1.077	0.281
Tasca	Blank	BED	0.357	0.232	0.054	-0.097	0.811	1.543	0.123
Wilfley 93	IPT	BED	-0.376	0.329	0.108	-1.020	0.267	-1.146	0.252
Wilfley 02	IPT	BED	0.164	0.158	0.025	-0.146	0.474	1.038	0.299
Kenardy	Blank	BED	0.320	0.337	0.114	-0.341	0.981	0.949	0.343
Muncsh	BWL	BED	0.147	0.223	0.050	-0.291	0.584	0.656	0.512
			0.184	0.068	0.005	0.051	0.317	2.702	0.007



Meta Analysis

Self-help CBT for BN versus control on binge/purge frequency at post tx

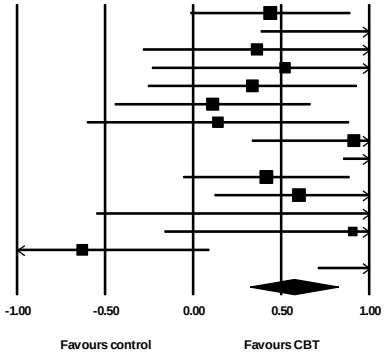
Study name	Comparison	Outcome	Hedges's g	Standard error	Variance	Lower limit	Upper limit	Z-Value	p-Value
Banasiak	WL	BN	0.533	0.137	0.019	0.265	0.802	3.891	0.000
Durand	WL	BN	-0.304	0.171	0.029	-0.640	0.031	-1.780	0.075
Sanchez	WL	BN	0.358	0.173	0.030	0.019	0.697	2.069	0.039
Schmidt	WL	BN	0.126	0.143	0.020	-0.154	0.406	0.882	0.378
Travis	WL	BN	0.077	0.170	0.029	-0.256	0.410	0.453	0.650
			0.165	0.142	0.020	-0.113	0.443	1.163	0.245



Meta Analysis

Self-help CBT for BED versus control on binge/purge frequency at post tx

Study name	Comparison	Outcome	Hedges's g	Standard error	Variance	Lower limit	Upper limit	Z-Value	p-Value
Carrard	WL	BED	0.438	0.233	0.054	-0.017	0.894	1.885	0.059
Carter 98 GSH	WL	BED	1.064	0.348	0.121	0.383	1.746	3.059	0.002
Carter 98 PSH	WL	BED	0.363	0.331	0.110	-0.287	1.012	1.095	0.274
Debar	WL	BED	0.523	0.387	0.150	-0.235	1.281	1.352	0.176
Grilo 05	WL	BED	0.337	0.303	0.092	-0.258	0.931	1.110	0.267
Grilo 13	WL	BED	0.111	0.284	0.081	-0.446	0.668	0.391	0.696
Kelly 15	WL	BED	0.141	0.381	0.145	-0.605	0.887	0.372	0.710
Kristella 14	WL	BED	0.913	0.296	0.088	0.333	1.494	3.083	0.002
Ljotsson 1	WL	BED	1.225	0.191	0.036	0.851	1.599	6.420	0.000
Peterson 09 GSH	WL	BED	0.416	0.242	0.059	-0.058	0.891	1.721	0.085
Peterson 09 TA	WL	BED	0.602	0.246	0.060	0.120	1.083	2.448	0.014
Peterson 98 GSH	WL	BED	1.088	0.836	0.699	-0.551	2.727	1.301	0.193
Peterson 98 TA	WL	BED	0.907	0.547	0.299	-0.164	1.978	1.660	0.097
Shapiro SH	WL	BED	-0.629	0.369	0.136	-1.352	0.094	-1.706	0.088
Wagner	WL	BED	1.060	0.180	0.032	0.707	1.413	5.890	0.000
			0.572	0.128	0.016	0.322	0.822	4.481	0.000

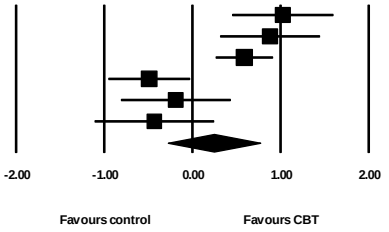


Meta Analysis

Cognitive Symptoms

Therapist-led CBT for BED versus control on cognitive symptoms at post tx

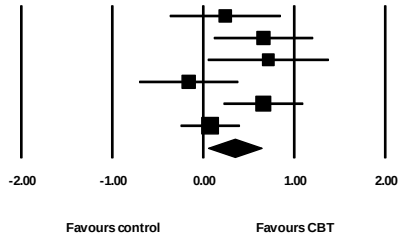
Study name	Comparison	Outcome	Hedges's g	Standard error	Variance	Lower limit	Upper limit	Z-Value	p-Value
Dingemans	WL	BED	1.026	0.294	0.087	0.449	1.603	3.485	0.000
Peterson 09 TL	WL	BED	0.881	0.290	0.084	0.312	1.450	3.034	0.002
Schlup 09	WL	BED	0.590	0.167	0.028	0.262	0.917	3.526	0.000
Tasca	WL	BED	-0.490	0.238	0.057	-0.957	-0.023	-2.058	0.040
Wilfley 93	WL	BED	-0.188	0.319	0.102	-0.812	0.437	-0.589	0.556
Agras 95	WL	BED	-0.432	0.347	0.121	-1.112	0.249	-1.243	0.214
			0.242	0.267	0.072	-0.282	0.766	0.906	0.365



Meta Analysis

Therapist-led CBT for BN versus control on cognitive symptoms at post tx

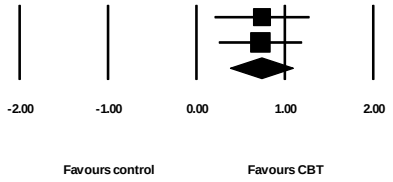
Studyname	Comparison	Outcome	Hedges's g	Standard error	Variance	Lower limit	Upper limit	Z-Value	p-Value
Telch	WL	BN	0.242	0.313	0.098	-0.371	0.855	0.774	0.439
Wolf	WL	BN	0.662	0.280	0.078	0.114	1.211	2.367	0.018
Agras 89	WL	BN	0.715	0.341	0.116	0.047	1.384	2.097	0.036
Davis 99	WL	BN	-0.160	0.279	0.078	-0.707	0.387	-0.572	0.567
Grifiths 94	WL	BN	0.660	0.225	0.050	0.220	1.101	2.939	0.003
Treasure	WL	BN	0.076	0.168	0.028	-0.252	0.404	0.453	0.650
			0.344	0.150	0.023	0.049	0.639	2.287	0.022



Meta Analysis

Therapist-led CBT for BED versus pharmacotherapy on cognitive symptoms at post tx

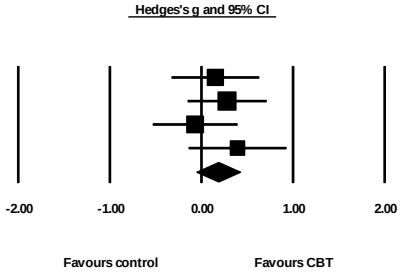
Studyname	Comparison	Outcome	Hedges's g	Standard error	Variance	Lower limit	Upper limit	Z-Value	p-Value
Grilo 05b	M	BED	0.742	0.275	0.076	0.203	1.281	2.699	0.007
Ricca	M	BED	0.724	0.241	0.058	0.251	1.198	3.000	0.003
			0.732	0.181	0.033	0.377	1.088	4.035	0.000



Meta Analysis

Therapist-led CBT for BN versus pharmacotherapy on cognitive symptoms at post tx

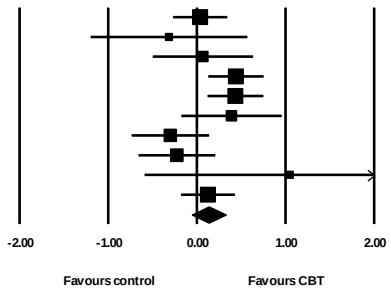
Studyname	Comparison	Outcome	Statistics for each study						
			Hedges's g	Standard error	Variance	Lower limit	Upper limit	Z-Value	p-Value
Agras 92	M	BN	0.152	0.246	0.060	-0.329	0.634	0.620	0.535
Goldbloom	M	BN	0.280	0.222	0.049	-0.155	0.715	1.262	0.207
Jacobi 02	M	BN	-0.068	0.237	0.056	-0.534	0.397	-0.288	0.773
Walsh 97	M	BN	0.394	0.274	0.075	-0.143	0.931	1.439	0.150
			0.180	0.121	0.015	-0.057	0.418	1.486	0.137



Meta Analysis

Therapist-led CBT for AN versus active comparison on cognitive symptoms at post tx

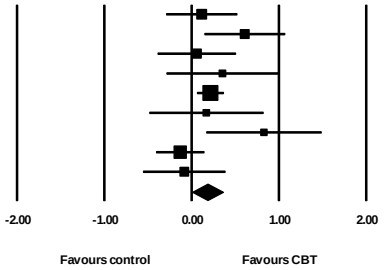
Studyname	Comparison	Outcome	Hedges's g	Standard error	Variance	Lower limit	Upper limit	Z-Value	p-Value
Zipfel	Blank	AN	0.037	0.157	0.025	-0.271	0.345	0.234	0.815
Ball 04	Blank	AN	-0.314	0.452	0.205	-1.201	0.572	-0.695	0.487
Lock 13	Blank	AN	0.069	0.290	0.084	-0.499	0.637	0.237	0.812
McIntosh 05 IPT	IPT	AN	0.441	0.161	0.026	0.126	0.755	2.745	0.006
McIntosh 05 SM	Blank	AN	0.435	0.162	0.026	0.118	0.752	2.689	0.007
Touyz	Blank	AN	0.391	0.290	0.084	-0.178	0.960	1.347	0.178
Byrne 2017	Blank	AN	-0.299	0.224	0.050	-0.738	0.140	-1.334	0.182
Byrne 2017MANT	Blank	AN	-0.225	0.222	0.049	-0.661	0.210	-1.014	0.310
Pike	Blank	AN	1.048	0.837	0.701	-0.592	2.689	1.252	0.210
Zipfel TAU	Blank	AN	0.125	0.156	0.024	-0.181	0.432	0.803	0.422
			0.131	0.097	0.009	-0.059	0.322	1.350	0.177



Meta Analysis

Therapist-led CBT for BED versus active comparison on cognitive symptoms at post tx

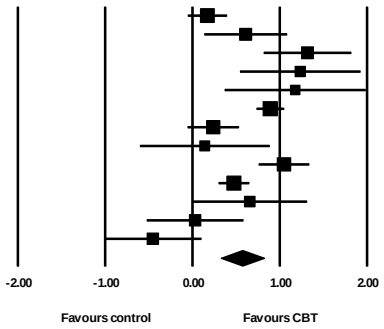
Studyname	Comparison	Outcome	Hedges's g	Standard error	Variance	Lower limit	Upper limit	Z-Value	p-Value
Grilo 11	BWL	BED	0.116	0.209	0.044	-0.294	0.526	0.555	0.579
Agras	BWL	BED	0.609	0.237	0.056	0.144	1.075	2.567	0.010
Tasca	Blank	BED	0.059	0.231	0.053	-0.393	0.511	0.258	0.797
Wilfley 93	IPT	BED	0.353	0.329	0.108	-0.291	0.997	1.074	0.283
Wilfley 2002	IPT	BED	0.216	0.080	0.006	0.060	0.372	2.713	0.007
Kenardy	Blank	BED	0.169	0.336	0.113	-0.488	0.827	0.505	0.614
Nauta	Blank	BED	0.829	0.339	0.115	0.165	1.492	2.447	0.014
Munsch	BWL	BED	-0.130	0.143	0.020	-0.409	0.150	-0.910	0.363
Chen	Blank	BED	-0.084	0.242	0.059	-0.559	0.391	-0.347	0.729
			0.179	0.089	0.008	0.004	0.355	2.004	0.045



Meta Analysis

Self-help CBT for BED versus control on cognitive symptoms at post tx

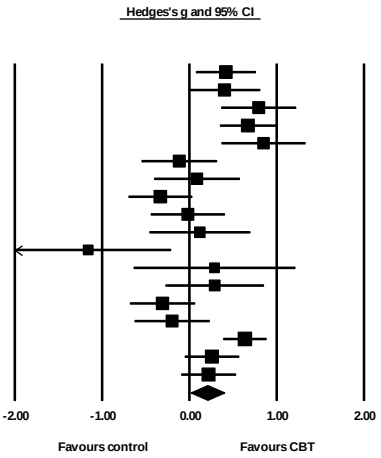
Study name	Comparison	Outcome	Hedges's g	Standard error	Variance	Lower limit	Upper limit	Z-Value	p-Value
Carrard 11	WL	BED	0.171	0.116	0.013	-0.056	0.398	1.479	0.139
Peterson 09 GSH	WL	BED	0.610	0.244	0.059	0.132	1.088	2.500	0.012
Peterson 09 TA	WL	BED	1.318	0.258	0.066	0.813	1.823	5.115	0.000
Carter GSH	WL	BED	1.235	0.354	0.125	0.542	1.929	3.492	0.000
Debar	WL	BED	1.177	0.413	0.171	0.367	1.987	2.847	0.004
DeBar	WL	BED	0.892	0.083	0.007	0.730	1.054	10.795	0.000
Grilo 05	WL	BED	0.238	0.152	0.023	-0.059	0.536	1.571	0.116
Kelly	WL	BED	0.141	0.381	0.145	-0.605	0.887	0.372	0.710
Ljottson	WL	BED	1.049	0.149	0.022	0.756	1.341	7.026	0.000
Striegel-Morre	WL	BED	0.475	0.091	0.008	0.296	0.654	5.196	0.000
Carter PSH	WL	BED	0.657	0.336	0.113	-0.002	1.316	1.953	0.051
Grilo 13	WL	BED	0.030	0.284	0.081	-0.527	0.588	0.107	0.915
Kristella	WL	BED	-0.454	0.285	0.081	-1.013	0.106	-1.590	0.112
			0.569	0.126	0.016	0.322	0.816	4.521	0.000



Meta Analysis

Therapist-led CBT for BN versus active comparison on cognitive symptoms at post tx

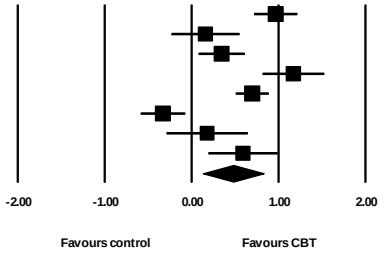
Studyname	Comparison	Outcome	Hedges's g	Standard error	Variance	Lower limit	Upper limit	Z-Value	p-Value
Agras 2000	IPT	BN	0.419	0.177	0.031	0.072	0.765	2.365	0.018
Fairburn 91 IPT	IPT	BN	0.403	0.210	0.044	-0.008	0.814	1.921	0.055
Fairburn 91 BT	BT	BN	0.794	0.221	0.049	0.362	1.227	3.599	0.000
Garner 93	Blank	BN	0.671	0.165	0.027	0.347	0.995	4.060	0.000
Poulsen	Blank	BN	0.849	0.247	0.061	0.365	1.334	3.436	0.001
Wonderlich	Blank	BN	-0.116	0.222	0.049	-0.551	0.319	-0.522	0.601
Wolf	BT	BN	0.088	0.252	0.064	-0.406	0.581	0.347	0.728
Cooper 1995	BT	BN	-0.333	0.188	0.035	-0.701	0.036	-1.768	0.077
Griffiths	BT	BN	-0.018	0.218	0.048	-0.445	0.410	-0.080	0.936
Lavender	Blank	BN	0.120	0.297	0.088	-0.461	0.701	0.404	0.686
Thackray BT	BT	BN	-1.159	0.486	0.236	-2.111	-0.207	-2.387	0.017
Thackray NS	Blank	BN	0.288	0.474	0.224	-0.641	1.216	0.607	0.544
Walsh 97	Blank	BN	0.290	0.289	0.084	-0.277	0.857	1.003	0.316
LEGRANGE	Blank	BN	-0.308	0.192	0.037	-0.684	0.068	-1.605	0.108
Stefani	Blank	BN	-0.198	0.221	0.049	-0.631	0.235	-0.897	0.370
Fairburn 15	IPT	BN	0.636	0.128	0.016	0.385	0.887	4.961	0.000
McIntosh 16 CBBN	Blank	BN	0.258	0.161	0.026	-0.057	0.574	1.607	0.108
McIntosh 16 CBTA	Blank	BN	0.220	0.162	0.026	-0.097	0.537	1.360	0.174
			0.204	0.098	0.010	0.012	0.396	2.085	0.037



Meta Analysis

Self-help CBT for BN versus control on cognitive symptoms at post tx

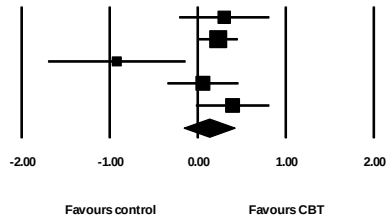
Study name	Comparison	Outcome	Hedges's g	Standard error	Variance	Lower limit	Upper limit	Z-Value	p-Value
Sanchez 11	WL	BN	0.968	0.128	0.017	0.716	1.220	7.531	0.000
Schmidt	WL	BN	0.160	0.201	0.041	-0.235	0.554	0.793	0.428
Ter humme	WL	BN	0.346	0.138	0.019	0.076	0.615	2.514	0.012
Wagner	WL	BN	1.172	0.183	0.033	0.813	1.530	6.402	0.000
Banasiak	WL	BN	0.698	0.098	0.010	0.505	0.891	7.101	0.000
Carter 03	WL	BN	-0.329	0.132	0.017	-0.588	-0.070	-2.493	0.013
Durand	WL	BN	0.179	0.240	0.058	-0.292	0.650	0.745	0.456
Treasure GSH	WL	BN	0.591	0.205	0.042	0.189	0.992	2.885	0.004
			0.476	0.178	0.032	0.127	0.825	2.676	0.007



Meta Analysis

Self-help CBT for BED versus active comparison on cognitive symptoms at post tx

	Comparison	Outcome	Hedges's g	Standard error	Variance	Lower limit	Upper limit	Z-Value	p-Value
Kristella	Blank	BED	0.301	0.263	0.069	-0.214	0.816	1.146	0.252
Grilo 05	BWL	BED	0.234	0.115	0.013	0.009	0.459	2.034	0.042
Kelly 15	Blank	BED	-0.919	0.400	0.160	-1.703	-0.135	-2.298	0.022
Wilson IPT	IPT	BED	0.060	0.208	0.043	-0.347	0.466	0.287	0.774
Wilson BWL	BWL	BED	0.397	0.214	0.046	-0.023	0.817	1.852	0.064
			0.127	0.147	0.022	-0.162	0.416	0.862	0.389



Meta Analysis