# The cognitive-behavioural theory and treatment of bulimia nervosa:

# An examination of treatment mechanisms and future directions

Running Head: BULIMIA NERVOSA

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## Abstract

Enhanced cognitive-behavioural therapy (CBT-E) is the current treatment of choice for bulimia nervosa. While the cognitive-behavioural theory and treatment of bulimia nervosa have made a substantial contribution to our understanding of the disorder, approximately half of patients treated with CBT-E fail to achieve remission of binge eating and purging. There is evidence showing that mechanisms proposed by the CBT-E model are associated with binge eating and purging symptoms, and therefore likely important targets for treatment. To identify future directions in improving the efficacy of this treatment, and informed by a model of the client change process, we review the evidence for the hypothesised treatment mechanisms of CBT-E. We conclude that while the proposed treatment mechanisms of CBT-E largely change over the course of treatment, there is limited evidence that the treatment manipulations of CBT-E are responsible for the specific changes in the proposed treatment mechanisms. In addition, given a lack of research in this area, we could find no evidence that changes in the additional treatment mechanisms outlined in CBT-E are associated with changes in the core symptomatology of binge eating and purging. Based on these findings we recommend that future efforts are directed towards understanding the client change process in CBT-E and outline three clear directions for research.

# The cognitive-behavioural theory and treatment of bulimia nervosa: An examination of treatment mechanisms and future directions

It is estimated that approximately 1 in 35 Australian women will be affected by bulimia nervosa (BN) in their lifetime (Wade, Bergin, Tiggemann, Bulik, & Fairburn, 2006), resulting in significant social, mental and physical health impairments. BN is characterised by repeated episodes of objective binge eating (i.e., consumption of a large amount of food in a discrete period of time, accompanied by a sense of loss of control over eating) and the use of compensatory behaviours intended to prevent weight gain following binge eating episodes (e.g., self-induced vomiting, misuse of laxatives and diuretics, fasting, and compulsive exercise). Understanding the cognitions, emotions, and behaviours that maintain BN is central to the design and delivery of effective psychological treatments.

To this end, the cognitive-behavioural theory of BN outlines the factors that are hypothesised to maintain this disorder. A specific form of cognitive-behaviour therapy (CBT-BN) has been developed to directly target the proposed maintaining mechanisms outlined in the original cognitive-behavioural model of BN (Fairburn, Marcus, & Wilson, 1993). This treatment is recognised as the front-running treatment for BN (National Institute of Clinical Excellence, 2004). Randomised controlled trials (RCTs) evaluating this treatment have observed that approximately 30 to 50% of patients experience remission of binge eating and purging following treatment (Agras, Walsh, Fairburn, Wilson, & Kraemer, 2000; Bulik, Sullivan, Carter, McIntosh, & Joyce, 1998; Cooper & Steere, 1995; Fairburn, Jones, Peveler, Hope, & O'Connor, 1993; Fairburn et al., 1991; Garner et al., 1993).

This original model was more recently expanded to include four additional factors (mood intolerance, perfectionism, interpersonal problems, and low self-esteem) that have broad empirical support in the maintenance of BN (Figure 1). An enhanced CBT (CBT-E) treatment for BN (and all eating disorders) has been devised based upon this enhanced cognitive-behavioural model (Fairburn, Cooper, & Shafran, 2003), which targets these factors with additional treatment modules. Despite the additional modules, RCTs of CBT-E indicate that limitations in treatment response are still observed. At the end of treatment, trials have observed abstinence rates from binge eating and purging of 42 to 47% (Fairburn et al., 2009; Poulsen et al., 2014) for "focused" CBT-E (which did not include additional modules for mood intolerance, perfectionism, interpersonal problems, and low self-esteem), 22.5% for "focused" CBT-E plus the additional mood intolerance module only (Wonderlich et al., 2014) and 49% (Fairburn et al., 2009) for "broad" CBT-E (which includes all four additional treatment modules). At follow-up, abstinence rates are similar, ranging from 22.5 to 54% (Fairburn et al., 2009; Poulsen et al., 2014; Wonderlich et al., 2014; Fairburn et al., 2009). These abstinence rates are noteworthy, as they are approximately equivalent to rates observed in earlier trials of the original CBT-BN, indicating that advances in the formulation of maintaining mechanisms of BN have not translated into improved treatment outcomes. In addition, in the only trial that presented relapse data (Poulsen et al., 2014), 33% of those who were abstinent from binge eating and purging at the end of CBT-E had re-initiated these behaviours at follow-up. Specific evidence of the limited efficacy of the additional CBT-E modules as currently delivered was found in a dismantling study where patients were either assigned to "broad" CBT-E or "focused" CBT-E (Fairburn et al., 2009). Overall, there was no benefit in the broad CBT-E on change in eating disorder symptoms from pre-treatment to post-treatment. Some support for the additional modules was found in

subsequent exploratory analyses, with a trend showing an advantage of broad CBT-E for patients who had at least two additional "complex" clinical problems (i.e., mood intolerance, perfectionism, interpersonal problems, or low self-esteem). However, this benefit was very small ($d$ = .13) and non-significant. Thus there is limited evidence for the collective benefit of the additional CBT-E components.

**Understanding the client change process of CBT-E**

Given limitations in treatment response and limited evidence for the efficacy of the additional treatment modules in CBT-E, we need to consider how to better translate the cognitive-behavioural model of BN into improved treatment outcomes; an issue that will form the focus of this review. It has been argued that it is essential to examine mechanisms of change and identify the 'active ingredients' of a treatment, in order to optimise treatment outcomes (Kazdin, 2007; Murphy, Cooper, Hollon, & Fairburn, 2009). Hollon and Kriss (1984) outline a useful framework for examining this issue in their model of the client change process. In this conceptualisation, *treatment manipulations* (e.g., cognitive or behavioural techniques) lead to changes in *treatment mechanisms*, which in turn lead to changes in *clinical symptoms*. A treatment mechanism is a client process that mediates change in relevant client symptoms. For example, in CBT-E, the mood intolerance module (treatment manipulation) is proposed to lead to changes in mood intolerance (treatment mechanism) which in turn leads to reductions in binge eating (clinical symptom).

Informed by this model, we argue that in order to refine CBT-E treatment in a manner that improves treatment outcomes, we must determine: (i) the extent to which treatment mechanisms change over the course of CBT-E; (ii) whether these changes in treatment

mechanism correspond to reductions in symptom, and (iii) the extent to which treatment manipulations in CBT-E for BN are associated with change in the targeted treatment mechanisms. The answers to these questions are pertinent because if the putative mechanism is not associated with symptom reduction, then it is not necessary to target the mechanism in order to change the core symptoms of binge eating and purging. Secondly, if the treatment manipulations do not confer a specific benefit on treatment mechanisms, then they could be replaced with techniques that do have a specific effect on such mechanisms.

**The original cognitive-behavioural model of the maintenance of bulimia nervosa**

The original cognitive-behavioural model of BN (Fairburn, Cooper, & Cooper, 1986) outlines the core psychopathology of BN as a dysfunctional system of self-evaluation, whereby control over weight, shape, or eating is the primary criterion for the evaluation of self-worth. It is proposed that over-evaluating the importance of weight, shape or eating promotes dietary restraint, which involves the cognitive control of eating. Those with high levels of dietary restraint attempt to guide dietary intake with strict and inflexible dietary rules. Informed by restraint theory (Polivy & Herman, 1985), the model outlines multiple pathways by which this dietary restraint maintains binge eating. First, attempting to maintain cognitive control over eating leaves the individual vulnerable to episodes of disinhibited eating when this cognitive control is disrupted, for example during heightened emotional states. Second, hunger and related physiological mechanisms are proposed to override cognitive control resulting in a loss of control over eating. Third, transgression from rigid dietary rules may invoke the abstinence violation effect (Marlatt & Gordon, 1985), whereby eating is construed as a catastrophic loss of control, which promotes permissive thoughts about further overeating. Following binge eating, dietary restraint and

compensatory behaviours, including purging (self-induced vomiting and/or laxative misuse), fasting, or compulsive exercise, are prompted by fears about the effect of the binge on weight and shape. The belief that compensatory behaviour prevents weight gain following a binge episode removes a future deterrent against binge eating and contributes to a pattern of repeated binge eating and purging.

**Treatment mechanisms in cognitive-behavioural therapy for bulimia nervosa**

As formulated in the cognitive-behavioural model of BN, client change in over-evaluation of weight and shape and dietary restraint are two key treatment mechanisms of CBT-BN. Changes in these factors have been shown to relate to changes in symptoms, including reductions in binge eating and purging. A prospective study of the natural course of BN in a community sample over a 15 month period found that higher over-evaluation of weight and shape at baseline was associated with a greater increase in dietary restraint, which in turn was significantly associated with a simultaneous increase in binge-eating episodes (Fairburn, Stice, et al., 2003). In addition, two cross-sectional investigations using structural equation modelling in clinical BN samples have also largely supported the model, jointly providing support for the relationship between over-evaluation of weight and shape, dietary restraint, and binge eating in BN (Lampard, Byrne, McLean, & Fursland, 2011; Lampard, Tasca, Balfour, & Bissada, 2013).

Studies of mediators of symptom change in CBT-BN have also largely supported these treatment mechanisms. Early change in dietary restraint during treatment mediates the benefit of CBT treatment (relative to IPT) on the change in binge eating and purging frequency at post-treatment (Wilson, Fairburn, Agras, Walsh, & Kraemer, 2002), and change in body-related

dysfunctional beliefs is associated with decreases in weight concern over the course of treatment (Spangler, Baldwin, & Agras, 2004).

*Treatment manipulations*

While these treatment mechanisms have been supported, the treatment manipulations hypothesised to lead to changes in these treatment mechanisms have not been largely investigated. One study, however, failed to support the proposed treatment manipulations in CBT-BN. Specifically, Spangler et al. (2004) found that the application of cognitive techniques was not associated with change in body-related dysfunctional beliefs, nor was the application of behavioural techniques related to changes in dietary restraint.

## The enhanced cognitive-behavioural model of the maintenance of bulimia nervosa

The enhanced cognitive-behavioural theory of eating disorders (Fairburn, Cooper, & Shafran, 2003) builds on the original cognitive-behavioural model of BN and outlines how, in some individuals, the core maintaining factors and symptoms of the disorder (i.e., over-evaluation of weight and shape, dietary restraint, binge eating, and purging) are themselves maintained by four additional factors: mood intolerance, clinical perfectionism, core low self-esteem, and interpersonal problems.

The CBT-E model of BN proposes that one of the primary functions of binge eating and purging is to regulate emotions and that the impulse to regulate emotions by binge eating or purging is related to the degree to which the experience of emotion (either positive or negative) is perceived as intolerable (*mood intolerance*). This formulation greatly expands on the role of mood and emotions within the original model of CBT-BN, where adverse mood was considered to act primarily by disrupting dietary restraint thus indirectly contributing to binge episodes.

Clinical perfectionism refers to the general tendency to strive for excessively high personal standards in all aspects of life (Fairburn, Cooper, & Shafran, 2003). This tendency to strive for high standards becomes maladaptive when the pursuit of these rigid standards continues in spite of significant adverse consequences. Clinical perfectionism is proposed to maintain eating disorders in two ways. First, the negative effects of the over-evaluation of the importance of weight and shape are exacerbated by having perfectionist standards in the domain of weight and shape control. Second, the negative effects of dietary restraint are exacerbated by perfectionist standards in the domain of control of eating.

Core low self-esteem in the CBT-E model refers to a pervasive and unconditional negative view of self-worth. Processes related to self-worth are a defining feature of BN; specifically that self-worth is overly dependent, sometimes almost exclusively, on the evaluation of weight and shape (i.e., overevaluation of weight and shape). Judging self-worth by these limited criteria makes low self-esteem especially likely amongst people with BN, particularly when combined with perfectionistic standards of weight and shape. However, 'core' low self-esteem specified in the enhanced theory differs from this over-evaluation mechanism as it involves pervasive, unconditional beliefs about low self-worth.

Finally, interpersonal problems are proposed to maintain the binge eating and purging symptoms of BN through two pathways. First, interpersonal problems, or heightened sensitivity to perceiving interpersonal problems, lead to negative mood states, which further prime binge eating and purging episodes in an attempt to regulate emotion (Steiger, Gauvin, Jabalpurwala, Séguin, & Stotland, 1999). Second, repeated experience of interpersonal problems further exacerbates and contributes to the development of core low self-esteem.

## The additional treatment mechanisms in enhanced cognitive-behavioural therapy for bulimia nervosa

There is broad support for the relationship between the additional treatment mechanisms and BN symptoms from two cross-sectional modelling studies in BN treatment seekers. The first study found that low self-esteem was associated with over-evaluation of weight and shape, and that greater interpersonal problems were associated with greater dietary restraint (Lampard et al., 2011). The expected association between mood intolerance and binge eating and purging was not observed, but this was likely due to measurement issues in the assessment of mood intolerance. The second study did observe the expected relationship between mood intolerance and elevated binge eating (Lampard et al., 2013). Neither study, however, observed an association between perfectionism and dietary restraint in BN. Thus there is cross-sectional evidence that mood intolerance, low self-esteem and interpersonal problems are important treatment mechanisms in BN that should be targeted in treatment.

However, importantly, we were unable to identify any studies reporting on the association between change in the additional treatment mechanisms of CBT-E (i.e., mood intolerance, perfectionism, self-esteem and interpersonal problems) and core symptoms of binge eating and purging. Thus, while available evidence indicates that amelioration of these treatment mechanisms would reduce binge eating and purging symptoms, this has not been explicitly demonstrated in treatment research. Without this data it is not possible to determine the causal relationship between the putative mechanisms and binge eating and purging symptoms.

## Treatment manipulations in the enhanced cognitive-behavioural therapy for bulimia nervosa

Treatment manipulations for the additional treatment mechanisms in the enhanced model involve optional treatment modules that are used when these mechanisms are deemed to be obstructing change in client outcomes. Mood intolerance, clinical perfectionism and core low self-esteem are targeted with conventional CBT techniques, while interpersonal problems are targeted with a module based on interpersonal psychotherapy (IPT). The interpersonal problems module focuses on the interpersonal context in which the eating disorder developed and supports the client to identify and address interpersonal problems in a non-directive manner. In order to determine whether these additional treatment manipulations are effective we need to know whether the targeted treatment mechanisms change over the course of CBT-E treatment, and whether the treatment manipulations are responsible for this change.

***I. To what extent do the proposed treatment mechanisms change during CBT-E for BN?***

Three trials have provided data on change in treatment mechanisms over the course of CBT-E for BN: an RCT of 'focused' and 'broad' CBT-E for a non-underweight transdiagnostic eating disorder sample (38% BN; Fairburn et al., 2009); an RCT of a 'focused' CBT-E variant for BN (Wonderlich et al., 2014); and an effectiveness trial of CBT-E in a transdiagnostic eating disorder sample (36% BN; Byrne, Fursland, Allen, & Watson, 2011). Across all trials, changes in dietary restraint were large (*d* range = 1.15 to 1.81). However, the findings regarding mood intolerance were mixed, with one report of no significant changes on three measures of mood intolerance (*d* range = -.33 to .16; Byrne et al., 2011), and one report of a medium effect on a measure of emotion regulation (*d* = .50; Wonderlich et al., 2014). The effectiveness trial was the only broad CBT-E study to report data on interpersonal problems, self-esteem, and perfectionism, observing

large changes in interpersonal problems ($d$ = 1.00) and self-esteem ($d$ = 1.00) and a small non-significant change on perfectionism ($d$ = .13) for treatment completers.

While CBT-E is associated with changes in relevant treatment mechanisms, it is worth noting that residual symptoms may still be an issue. For example, while CBT-E was associated with a medium positive effect on mood intolerance in the Wonderlich et al. (2014) study, patients still had moderate to high levels of mood intolerance post treatment (end of treatment: $d$ = .63; follow up: $d$ = .76) relative to previously reported non-clinical student samples (Gratz, 2004). Similarly, self-esteem following CBT-E (Byrne et al., 2011) was lower post treatment ($d$ = .47) compared to population estimates (Schmitt & Allik, 2005). In addition, approximately 33% of treatment completers in the Byrne et al. study had interpersonal problem scores within the clinical range post-treatment (Woodward, Murrell, & Bettler, 2005). It is therefore possible that for some people, these treatment mechanisms are insufficiently addressed during treatment.

Residual symptoms in these treatment mechanisms have implications for clinical outcomes. Mood intolerance has been shown to predict treatment dropout from CBT-E for eating disorders (Carter et al., 2012). One trial comparing two versions of CBT for BN observed a non-significant trend whereby those patients who relapsed by 6 months post-treatment had achieved less improvement in self-esteem during treatment than those who did not relapse (Ghaderi, 2006; Welch & Ghaderi, 2013). Similarly, emotion regulation (Safer, Lively, Telch, & Agras, 2002) and interpersonal problems (Keel, Dorer, Franko, Jackson, & Herzog, 2005; Olmsted, Kaplan, & Rockert, 1994) are associated with relapse following psychological treatment for eating disorders. This latter finding is also supported by a qualitative analysis of relapse following treatment for anorexia nervosa, where patients described difficulties tolerating negative affect as a factor in their relapse (Federici & Kaplan, 2008). These findings suggest that for at least some

clients, unresolved mood intolerance, low self-esteem, and interpersonal problems may be a contributor to poor treatment response and to relapse.

***II. Are the treatment manipulations (i.e., treatment modules) associated with change in the treatment mechanisms of CBT-E?***

There have been no direct tests of the efficacy of each additional CBT-E treatment module in facilitating client change. In the absence of this data, there is some research that can be used to evaluate the extent to which the core low self-esteem and interpersonal problem modules are directly associated with change in the treatment mechanisms of CBT-E.

*Core low self-esteem*

As outlined earlier, there is indication that self-esteem improves following CBT-E, as an open effectiveness trial of CBT-E in a community transdiagnostic outpatient sample observed a large significant improvement in self-esteem (d = 1.00 in treatment completers; 36% BN; Byrne et al., 2011). However, there is some evidence that this change occurs without the additional self-esteem module, as CBT treatments that don't include the CBT-E low self-esteem module have found similarly large improvements in self-esteem (CBT-BN: *d* = .96, Agras et al., 2000; *d* = .65, Leitenberg, Rosen, Gross, Nudelman, & Vara, 1988; *d* = 2.25, Wilson, Eldredge, Smith, & Niles, 1991; focused CBT-E: *d* = .65, Wonderlich et al., 2014). This suggests that while CBT-E improves self-esteem, the additional self-esteem module might not be specifically responsible for this effect. It is possible that the improvement in self-esteem may be due to other aspects of the CBT package, or a downstream effect of the reduction in binge eating or improvement in interpersonal problems.

*Interpersonal Problems*

As outlined earlier, CBT-E targets interpersonal problems with an additional module based on interpersonal psychotherapy (IPT). CBT-E including this IPT module has led to a significant and large improvement in interpersonal problems (d = 1.00) in a transdiagnostic eating disorder sample (Byrne et al., 2011). However, the IPT module may not be responsible for this change. Some indication of the effect of IPT on interpersonal problems in BN can be inferred from RCT studies that compare IPT alone with the original CBT-BN, which does not focus on interpersonal problems. These studies have not shown consistent benefit of IPT on interpersonal problems. Three studies have found no benefit of IPT alone compared to CBT-BN (CBT: *d* = .33 to .36, IPT: *d* = .33 to .76, Between CBT and IPT groups: *d* = .00 to .36; Wilfley, Welch, & Stein, 2002; Wilfley et al. 1993[1]; Wilson et al., 2002). Given IPT alone doesn't show a consistent effect on interpersonal problems, the additional IPT module might not be responsible for the observed change in interpersonal problems following CBT-E. It is possible that the effect of the CBT-E package on interpersonal problems may be due to other factors, such as a down-stream effect of symptom remission, or due to common factors in the therapeutic relationship. The treatment manipulations that lead to change in interpersonal problems during CBT-E remain unclear and require further investigation.

[1] The Wilfley et al. (1993) study observed a small non-significant trend in the pre- to post-treatment scores (*p* = .09) towards greater improvement in interpersonal problems for IPT alone (*d* = .76) relative to CBT-BN (*d* = .36) and a waitlist condition (*d* = .36). This difference was due to slightly higher pre-treatment interpersonal problems in the IPT condition, but there was no difference between the IPT and waitlist groups in post-treatment interpersonal problems (*d* = .00).

**Where to from here? Conclusions and future directions**

Undeniably, the development of CBT-E represents a significant milestone in the effective treatment of BN. Nevertheless, it is clear that improvements in treatment response are still necessary, and to achieve this, we must have a greater understanding of the client change process in CBT-E for BN. The above review can draw two important conclusions, with recommendations for future work. First, there is little evidence that the treatment manipulations in CBT-E are specifically responsible for change in the hypothesised treatment mechanisms. This is largely due to the fact that this type of research has not been conducted in CBT-E. There has been only one dismantling study of CBT-E (Fairburn et al., 2009), showing that overall the additional modules do not confer a benefit in treatment outcome, and there have been no studies that have examined the effect of individual treatment modules on specific treatment mechanisms. Treatment research needs to be conducted to determine whether the application of each additional CBT-E module is specifically associated with changes in the relevant treatment mechanism and symptoms.

Second, given that a proportion of clients do not improve following CBT-E for BN, and that residual mechanisms of mood intolerance, low self-esteem, and interpersonal problems contribute to relapse and treatment dropout, we also need to enhance the active ingredients in CBT-E that target these mechanisms. There are two ways that this can be achieved: (i) determine the active treatment mechanisms in CBT-E and increase the dose of manipulations that target that mechanism (as suggested by Murphy et al., 2009); and (ii) identify alternative treatment manipulations that have been shown to target the treatment mechanisms, potentially drawing from treatment traditions beyond CBT, and incorporate these treatment manipulations into CBT-E.

As has been called for by Murphy et al. (2009) and Nock (2007), further research is required to determine the specific active ingredients in treatment, beyond treatment modules, that lead to change in client cognition or behaviour, and determine how these treatment ingredients lead to change. For example, there is evidence that therapeutic alliance is an important treatment manipulation factor that is associated with downstream change in 'core' treatment mechanisms, including dietary restraint, body-related dysfunctional cognitions, and client engagement in treatment (Spangler, Baldwin, & Agras, 2004; Tasca & Lampard, 2012) and change in BN symptoms (Loeb, Lock, Greif, & le Grange, 2012). What is missing is an understanding of the specific treatment mechanisms within the alliance that are responsible for this effect.

Understanding this treatment process can lead to important treatment refinements. For example, it has been proposed that therapeutic alliance leads to improvements in symptoms via alterations to interpersonal schemata, which is the internal representation of interpersonal interactions (Safran & Segal, 1996; Safran, 1998). These interpersonal schemata can be altered by disconfirming experiences in a positive therapeutic alliance, and particularly by repairing alliance ruptures. This theory led to the addition of rupture resolution procedures (Safran & Christopher, 2000) into a CBT protocol for generalised anxiety disorder (Borkovec, Newman, & Castonguay, 2004; Newman et al., 2011), effectively increasing the dose of the active component of the alliance (manipulation) responsible for changing the interpersonal-schemata (putative mechanism). These procedures could also benefit BN treatments, and are one illustration of how understanding the mechanisms by which treatment manipulations lead to client change may lead to treatment refinements.

Another possibility for adaptation is to integrate CBT-E for BN with additional treatment manipulations that (i) have shown good outcomes in targeting the proposed treatment

mechanisms of CBT-E; and (ii) have research evidence demonstrating the link between application of the treatment manipulation and change in the treatment mechanism. The proposal to integrate CBT-E with techniques from other therapeutic traditions is not novel. For example, the integration of motivational interviewing with CBT-E has been investigated, guided by the hypothesis that this integration would increase patient readiness to change and improve treatment outcomes (Geller & Dunn, 2011). In recent years there has been a push towards treatment integration, which recognises that each of the therapeutic orientations have strengths that could be combined in complementary ways to enhance treatment outcome (Norcross & Goldfried, 2005). Several authors have recommended that such an expansion of the CBT tradition would best be achieved through assimilative integration (Alford & Beck, 1998; Castonguay, Newman, Borkovec, Holtforth, & Maramba, 2005; Messer, 2001; Safran, 1998; Safran, Eubanks-Carter, & Muran, 2010), maintaining the central theoretical base of CBT and expanding it to incorporate other techniques and theoretical perspectives.

One promising intervention that has evidence linking the treatment manipulation to changes in treatment mechanisms relevant to CBT-E are Gestalt chair dialogues (Elliott, Goldman, Watson, & Greenberg, 2004; Greenberg, Rice, & Elliott, 1993; Perls, Hefferline, & Goodman, 1951). Broadly, experiential treatments that include chair dialogues have been shown to be a promising treatment for binge-eating related disorders (Compare, Calugi, Marchesini, Molinari, & Dalle Grave, 2013; Dolhanty & Greenberg, 2007; Tweed, 2013; Wnuk, 2009). Importantly there is also evidence that chair dialogues act through a specific treatment mechanism, emotion processing, and that this mechanism is associated with downstream change in several of the maintaining mechanisms in the CBT-E model. For example, empty-chair dialogues work through a specific emotional processing sequence (Greenberg & Foerster, 1996),

and change in this treatment mechanism has been specifically linked to change in interpersonal problems (Greenberg & Malcolm, 2002; Paivio, Hall, Holowaty, Jellis, & Tran, 2001). This is an example of a treatment technique that has an underlying research base regarding the client change process, and therefore has the potential to be usefully incorporated into CBT-E and the treatment of BN.

Given the knowledge gaps that have been identified in this review, we have argued for the importance of research that builds an understanding of the client change process during CBT-E. Further developments in CBT-E for BN that are able to both enhance the effective elements of the current treatment, and integrate other effective elements have the potential to enhance the therapeutic efficacy of CBT for BN. These developments are dependent on a close examination of mechanisms of change.

Figure title

*Figure 1*. A representation of the maintaining mechanisms outlined in the enhanced cognitive-behavioural model of eating disorders (Fairburn, Cooper, & Shafran, 2003)

## References


Agras, W., Walsh, B. T., Fairburn, C. G., Wilson, G. T., & Kraemer, H. C. (2000). A multicenter comparison of cognitive-behavioral therapy and interpersonal psychotherapy for bulimia nervosa. *Archives of General Psychiatry, 57*, 459–466. doi:10.1001/archpsyc.57.5.459

Alford, B. A., & Beck, A. T. (1998). *The integrative power of cognitive therapy*. New York, NY: Guilford Press.

Borkovec, T. D., Newman, M. G., & Castonguay, L. G. (2004). Cognitive-behavioral therapy for generalized anxiety disorder with integrations from interpersonal and experiential therapies. *Focus, 2*, 392–401.

Bulik, C. M., Sullivan, P. F., Carter, F. A., McIntosh, V. V., & Joyce, P. R. (1998). The role of exposure with response prevention in the cognitive-behavioural therapy for bulimia nervosa. *Psychological Medicine, 28*, 611–623.

Byrne, S. M., Fursland, A., Allen, K. L., & Watson, H. (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

Carter, O., Pannekoek, L., Fursland, A., Allen, K. L., Lampard, A. M., & Byrne, S. M. (2012). Increased wait-list time predicts dropout from outpatient enhanced cognitive behaviour therapy (CBT-E) for eating disorders. *Behaviour Research and Therapy, 50*, 487–492. doi:10.1016/j.brat.2012.03.003

Castonguay, L. G., Newman, M. G., Borkovec, T. D., Holtforth, M. G., & Maramba, G. G. (2005). Cognitive-behavioral assimilative integration. In J. C. Norcross & M. R. Goldfried (Eds.), *Handbook of Psychotherapy Integration* (2nd ed., Vol. 2, pp. 241–260). New York: Oxford University Press.

Compare, A., Calugi, S., Marchesini, G., Molinari, E., & Dalle Grave, R. (2013). Emotion-focused therapy and dietary counseling for obese patients with binge eating disorder: A propensity score-adjusted study. *Psychotherapy and Psychosomatics*, *82*, 193–194. doi:10.1159/000343209

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. doi:10.1016/0005-7967(95)00033-T

Dolhanty, J., & Greenberg, L. S. (2007). Emotion-focused therapy in the treatment of eating disorders. *European Psychotherapy, 7*, 97–116.

Elliott, R., Goldman, R. N., Watson, J. C., & Greenberg, L. S. (2004). *Learning emotion-focused therapy: The process-experiential approach to change*. American Psychological Association.

Fairburn, C. G., Cooper, Z., & Cooper, P. J. (1986). The clinical features and maintenance of bulimia nervosa. In K. D. Brownell & J. P. Foreyt (Eds.), *Handbook of eating disorders: Physiology, psychology and treatment of obesity, anorexia and bulimia* (pp. 389–404). New York, NY: Basic Books.

Fairburn, C. G., Cooper, Z., Doll, H. A., O'Connor, M. E., Bohn, K., Hawker, D., … Palmer, R. (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. doi: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. doi:10.1016/S0005-7967(02)00088-8

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. doi:10.1001/archpsyc.1991.01810290075014

Fairburn, C. G., Jones, R., Peveler, R. C., Hope, R. A., & O'Connor, M. (1993). 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., Marcus, M. D., & Wilson, G. T. (1993). 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: Guilford Press.

Fairburn, C. G., Stice, E., Cooper, Z., Doll, H. A., Norman, P. A., & O'Connor, M. E. (2003). Understanding persistence in bulimia nervosa: A 5-year naturalistic study. *Journal of Consulting and Clinical Psychology, 71*, 103–109. doi:10.1037/0022-006X.71.1.103

Federici, A., & Kaplan, A. S. (2008). The patient's account of relapse and recovery in anorexia nervosa: a qualitative study. *European Eating Disorders Review, 16*, 1–10. doi:10.1002/erv.813

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.

Geller, J., & Dunn, E. C. (2011). Integrating motivational interviewing and cognitive behavioral therapy in the treatment of eating disorders: tailoring interventions to patient readiness for

change. *Cognitive and Behavioral Practice*, *18*, 5–15. doi:10.1016/j.cbpra.2009.05.005

Ghaderi, A. (2006). Does individualization matter? A randomized trial of standardized (focused) versus individualized (broad) cognitive behavior therapy for bulimia nervosa. *Behaviour Research and Therapy*, *44*, 273–88. doi:10.1016/j.brat.2005.02.004

Gratz, K. (2004). Multidimensional Assessment of Emotion Regulation and Dysregulation: Development, Factor Structure, and Initial Validation of the Difficulties in Emotion Regulation Scale. *Journal of Psychopathology and Behavioral Assessment*, *26*, 41–54.

Greenberg, L. S., & Foerster, F. S. (1996). Task analysis exemplified: the process of resolving unfinished business. *Journal of Consulting and Clinical Psychology*, *64*, 439–446. doi:10.1037/0022-006X.64.3.439

Greenberg, L. S., & Malcolm, W. (2002). Resolving unfinished business: Relating process to outcome. *Journal of Consulting and Clinical Psychology*, *70*, 406.

Greenberg, L. S., Rice, L. N., & Elliott, R. (1993). *Facilitating emotional change: The moment-by-moment process*. New York, NY, USA: Guilford Press.

Hollon, S. D., & Kriss, M. R. (1984). Cognitive factors in clinical research and practice. *Clinical Psychology Review*, *4*, 35–76. doi:10.1016/0272-7358(84)90036-9

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

Keel, P. K., Dorer, D. J., Franko, D. L., Jackson, S. C., & Herzog, D. B. (2005). Postremission predictors of relapse in women with eating disorders. *American Journal of Psychiatry*, *162*, 2263–2268. doi:10.1176/appi.ajp.162.12.2263

Lampard, A. M., 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. doi:10.1016/j.brat.2011.06.002

Lampard, A. M., Tasca, G. A., Balfour, L., & Bissada, H. (2013). An evaluation of the transdiagnostic cognitive-behavioural model of eating disorders. *European Eating Disorders Review*, *21*, 99–107. doi:10.1002/erv.2214

Leitenberg, H., Rosen, J. C., Gross, J., Nudelman, S., & Vara, L. S. (1988). Exposure plus response-prevention treatment of bulimia nervosa. *Journal of Consulting and Clinical Psychology*, *56*, 535–541. doi:http://dx.doi.org.ezproxy.library.yorku.ca/10.1037/0022-006X.56.4.535

Loeb, K., Lock, J., Greif, R., & le Grange, D. (2012). Transdiagnostic theory and application of family-based treatment for youth with eating disorders. *Cognitive and Behavioral Practice*, *19*, 17 ‑ 30. doi:http://dx.doi.org/10.1016/j.cbpra.2010.04.005

Marlatt, G. A., & Gordon, L. R. (1985). *Relapse prevention: Maintenance strategies in addictive behavior change.* New York: Guilford.

Messer, S. B. (2001). Introduction to the special issue on assimilative integration. *Journal of Psychotherapy Integration*, *11*, 1–4. doi:10.1023/A:1026619423048

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

National Institute of Clinical Excellence. (2004). *Eating disorders: Core interventions in the treatment and management of anorexia nervosa, bulimia nervosa and related eating disorders* (No. 9).

Newman, M. G., Castonguay, L. G., Borkovec, T. D., Fisher, A. J., Boswell, J. F., Szkodny, L. E., & Nordberg, S. S. (2011). A Randomized Controlled Trial of Cognitive-Behavioral

Therapy for Generalized Anxiety Disorder With Integrated Techniques From Emotion-Focused and Interpersonal Therapies. *Journal of Consulting and Clinical Psychology, 79*, 171–181. doi:doi: 10.1037/a0022489

Nock, M. K. (2007). Conceptual and design essentials for evaluating mechanisms of change. *Alcoholism: Clinical and Experimental Research, 31*, 4s–12s. doi:10.1111/j.1530-0277.2007.00488.x

Norcross, J. C., & Goldfried, M. R. (2005). *Handbook of psychotherapy integration* (2nd ed.). Cary, NC: Oxford University Press.

Olmsted, M. P., Kaplan, A. S., & Rockert, W. (1994). Rate and prediction of relapse in bulimia nervosa. *The American Journal of Psychiatry, 151*, 738–43.

Paivio, S. C., Hall, I. E., Holowaty, K. A. M., Jellis, J. B., & Tran, N. (2001). Imaginal confrontation for resolving child abuse issues. *Psychotherapy Research*, *11*, 433–453. doi:10.1093/ptr/11.4.433

Perls, F. S., Hefferline, R. F., & Goodman, P. (1951). *Gestalt therapy: excitement and growth in the human personality*. New York, NY: Julian Press.

Polivy, J., & Herman, C. P. (1985). Dieting and binging: A causal analysis. *American Psychologist*, *40*, 193–201.

Poulsen, S., Lunn, S., Daniel, S. I. F., 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. doi:10.1176/appi.ajp.2013.12121511

Safer, D. L., Lively, T. J., Telch, C. F., & Agras, W. S. (2002). Predictors of relapse following successful dialectical behavior therapy for binge eating disorder. *International Journal of*

*Eating Disorders*, *32*, 155–163. doi:10.1002/eat.10080

Safran, J. D. (1998). *Widening the scope of cognitive therapy: The therapeutic relationship, emotion, and the process of change*. Northvale, NJ: Jason Aronson.

Safran, J. D., & Christopher, J. (2000). *Negotiating the therapeutic alliance: A relational treatment guide*. New York, NY: Guilford Press.

Safran, J. D., Eubanks-Carter, C., & Muran, J. C. (2010). Emotion-focused/interpersonal cognitive therapy. In N. Kazantzis, M. A. Reinecke, & A. Freeman (Eds.), *Cognitive and behavioral theories in clinical practice* (pp. 332–362).

Safran, J. D., & Segal, Z. V. (1996). *Interpersonal process in cognitive therapy*. Northvale, NJ: Jason Aronson.

Schmitt, D. P., & Allik, J. (2005). Simultaneous administration of the Rosenberg Self-Esteem Scale in 53 nations: exploring the universal and culture-specific features of global self-esteem. *Journal of Personality and Social Psychology*, *89*, 623–642. doi:http://dx.doi.org.ezproxy.library.yorku.ca/10.1037/0022-3514.89.4.623

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

Steiger, H., Gauvin, L., Jabalpurwala, S., Séguin, J. R., & Stotland, S. (1999). Hypersensitivity to social interactions in bulimic syndromes: Relationship to binge eating. *Journal of Consulting and Clinical Psychology*, *67*, 765–775. doi:http://dx.doi.org.ezproxy.library.yorku.ca/10.1037/0022-006X.67.5.765

Tasca, G. A., & Lampard, A. M. (2012). Reciprocal influence of alliance to the group and outcome in day treatment for eating disorders. *Journal of Counseling Psychology, 59,*

507 – 517. doi:10.1037/a0029947

Tweed, S. (2013). *Group-based emotion focused therapy (EFT) for women with binge spectrum eating disorders in an outpatient setting: A preliminary comparison*.

Wade, T. D., Bergin, J. L., Tiggemann, M., Bulik, C. M., & Fairburn, C. G. (2006). Prevalence and long-term course of lifetime eating disorders in an adult Australian twin cohort. *Australian and New Zealand Journal of Psychiatry, 40,* 121–128. doi:10.1111/j.1440-1614.2006.01758.x

Welch, E., & Ghaderi, A. (2013). Eating disorders and self-esteem. In A. K. Andershed (Ed.), *Girls at Risk* (pp. 35–56). New York, NY: Springer. Retrieved from http://link.springer.com.ezproxy.library.yorku.ca/chapter/10.1007/978-1-4614-4130-4_3

Wilfley, D. E. (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.

Wilfley, D. E., Welch, R., & Stein, R. I. (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. doi:10.1001/archpsyc.59.8.713

Wilson, G. T., Eldredge, K. L., Smith, D., & Niles, B. (1991). Cognitive-behavioral treatment with and without response prevention for Bulimia. *Behaviour Research and Therapy, 29,* 575–583. doi:10.1016/0005-7967(91)90007-P

Wilson, G. T., Fairburn, C. G., 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,* 267–274. doi:http://dx.doi.org/10.1037/0022-

006X.70.2.267

Wnuk, S. (2009). *Treatment development and evaluation of emotion-focused Group therapy for women with symptoms of bulimia nervosa*. York University, Canada.

Wonderlich, S. A., Peterson, C. B., Crosby, R. D., Smith, T. L., Klein, M. H., 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. doi:10.1017/S0033291713001098

Woodward, L. E., Murrell, S. A., & Bettler, R. F. (2005). Stability, reliability, and norms for the inventory of interpersonal problems. *Psychotherapy Research, 15*, 272–286. doi:10.1080/10503300512331334977