



PERGAMON

Behaviour Research and Therapy 41 (2003) 509–528

**BEHAVIOUR
RESEARCH AND
THERAPY**

www.elsevier.com/locate/brat

Cognitive behaviour therapy for eating disorders: a “transdiagnostic” theory and treatment[☆]

Christopher G. Fairburn^{*}, Zafra Cooper, Roz Shafran

Oxford University Department of Psychiatry, Warneford Hospital, Oxford OX3 7JX, UK

Received 28 March 2002; received in revised form 27 May 2002; accepted 27 May 2002

Abstract

This paper is concerned with the psychopathological processes that account for the persistence of severe eating disorders. Two separate but interrelated lines of argument are developed. One is that the leading evidence-based theory of the maintenance of eating disorders, the cognitive behavioural theory of bulimia nervosa, should be extended in its focus to embrace four additional maintaining mechanisms. Specifically, we propose that in certain patients one or more of four additional maintaining processes interact with the core eating disorder maintaining mechanisms and that when this occurs it is an obstacle to change. The additional maintaining processes concern the influence of clinical perfectionism, core low self-esteem, mood intolerance and interpersonal difficulties. The second line of argument is that in the case of eating disorders shared, but distinctive, clinical features tend to be maintained by similar psychopathological processes. Accordingly, we suggest that common mechanisms are involved in the persistence of bulimia nervosa, anorexia nervosa and the atypical eating disorders. Together, these two lines of argument lead us to propose a new transdiagnostic theory of the maintenance of the full range of eating disorders, a theory which embraces a broader range of maintaining mechanisms than the current theory concerning bulimia nervosa. In the final sections of the paper we describe a transdiagnostic treatment derived from the new theory, and we consider in principle the broader relevance of transdiagnostic theories of maintenance.

© 2003 Elsevier Science Ltd. All rights reserved.

Keywords: Eating disorders; Bulimia nervosa; Anorexia nervosa; Diagnosis; Theory; Cognitive behaviour therapy

^{*} Corresponding author. Tel.: +44-1865-226479; fax: +44-1865-226244.

E-mail address: credo@medicine.ox.ac.uk (C.G. Fairburn).

[☆] This paper is based upon a keynote address given by CGF at the Annual Meeting of the British Association for Behavioural and Cognitive Psychotherapies held in Glasgow in July 2001. The three authors contributed equally to this paper

1. Introduction

This paper is concerned with the psychopathological processes that account for the persistence of the full range of severe eating disorders. For clarity, we take as our starting point bulimia nervosa and its cognitive behavioural theory and treatment (CBT-BN) since both have been well-specified and extensively studied. The treatment was first described by Fairburn in 1981 (Fairburn, 1981). Several years later further procedural details were described (Fairburn, 1985) together with a more complete exposition of the theory upon which it was based (Fairburn, Cooper, & Cooper, 1986). A full treatment manual was published in 1993 (Fairburn, Marcus, & Wilson, 1993b) and this has been widely used in subsequent treatment trials (e.g., Agras et al., 2000a; Agras, Walsh, Fairburn, Wilson, & Kraemer, 2000b; Fairburn, Jones, Peveler, Hope, & O'Connor, 1993a). As a result of further experience across different treatment centres, a supplement to the manual was published in 1997 in which aspects of its implementation were discussed (Wilson, Fairburn, & Agras, 1997). The theory was elaborated in the same year (Fairburn, 1997a).

2. Cognitive behaviour therapy for bulimia nervosa

2.1. *The cognitive behavioural theory*

The theory that underpins CBT-BN is primarily concerned with the processes which maintain bulimia nervosa, although it is also of relevance to the development of the disorder. According to the theory, central to the maintenance of bulimia nervosa is a dysfunctional system for evaluating self-worth. Whereas most people evaluate themselves on the basis of their perceived performance in a variety of domains of life (e.g., the quality of their relationships, work, parenting, sporting ability, etc), people with eating disorders judge themselves largely, or even exclusively, in terms of their eating habits, shape or weight (and often all three) and their ability to control them. As a result, their lives become focused on their eating, shape and weight, with dietary control, thinness and weight loss being actively pursued whilst overeating, 'fatness' and weight gain are assiduously avoided. These distinctive, and highly characteristic, behavioural and attitudinal features are prominent and well-recognised, as is the dysfunctional system for evaluating self-worth (e.g., American Psychiatric Association, 2000).

According to the cognitive behavioural theory of the maintenance of bulimia nervosa, this over-evaluation of eating, shape and weight and their control is of primary importance in maintaining the disorder. Most of the other clinical features can be understood as stemming directly from this 'core psychopathology', including the extreme weight-control behaviour (viz., the persistent attempts to restrict food intake (dietary restraint), the self-induced vomiting, misuse of laxatives and diuretics, and the over-exercising), the various forms of body checking and avoidance, and the preoccupation with thoughts about eating, shape and weight. Fig. 1 provides a schematic representation of the main processes involved.

The only feature that is not obviously a direct expression of the core psychopathology is these patients' 'binge eating' (episodes of uncontrolled overeating). The cognitive behavioural theory proposes that binge eating is largely a product of the particular way that these patients attempt to restrict their eating (i.e., their form of dietary restraint), whether or not there is an actual energy

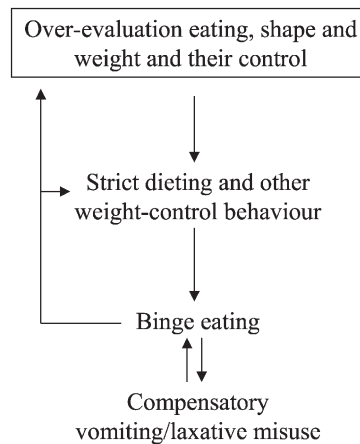


Fig. 1. A schematic representation of the cognitive behavioural theory of the maintenance of bulimia nervosa (modified from Fairburn, Marcus, & Wilson, 1993b).

deficit. Rather than adopting general guidelines about how they should eat, they try to adhere to multiple extreme, and highly specific, dietary rules. Accompanying these dietary rules is a tendency to react negatively to the (almost inevitable) breaking of them with even minor dietary slips being interpreted as evidence of their lack of self-control, the result being that they respond by temporarily abandoning their efforts to restrict their eating. Patient reports indicate that this is a major trigger of binge eating. The consequence is a highly distinctive pattern of eating in which sustained dietary restraint is repeatedly punctuated by episodes of binge eating. This binge eating in turn maintains the core psychopathology by magnifying patients' concerns about their ability to control their eating, shape and weight. This encourages yet greater dietary restraint, thereby further increasing the risk of binge eating.

In the original cognitive behavioural formulation (Fairburn, Cooper & Cooper, 1986), it was noted that these patients' dietary slips and binges do not happen at random; rather, they are particularly likely to occur in response to acute changes in mood (typically adverse mood states) since these appear to interfere with the ability to maintain dietary restraint. However, since binge eating can have the effect of temporarily neutralising such states, and distracting patients from life difficulties, the result is that it is reinforced (as discussed later).

A further process maintains binge eating amongst those patients who practise compensatory 'purging' (i.e., those who induce vomiting or take laxatives in response to specific episodes of binge eating). These patients' faith in the ability of such purging to minimise weight gain results in a major deterrent against binge eating being undermined¹.

One other maintaining process has been highlighted in more recent accounts of the theory (Fairburn, Marcus & Wilson, 1993b; Fairburn, 1997a). These patients tend to be extremely self-critical. They set themselves demanding standards in terms of their eating, shape and weight, and their control, and when they cannot meet them, they see themselves as being at fault rather than

¹ This view is mistaken since vomiting only retrieves part of what has been eaten, and laxatives have little or no effect on energy absorption (see Fairburn, 1995, pp. 48–54).

their standards as being too harsh. The result is secondary negative self-evaluation. This too maintains the eating disorder since it leads patients to strive even harder to achieve ‘success’ in the area of life that is most important to them; that is, controlling their eating, shape and weight. In this way a further vicious circle serves to maintain the eating disorder.

2.1.1. Evidence supporting the theory

This cognitive behavioural theory is supported by variety of lines of evidence:

1. Indirect support comes from the large body of research indicating that CBT-BN, a treatment that is targeted at these specific maintaining mechanisms, is effective in many cases and is superior to comparison treatments that control for the non-specific effects of psychological treatment and to pharmacotherapy (see below). This is not to say that CBT-BN could not be more effective.
2. Also consistent with the theory is the finding from one study that, among patients who made a full response to treatment, those with the highest residual level of over-evaluation of shape and weight were most prone to relapse (Fairburn, Peveler, Jones, Hope, & Doll, 1993c), although another study failed to replicate this finding (Cooper & Steere, 1995).
3. Additional support comes from a study of mediators of response to CBT-BN which found that a decrease in dietary restraint mediated the treatment’s effect on binge eating (Wilson, Fairburn, Agras, Walsh, & Kraemer, 2002).
4. Further support comes from two treatment trials in which largely behavioural versions of the treatment (which did not address the over-evaluation of eating, shape and weight) were found to be associated with an increased risk of relapse compared to the full treatment (Cooper & Steere, 1995; Fairburn, Jones, Peveler, Hope & O’Connor, 1993a).
5. Many of the main predictions of the theory were supported by the findings from a cross-sectional structural equation modeling study which examined the relevant psychopathological relationships, although its findings concerning binge eating and purging are difficult to interpret as a result of the specific assessment measures used (Byrne & McLean, 2002).
6. Support also comes from the findings from a prospective study of the natural course of bulimia nervosa in which it was found that the baseline level of over-evaluation of shape and weight predicted persistence of binge eating (over 15 months), and that the degree of dietary restraint partially mediated this relationship (Fairburn et al., in press).

2.2. The cognitive behavioural treatment

The cognitive behavioural theory of the maintenance of bulimia nervosa has clear implications for treatment. Specifically, it suggests that the focus of treatment should not solely be on these patients’ binge eating, despite the fact that binge eating is often their primary (and sometimes only) complaint. Rather, it suggests that to achieve a full and lasting response, these patients’ dietary restraint also needs to be addressed, as does their response to adverse mood states and their over-evaluation of eating, shape and weight and their control.

The cognitive behavioural treatment for bulimia nervosa stems from this theory. It is designed to address each of the maintaining processes outlined above. The treatment is outpatient-based and, as evaluated in treatment trials, involves 15–20 sessions over approximately five months. A

range of cognitive behavioural procedures are used with its cornerstone being a specific sequence of cognitive behavioural tasks and “experiments” set within the context of a personalised version of the cognitive behavioural theory of maintenance (Fairburn, Marcus & Wilson, 1993b).

2.2.1. Evidence supporting the treatment

Given that bulimia nervosa was first described relatively recently (American Psychiatric Association, 1980; Russell, 1979), there has been a remarkable amount of research on its treatment. Over 50 randomised controlled trials have been completed and there have been over 20 studies of CBT-BN. Although almost all of these studies have been “efficacy” rather than “effectiveness” trials, there are good reasons to think that their findings are relevant to everyday patient care (see Wilson, 1998a; Wilson & Fairburn, 2002), not least because the patients have closely resembled those seen in routine clinical practice (Wilson, 1998b).

The main findings of relevance may be summarised as follows (see Wilson and Fairburn, 2002) for a more detailed review of the findings)²:

- CBT-BN has a substantial effect on the frequency of binge eating and purging, and the full range of the psychopathology of bulimia nervosa. Among treatment completers (typically 80 to 85%), between 40–50% cease binge eating and purging altogether. These patients generally improve in all respects, and it seems that most remain well in the long-term (e.g., Fairburn et al., 1995). The remaining patients range in outcome from substantially improved to not improved at all.
- CBT-BN is more effective than both delayed treatment and pharmacotherapy.
- CBT-BN has been compared with a wide range of psychological treatments including supportive psychotherapy, focal psychotherapy, supportive-expressive psychotherapy, interpersonal psychotherapy (IPT), hypnobeavioural treatment, stress management, nutritional counselling, behavioral versions of CBT-BN, and various forms of exposure with response prevention (see Wilson & Fairburn, 2002). It has proved as effective as, or more effective than, all these treatments. The leading alternative treatment is IPT which, in statistical terms, is comparable in its eventual effects but much slower to act (Agras, Walsh, Fairburn, Wilson & Kraemer, 2000b; Fairburn, Jones, Peveler, Hope & O'Connor, 1993a).
- Consistent predictors of response to CBT-BN have been difficult to find but there is emerging evidence that the most powerful and consistent predictors are the frequency of binge eating and purging at the start of treatment (the higher the frequency, the worse the prognosis) and, most especially, the extent of their reduction over the initial weeks of treatment (Agras et al., 2000a)³. As Wilson (1999a) has highlighted, the predictive utility of early response to CBT is a phenomenon not confined to bulimia nervosa but has been reported in other disorders including depression, alcohol abuse and obesity.

Therefore, two clear findings emerge from this large body of research. *The first is that CBT-*

² The findings of the Cochrane systematic review of studies of the treatment of bulimia nervosa (Hay & Bacaltchuk, 2000) are impossible to interpret because the review conflates studies of binge eating disorder with those of bulimia nervosa.

³ This finding has been replicated using data from the Stanford-Oxford-Columbia CBT-IPT trial. (See Agras and colleagues (2000b) for details of this study.).

BN is an effective treatment for bulimia nervosa: indeed, given the quantity, quality and breadth of the research evidence, bulimia nervosa is one of the clearest indications for CBT. The second finding is that CBT-BN is not effective enough: at best, only half the patients make a full and lasting response. This raises the important question “Why aren’t more people getting better?”

2.3. Why aren’t more people getting better?

In principle, four main explanations may be proposed as to why the response to CBT-BN is not greater than it is.

2.3.1. The theory upon which CBT-BN is based is incorrect, and therefore the targeted maintaining mechanisms are the wrong ones

As outlined above, there are strong grounds for thinking that this is not the case. This is not to say that the theory is a complete account of the processes that maintain bulimia nervosa.

2.3.2. CBT-BN has not been implemented optimally in the research trials

This seems unlikely since the larger and most influential trials have been conducted at major research centres with principal investigators who are experts in psychological treatment research. Furthermore, quality control measures were in place. Having said this, adherence findings have yet to be published. It might be argued that the response rates would have been higher had the treatment not had to conform to a research protocol (e.g., being restricted to a fixed number of sessions over a predetermined time period) and a specific treatment manual (Fairburn, Marcus & Wilson, 1993b). The former point is a reasonable one but the latter neglects the clinical flexibility inherent within such manuals and the arguments and evidence supporting manual-based treatment in general (see Wilson, 1996, 1998a).

2.3.3. While the theory underpinning CBT-BN is valid and the focus of treatment is therefore appropriate, the existing treatment procedures are not sufficiently potent

Clinical experience suggests that this is undoubtedly true in some cases. Even when the treatment is well implemented, the targeted psychopathology can prove resistant to change.

There are a number of ways in which the effectiveness of CBT-BN might be improved without shifting its focus from the maintaining mechanisms specified above. First, new generic cognitive behavioural procedures have been developed since CBT-BN was first devised and these could be capitalised upon (for example, see Beck, 1995; Greenberger and Padesky, 1995; Padesky and Greenberger, 1995; Segal, Williams, & Teasdale, 2002). Second, certain aspects of the core psychopathology have received insufficient attention in CBT-BN yet accumulating clinical experience suggests that they may play an important role in maintaining the disorder. For example, it has only recently been appreciated that many patients repeatedly check aspects of their body in highly idiosyncratic ways, and that their means of doing so is likely to intensify their concerns and dissatisfaction (Fairburn, Shafran, & Cooper, 1999; Rosen, 1997). The treatment procedures for addressing body checking therefore need to be enhanced (Rosen, 1996; Wilson, 1999b). Indeed, it is our view that the entire strategy for addressing the over-evaluation of eating, shape and weight, and its various expressions, needs to be reformulated, and that greater emphasis needs to be placed on it. A third way of possibly improving CBT-BN stems from the finding that the

magnitude of initial behavioural response is a powerful predictor of outcome. This suggests that increasing the proportion of patients who do well early on in treatment might increase the overall response rate. For example, benefits might come from reverting to the original CBT-BN strategy of seeing patients twice a week for the initial four weeks (Fairburn, 1985), rather than for just two weeks as in recent research protocols, since in our experience doing so results in greater behaviour change. It is also our view that effort should be made to ensure that the momentum of treatment is not interrupted, particularly in the early stages, since even quite short breaks can result in substantial setbacks.

2.3.4. *The theory is valid but it needs to be extended to embrace additional maintaining mechanisms*

Several authors have suggested that the existing theory is too narrowly focused in that it concentrates primarily on the “specific psychopathology” of eating disorders (i.e., clinical features peculiar to eating disorders) (e.g., Hollon & Beck, 1993; Meyer, Waller, & Waters, 1998). We too think that there is a need for a revised conceptualisation of the processes that maintain bulimia nervosa. Accordingly, we have developed one and it is described below.

3. A new cognitive behavioural theory of the maintenance of bulimia nervosa

The new theory represents an extension of the original theory illustrated in Fig. 1. Specifically, it is proposed that *in certain patients* one or more of four additional maintaining processes interact with the core eating disorder maintaining mechanisms shown in Fig. 1 and that when this occurs it is an obstacle to change. The first of these additional maintaining mechanisms concerns the influence of severe perfectionism (“clinical perfectionism”); the second concerns the impact of unconditional and pervasive low self-esteem (“core low self-esteem”); the third concerns difficulty coping with intense mood states (“mood intolerance”); and the fourth is interpersonal and developmental in character (“interpersonal difficulties”). Thus this new cognitive behavioural theory is broader in its scope than the original more focused one. However, it must be stressed that this theory is not intended to replace the former one—this would make little sense given the weight of evidence supporting it. Rather, the new theory is designed to supplement it. This point is illustrated in Fig. 2 which shows in schematic form some of the main hypothesised maintaining mechanisms. These will now be described.

3.1. *Clinical perfectionism*

Although the notion of perfectionism is widely used, there have been few attempts to define and characterise it. We have recently provided a cognitive behavioural analysis of what we term “clinical perfectionism”—that is, perfectionism of clinical significance (Shafran, Cooper, & Fairburn, 2002). This we define as the over-evaluation of the striving for, and achievement of, personally demanding standards, despite adverse consequences. In other words we suggest that at the heart of the psychopathology of clinical perfectionism is a system for self-evaluation in which self-worth is judged largely on the basis of striving to achieve demanding goals and success at meeting them. It should be evident therefore that we view clinical perfectionism as a form of

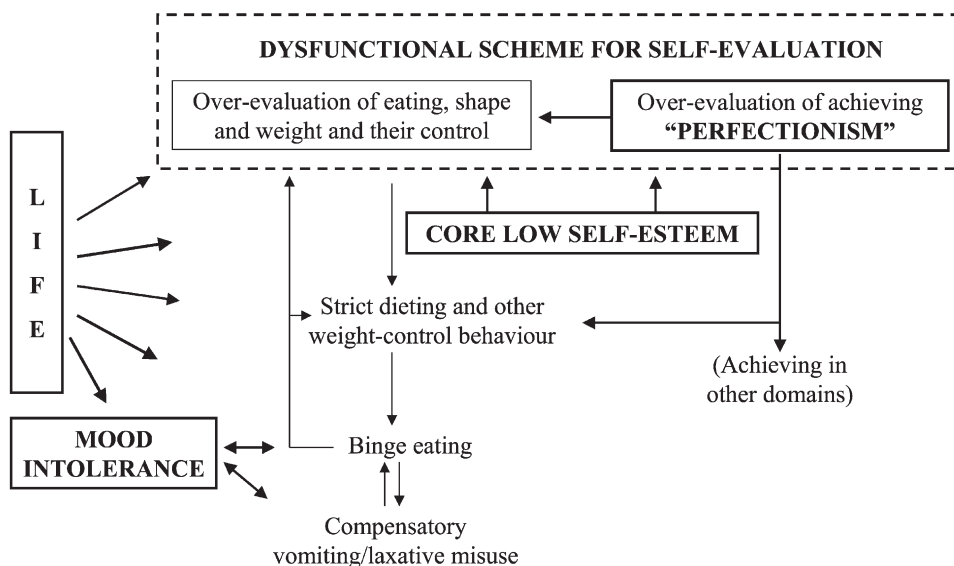


Fig. 2. A schematic representation of the extended cognitive behavioural theory of the maintenance of bulimia nervosa. 'Life' is shorthand for interpersonal life.

psychopathology that is similar in nature to the "core psychopathology" of eating disorders in that both are examples of dysfunctional systems for self-evaluation.

Perfectionism is well-known to co-occur with eating disorders (see Wonderlich, 2002; Shafran et al., 2002). Under these circumstances, there is often an interaction between the two forms of psychopathology with the patient's perfectionist standards being applied to the attempts to control eating, shape and weight, as well as to other aspects of their life (e.g., their performance at work or sport). As in other expressions of clinical perfectionism (Shafran et al., 2002), there is fear of failure (i.e., in these patients, fear of overeating, "fatness", weight gain); frequent and selective attention to performance (in these patients, repeated calorie-counting, frequent shape and weight checking); and self-criticism arising from negatively biased appraisals of their performance. The resulting secondary negative self-evaluation in turn encourages even more determined striving to meet valued goals—including, in this case, striving to meet goals in the domain of controlling eating, shape and weight—thereby serving to maintain the eating disorder. Therefore in such patients it may be predicted that were their clinical perfectionism to be corrected, a potent additional network of maintaining mechanisms would be removed thereby facilitating change.

3.2. Core low self-esteem

Whilst most patients with bulimia nervosa are self-critical as a result of their failure to achieve their goals, a form of negative self-evaluation that generally reverses with successful treatment, there is a subgroup that has a more global negative view of themselves. Thus, rather than simply thinking negatively about themselves as a result of their inability to control their eating, shape and weight, these patients have an unconditional and pervasive negative view of themselves which is seen as part of their permanent identity. Their negative self-judgements are autonomous and

largely independent of performance: in other words, they are less affected by changes in the state of the eating disorder.

Such “core low self-esteem” tends to obstruct change in general. This is through two main mechanisms. First, it creates in patients hopelessness about their capacity to change, thereby undermining their compliance with treatment; and second, it results in them pursuing, with particular determination, achievement in their valued domains (in this case the pursuit of control over eating, shape and weight) thereby making change in these areas all the more difficult. The state is also self-perpetuating since these patients show particularly pronounced negative cognitive processing biases, coupled with over-generalisation, with the result that any perceived “failure” is interpreted as confirmation that they are failures as people thereby reaffirming their overall negative view of themselves.

Given the many barriers to change that arise as a result of core low self-esteem, it is not surprising that clinical experience and some research findings suggest that such patients respond particularly poorly to treatment (Fairburn, Kirk, O'Connor, Anastasiades, & Cooper, 1987, 1993c). On the other hand, were their core low self-esteem to be corrected, it would be predicted that their outcome would improve as a result.

3.3. *Mood intolerance*

It was recognised in the original cognitive behavioural theory that adverse mood states can be a trigger of binge eating, and it was suggested that their primary effect was to disrupt dietary restraint (Fairburn, Cooper & Cooper, 1986). It is now clear that in some patients there is a more complex relationship between emotional states and binge eating (e.g., Meyer, Waller & Waters, 1998; Polivy & Herman, 1993; Steinberg, Tobin, & Johnson, 1990; Stice, 1994; Waller, 2002).

A subgroup of patients with eating disorders have what may be termed “mood intolerance”. We use this term to refer to an inability to cope appropriately with certain emotional states. Usually this intolerance is of adverse mood states, such as anger, anxiety or depression, but in some cases there is intolerance of all intense mood states including positive ones (e.g., excitement). Instead of accepting changes in mood and dealing appropriately with them, these patients engage in what may be termed “dysfunctional mood modulatory behaviour.” This reduces their awareness of the triggering mood state (and the associated cognitions), and also neutralises it, but at a personal cost. The dysfunctional mood modulatory behaviour may take the form of self-injury (e.g., cutting, punching or burning themselves) which has the effect of rapidly dissipating the initial mood state, or it may involve taking psychoactive substances (e.g., alcohol, tranquillizers) to directly modify how they feel. Both classes of behaviour are not uncommon among patients with eating disorders (e.g., Claes, Vandereycken, & Vertommen, 2001; Holder-ness, Brooks-Gunn, & Warren, 1994; Paul, Schroeter, Dahme, & Nutzinger, 2002). In patients with bulimia nervosa, binge eating, self-induced vomiting and intense exercising may also be used as forms of mood modulatory behaviour, binge eating most commonly serving this purpose. Among these patients (i.e., those with an eating disorder and mood intolerance), such forms of behaviour (i.e., binge eating, vomiting, intense exercising) can become habitual means of mood modulation.

It is not clear whether these patients actually experience unusually intense mood states or whether they are especially sensitive to them. Often both appear to be the case. Either way,

cognitive processes commonly contribute to the phenomenon; for example, such patients typically respond to incipient mood change by thinking that they will not be able to cope with the resulting feelings and thoughts, a reaction that can amplify the mood state.

3.4. *Interpersonal difficulties*

The original cognitive behavioural account of the maintenance of bulimia nervosa existed in something of a vacuum in that it paid little attention to patients' circumstances—other than acknowledging that the proximal triggers of binge eating were commonly interpersonal in nature. The need to broaden this focus has been made especially clear by the research on IPT. This indicates that an exclusively interpersonal form of this treatment is about as effective as cognitive behaviour therapy in the longer-term (Agras, Walsh, Fairburn, Wilson & Kraemer, 2000b; Fairburn, Jones, Peveler, Hope & O'Connor, 1993a, 1995) despite the fact that it does not directly address any of the maintaining mechanisms identified by the cognitive behavioural theory (Fairburn, 1997b).

There can be no doubt that interpersonal processes contribute in a variety of ways to the maintenance of eating disorders. Four examples may be used to illustrate this point. First, in younger patients, family tensions often intensify resistance to eating. It can be argued that this reflects a short-term intensification of their need for a sense of “control”, a need which is displaced onto dietary self-control (Fairburn, Shafran & Cooper, 1999). Second, it is obvious that certain interpersonal environments magnify concerns about controlling eating, shape and weight. These include families in which there are other members with an eating disorder, and occupations in which there is pressure to be slim. Third, as already noted, adverse interpersonal events commonly precipitate episodes of binge eating, and there is evidence that patients with bulimia nervosa may be especially sensitive to social interactions (Steiger, Gauvin, Jabalpurwala, Seguin, & Stotland, 1999). Fourth, long-term interpersonal difficulties undermine self-esteem which, as noted earlier, is prone to result in patients striving even harder to achieve valued goals, such as success at controlling eating, shape and weight. It is also relevant that there is evidence that disturbed interpersonal functioning predicts a poor response to treatment (Agras et al., 2000a; Steiger, Leung, & Thibaudeau, 1993).

Having said this, it is not known how IPT achieves its beneficial effects. It is likely to be through several mechanisms (Fairburn, 1997b). First, it is clear from clinical experience that it succeeds in helping many patients overcome ongoing interpersonal difficulties, ones which may have been longstanding. For example, one of its foci is on interpersonal “role transitions” (see Klerman, Weissman, Rounsaville, & Chevron, 1984). This is especially pertinent to those patients who have missed out on the interpersonal challenges of late adolescence and early adulthood as a result of being immersed in their eating disorder. Second, it is our observation that IPT can also open up new interpersonal opportunities (“fresh start events”; Brown, Adler, & Bifulco, 1988) which may themselves have positive effects (Fairburn, 1997b). When this happens interpersonal aspects of life start to occupy a greater place in these patients' system for self-evaluation which, in turn, reduces the importance that they attach to eating, shape and weight and their control. Third, IPT can give patients a sense that they are capable of influencing their interpersonal lives (in some cases for the first time) which may also lessen their need to control their eating, shape and weight.

In summary, there are good reasons to think that interpersonal difficulties can perpetuate eating disorders, and there is evidence that their resolution facilitates change.

4. A transdiagnostic perspective

We now turn to our second line of argument, that concerning transdiagnostic maintaining mechanisms, starting with anorexia nervosa.

4.1. *Anorexia nervosa*

Anorexia nervosa is the other main eating disorder recognised by the leading classificatory systems in psychiatry. Anorexia nervosa and bulimia nervosa have much in common. They share essentially the same core psychopathology with both groups of patients over-evaluating eating, shape and weight and their control⁴, and this psychopathology is expressed in similar attitudes and behaviour. Thus patients with anorexia nervosa restrict their food intake in the same rigid and extreme way as patients with bulimia nervosa, and they too may vomit, misuse laxatives or diuretics, and over-exercise. In those anorectic patients who over-evaluate controlling their shape and weight as well as their eating (see footnote 4), there are the same forms of body checking as in bulimia nervosa, and both groups of patients complain of preoccupation with thoughts about eating, shape and weight. Nor does binge eating distinguish the two disorders, for in a subgroup of patients with anorexia nervosa binge eating (with or without compensatory purging) is also present (Casper, Eckert, Halmi, Goldberg, & Davis, 1980; Garfinkel, Moldofsky, & Garner, 1980). Thus anorexia nervosa and bulimia nervosa share the same distinctive and characteristic clinical features. The major difference between the two disorders lies in the relative balance of the under-eating and over-eating, and its effect on body weight. In bulimia nervosa the two forms of behaviour tend to cancel each other out with the result that body weight is usually unremarkable, whereas in anorexia nervosa under-eating predominates—indeed, there may be no binge eating at all—with the result that body weight is extremely low, and symptoms of starvation are prominent (see Garner, 1997).

If the commonalities between the two disorders are obvious when they are viewed cross-sectionally, they become even more evident when a longitudinal perspective is taken. In patients who do not recover from anorexia nervosa, cross-over to bulimia nervosa is a frequent occurrence (e.g., Sullivan, Bulik, Fear, & Pickering, 1998), the result being that about a quarter of patients with bulimia nervosa have had anorexia nervosa in the past (e.g., Agras, Walsh, Fairburn, Wilson & Kraemer, 2000b). This movement across from anorexia nervosa to bulimia nervosa reflects a shift in the balance of under-eating and over-eating with the former ceasing to be dominant in its influence on body weight. Generally other aspects of the psychopathology remain much the same but with the starvation symptoms fading as weight increases and with the fears of fatness being intensified by the episodes of binge eating.

⁴ If there is a difference in their core psychopathology, it is that in some patients with anorexia nervosa the primary focus is on controlling eating per se, rather than on shape and weight and their control. In our experience, this tends to be especially true of younger cases of short duration. (See Fairburn et al., 1999 for discussion of the issue of control in anorexia nervosa.)

4.2. Atypical eating disorders or EDNOS

Although the concept of an “eating disorder” tends to be equated with anorexia nervosa and bulimia nervosa, both the leading classificatory systems in psychiatry recognise a third residual category of eating disorder. These “atypical eating disorders” (Fairburn & Walsh, 2002)—the DSM-IV term is “eating disorder not otherwise specified” or EDNOS (American Psychiatric Association, 1994)—are “eating disorders” of clinical severity that do not meet the specific criteria for anorexia nervosa or bulimia nervosa. They are at least as prevalent as anorexia nervosa and bulimia nervosa, if not more so (e.g., Millar, 1998; Ricca et al., 2001), and they too have much in common with bulimia nervosa (and, indeed, anorexia nervosa). Some resemble the full syndromes of anorexia nervosa or bulimia nervosa but do not quite meet their diagnostic criteria, whereas in others the characteristic psychopathological features are combined in a somewhat different way. In our clinical experience rigid and extreme dietary restraint is prominent in most atypical eating disorders and sometimes it is accompanied by self-induced vomiting, over-exercising or laxative misuse. Binge eating (objective or subjective) is also a common feature, and in the majority of cases there is the over-evaluation of eating, shape and weight and their control that is the hallmark of the two prototypic eating disorders.

As with the comparison of anorexia nervosa and bulimia nervosa, cross-diagnostic commonalities become even more obvious when a longitudinal perspective is taken. For example, an atypical eating disorder is a common outcome of anorexia nervosa (e.g., Sullivan, Bulik, Fear & Pickering, 1998); bulimia nervosa typically starts as anorexia nervosa or an atypical eating disorder; and a particularly common outcome of bulimia nervosa is a chronic atypical eating disorder (e.g., Fairburn et al., 1995).

The significance of this migration of patients across the diagnostic categories of anorexia nervosa, bulimia nervosa and the atypical eating disorders has received surprisingly little attention. It is far from random and its basis is not understood. Age and/or duration of disorder appear to be relevant, the two being difficult to disentangle. The eating disorders of mid-adolescence typically take the form of anorexia nervosa or an anorexia nervosa-like state, whereas a bulimia nervosa-like picture is more typical of those of late adolescence or early adulthood. Thus disorders which persist from adolescence to adulthood commonly change in form from a restricting anorexia nervosa-like picture to one more typical of bulimia nervosa. Indeed, this transition is so common that it has recently been suggested that the restricting form of anorexia nervosa (i.e., one in which there is no regular binge eating or purging) should merely be viewed as a “phase” in the course of the eating disorder (Eddy et al., 2002). The phenomenon has encouraged us to take a transdiagnostic perspective on the maintenance of eating disorders.

4.3. Implications of the transdiagnostic perspective for the maintenance of eating disorders

In summary, anorexia nervosa, bulimia nervosa and the atypical eating disorders share the same distinctive psychopathology, and patients move between these diagnostic states over time. These two characteristics, together with the clinical observation that shared clinical features tend to be maintained by similar psychopathological processes, suggest that *common mechanisms are involved in the persistence of bulimia nervosa, anorexia nervosa and the atypical eating disorders*. This is illustrated in principle in Fig. 3 (left-hand figure) in which the focused cognitive behav-

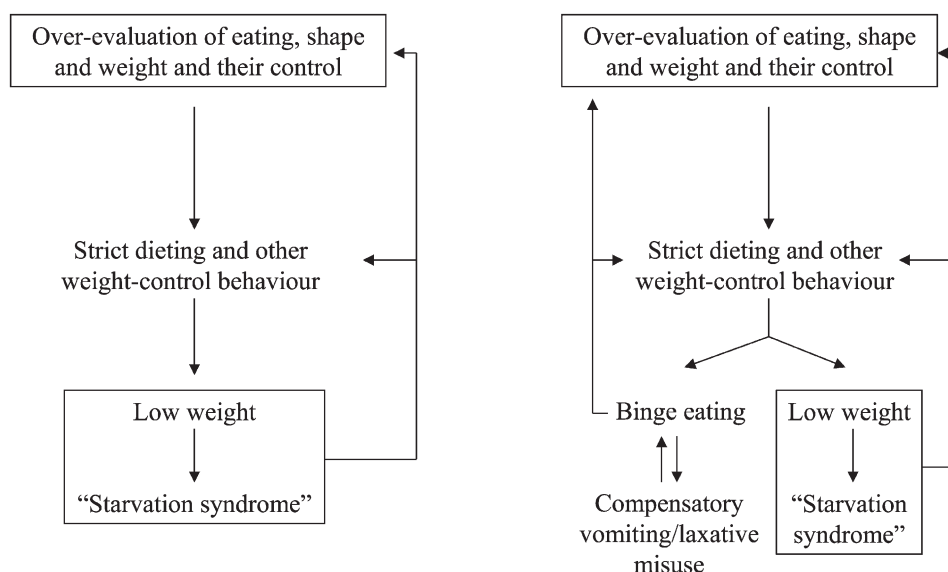


Fig. 3. A schematic representation of the maintenance of anorexia nervosa. The left-hand figure represents the 'restricting type' and the right-hand figure represents the 'binge-eating/purging type'.

ioal formulation of bulimia nervosa has been modified to represent the main processes maintaining anorexia nervosa (restricting type). As can be seen, the binge eating and compensatory purging have been replaced by a box labelled "starvation syndrome" since, as Garner and others (e.g., Fairburn, Shafran & Cooper, 1999; Garner, Rockert, Olmsted, Johnson & Coscina, 1985; Garner, 1997) have argued, various aspects of the starvation syndrome appear to maintain anorexia nervosa. Particularly important in our view is the pronounced social withdrawal seen in starvation since it has the effect of encouraging self-absorption whilst also isolating patients from external influences that might diminish their over-evaluation of eating, shape and weight and their control. In the binge-eating/purging type of anorexia nervosa both the starvation syndrome and the binge eating contribute to the maintenance of the eating disorder (see Fig. 3, right-hand figure), possibly explaining the relatively poor prognosis of such patients (Steinhausen, 1991). In our experience, equivalent processes serve to maintain the various forms of atypical eating disorder.

This transdiagnostic line of argument applies equally to the new broader conceptualisation of bulimia nervosa. Its cross-diagnostic relevance may be best illustrated with reference to anorexia nervosa since this disorder's clinical features are more consistent and better established than those of the atypical eating disorders. First, with respect to clinical perfectionism, the mechanisms that were specified as operating in some cases of bulimia nervosa also operate in certain cases of anorexia nervosa: indeed, clinical perfectionism is more prominent in anorexia nervosa than bulimia nervosa which may partly account for these patients success at restricting their eating. Core low self-esteem also occurs in anorexia nervosa especially in more chronic cases where it appears to be an important maintaining factor. In contrast, mood intolerance (as defined above) is less typical of patients with anorexia nervosa, although it is seen among some of those with the binge eating/purging form of the disorder. Lastly, interpersonal difficulties are frequently present in anorexia nervosa, both with the patient's family and with his or her peers, hence the

widespread use of family therapy (Dare & Eisler, 2002) and the recent interest in IPT (McIntosh, Bulik, McKenzie, Luty & Jordan, 2000). Thus, each of the four additional sets of maintaining processes specified in the broader conceptualisation of bulimia nervosa is also likely to operate among subgroups of patients with anorexia nervosa. And again, in our experience, the same applies in precisely the same way to subgroups of patients with atypical eating disorders⁵.

It is important to note that this broader conceptualisation of the maintenance of eating disorders is consistent with other accounts, particularly cognitive behavioural accounts of anorexia nervosa: for example, Garner, Vitousek/Bemis and Pike have particularly stressed the contribution of low self-esteem (e.g., Garner & Bemis, 1982, 1985; Garner, Vitousek, & Pike, 1997; Pike, Loeb, & Vitousek, 1996). It should also be noted that the emphasis on interpersonal processes is consistent with family therapy (Dare and Eisler, 2002; Lock, le Grange, Agras, & Dare, 2001) and IPT perspectives (Fairburn, 1997b; McIntosh, Bulik, McKenzie, Luty & Jordan, 2000). What is distinctive about the present theory is its transdiagnostic scope, its specificity and its clear implications for treatment.

5. The transdiagnostic treatment

In this paper we have proposed that a network of inter-related maintaining mechanisms accounts for the persistence of anorexia nervosa, bulimia nervosa and the atypical eating disorders. This network is illustrated in schematic form in Fig. 4. More specifically, we have suggested that in the great majority of cases there is a central cognitive disturbance characterised by the over-evaluation eating, shape and weight and their control, and that in subgroups of these patients one or more of four additional mechanisms also serve to maintain the eating disorder. We have not proposed that these additional mechanisms necessarily operate simultaneously, nor have we suggested that they are active in every case. Indeed, their partial independence may account in part for the varied and fluid form of these disorders. Rather, we have suggested that in individual patients they contribute to the maintenance of the eating disorder and that, under such circumstances, unless they are successfully corrected, treatment is not likely to result in full and lasting recovery.

On the basis of this cognitive behavioural theory, we have developed a new transdiagnostic treatment (Fairburn, Cooper, & Shafran, 2002). Its main characteristics are as follows:

1. The treatment has been designed to be suitable for all forms of clinical eating disorder so long as outpatient management is appropriate. In our experience this is true of the great majority of cases.
2. The patient's specific eating disorder diagnosis is not of relevance to the treatment. Rather, its

⁵ In the subtype of EDNOS termed "binge eating disorder", binge eating occurs in the absence of extreme weight-control behaviour (American Psychiatric Association, 2000; Grilo, 2002). Here the primary maintaining process appears to be the mood-modulatory effect of binge eating. Thus a relatively circumscribed maintaining mechanism is operating. This might account for the disorder's tendency to remit in the absence of treatment (Fairburn, Cooper, Doll, Norman, and O'Connor, 2000) and its responsiveness to a variety of interventions (Wilson & Fairburn, 2000).

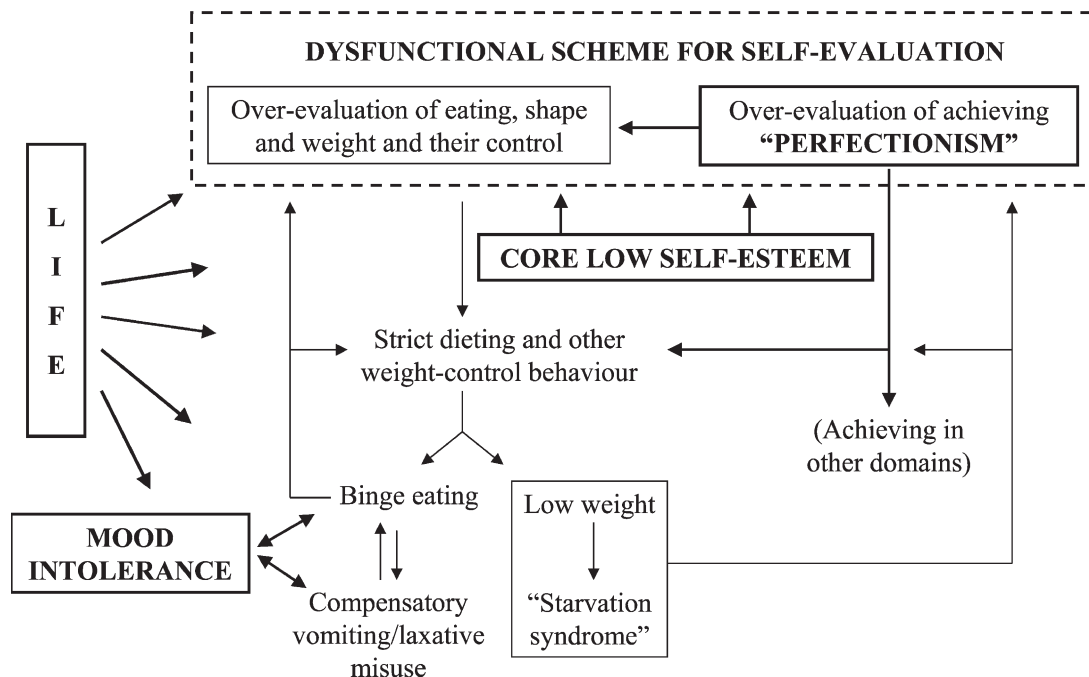


Fig. 4. A schematic representation of the ‘transdiagnostic’ theory of the maintenance of eating disorders. ‘Life’ is shorthand for interpersonal life.

content is dictated by the particular psychopathological features present and the processes that appear to be maintaining them.

3. The treatment has been designed to be practicable under normal outpatient conditions. Thus it can be provided by male or female therapists (unlike those treatments which involve in-session mirror exposure), and it is not unduly labour-intensive when compared with existing treatments. No special facilities are required.
4. As operationalised for an ongoing research trial, the treatment is provided in two versions, a 20-session treatment for the majority of patients, and a 40-session treatment for patients who are significantly underweight (defined as a BMI ≤ 17.5). The two forms of the treatment are delivered over 20 and 40 weeks respectively with the initial sessions being twice-weekly. The style and content of the two versions are essentially the same except that the longer treatment also includes procedures designed to help patients regain weight. The optimal length of treatment and number of sessions remains to be determined.
5. The treatment is provided on a one-to-one basis. We believe that this is likely to be the best way of delivering the treatment given its idiographic nature.
6. The treatment has four stages:
 - Stage One is an intensive initial stage which lasts four weeks, the focus being on engaging and educating the patient, creating an initial personalised formulation, and obtaining maximal early behaviour change.
 - In Stage Two (which occupies one to three sessions) there is a detailed review of progress so far, as well as the characterisation of any barriers to change. In addition, there is a formal

assessment of the likely contribution of each of the four additional maintaining mechanisms specified above. The resulting information is used to create, jointly with the patient, a revised and extended formulation.

- Stage Three occupies the largest part of the treatment and its content is dictated by the revised formulation. It always includes emphasis on modifying the patient's eating disorder psychopathology (for example, the over-evaluation of eating, shape and weight and their control, and its various expressions), but it also involves addressing those additional processes identified in the revised formulation. To this end there are treatment “modules” focused on clinical perfectionism, core low self-esteem, mood intolerance and interpersonal difficulties. These are deployed as indicated.
- The final stage in treatment, Stage Four, is similar to the final stage in CBT-BN (Fairburn, Marcus & Wilson, 1993b), although it is broader in scope. The emphasis is on ensuring that progress is maintained after treatment ends. Sessions are fortnightly by this stage.

We have evolved this new transdiagnostic treatment over the past three years selecting patients of a type that do not typically respond well to a more focused style of approach (as exemplified by CBT-BN). The results have been sufficiently encouraging for us to embark on a transdiagnostic treatment trial designed to test not only the new treatment, but also the theory upon which it is based.

6. Broader implications

It has not escaped our attention that this transdiagnostic approach to theory and treatment has implications beyond the field of eating disorders⁶. We therefore close this paper by addressing the circumstances under which such a strategy is likely to be of value.

In principle, transdiagnostic conceptualisation and treatment is relevant when major clinical features shared by two or more diagnostic states are maintained by common pathological processes. We propose that the presence of either of the following two characteristics is suggestive of the operation of common maintaining processes, but that neither is sufficient:

- shared distinctive clinical features
- movement of patients between the diagnostic states

To establish the operation of transdiagnostic pathological processes there would need to be direct clinical and research evidence that the processes maintaining the diagnostic states do indeed overlap (for example, by identifying common distinctive obstacles to change or by developing common distinctive methods of achieving change).

We leave readers to consider the frequency with which these conditions apply within psychiatry.

⁶ Those familiar with the origins of behaviour therapy will recognise in our strategy the focus on individualised case formulation rather than psychiatric diagnosis, and the theory-driven design of patient-specific treatment.

Acknowledgements

All three authors are supported by the Wellcome Trust: CGF is supported by a Principal Research Fellowship (046386); ZC is supported by a programme grant (046386); and RS is supported by a Research Career Development award (063209). Without the Wellcome Trust's sustained support this work would not have been possible. The ongoing transdiagnostic trial is being conducted in collaboration with Dr Robert Palmer of the University of Leicester. This trial is funded by the programme grant from the Wellcome Trust.

References

- Agras, W. S., Crow, S. J., Halmi, K. A., Mitchell, J. E., Wilson, G. T., & Kraemer, H. C. (2000a). Outcome predictors for the cognitive behavioral treatment of bulimia nervosa: data from a multisite study. *American Journal of Psychiatry*, 157, 1302–1308.
- 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.
- American Psychiatric Association (1980). *DSM-III: diagnostic and statistical manual of mental disorders*. Washington, DC: American Psychiatric Association.
- American Psychiatric Association (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: American Psychiatric Association.
- American Psychiatric Association (2000). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: American Psychiatric Association Text revision.
- Beck, J. S. (1995). *Cognitive therapy: basics and beyond*. New York: Guilford Press.
- Brown, G. W., Adler, Z., & Bifulco, A. (1988). Life events, difficulties and recovery from chronic depression. *British Journal of Psychiatry*, 152, 487–498.
- Byrne, S. M., & McLean, N. J. (2002). The cognitive-behavioural model of bulimia nervosa: a direct evaluation. *International Journal of Eating Disorders*, 31, 17–31.
- Casper, R. C., Eckert, E. D., Halmi, K. A., Goldberg, S. C., & Davis, J. M. (1980). Bulimia: its incidence and clinical importance in patients with anorexia nervosa. *Archives of General Psychiatry*, 37, 1030–1035.
- Claes, L., Vandereycken, W., & Vertommen, H. (2001). Self-injurious behaviors in eating-disordered patients. *Eating Behaviors*, 2, 263–272.
- 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.
- Dare, C., & Eisler, I. (2002). Family therapy and eating disorders. In C. G. Fairburn, & K. D. Brownell (Eds.), *Eating disorders and obesity: a comprehensive handbook* (2nd ed.) (pp. 314–319). New York: Guilford Press.
- Eddy, K. T., Keel, P. K., Dorner, D. J., Delinsky, S. S., Franko, D. L., & Herzog, D. B. (2002). Longitudinal comparison of anorexia nervosa subtypes. *International Journal of Eating Disorders*, 31, 191–201.
- Fairburn, C. G. (1981). A cognitive behavioural approach to the management of bulimia. *Psychological Medicine*, 11, 707–711.
- Fairburn, C. G. (1985). Cognitive-behavioral treatment for bulimia. In D. M. Garner, & P. E. Garfinkel (Eds.), *Handbook of psychotherapy for anorexia nervosa and bulimia* (pp. 160–192). New York: Guilford Press.
- Fairburn, C. G. (1995). *Overcoming binge eating*. New York: Guilford Press.
- Fairburn, C. G. (1997a). Eating disorders. In D. M. Clark, & C. G. Fairburn (Eds.), *Science and practice of cognitive behaviour therapy* (pp. 209–241). Oxford: Oxford University Press.
- Fairburn, C. G. (1997b). Interpersonal psychotherapy for bulimia nervosa. In D. M. Garner, & P. E. Garfinkel (Eds.), *Handbook of treatment for eating disorders* (pp. 278–294). New York: Guilford Press.
- 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: Basic Books.
- Fairburn, C. G., Cooper, Z., Doll, H. A., Norman, P., & O'Connor, M. E. (2000). The natural course of bulimia nervosa and binge eating disorder in young women. *Archives of General Psychiatry*, 57, 659–665.
- Fairburn, C. G., Cooper, Z., & Shafran, R. (2002). *Cognitive behaviour therapy for eating disorders*. Oxford: University of Oxford unpublished manuscript.
- Fairburn, C. G., Jones, R., Peveler, R. C., Hope, R. A., & O'Connor, M. E. (1993a). Psychotherapy and bulimia nervosa: The longer-term effects of interpersonal psychotherapy, behaviour therapy and cognitive behaviour therapy. *Archives of General Psychiatry*, 50, 419–428.
- Fairburn, C. G., Kirk, J., O'Connor, M. E., Anastasiades, P., & Cooper, P. J. (1987). Prognostic factors in bulimia nervosa. *British Journal of Clinical Psychology*, 26, 223–224.
- Fairburn, C. G., Marcus, M. D., & Wilson, G. 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: Guilford Press.
- Fairburn, C. G., Norman, P. A., Welch, S. L., O'Connor, M. E., Doll, H. A., & Peveler, R. C. (1995). A prospective study of outcome in bulimia nervosa and the long-term effects of three psychological treatments. *Archives of General Psychiatry*, 52, 304–312.
- Fairburn, C. G., Peveler, R. C., Jones, R., Hope, R. A., & Doll, H. A. (1993c). Predictors of twelve-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., Shafran, R., & Cooper, Z. (1999). A cognitive behavioural theory of anorexia nervosa. *Behaviour Research and Therapy*, 37, 1–13.
- Fairburn, C.G., Stice, E., Cooper, Z., Doll, H.A., Norman, P.A., & O'Connor, M.E. (in press). Understanding persistence in bulimia nervosa: a five-year naturalistic study. *Journal of Consulting and Clinical Psychology*.
- Fairburn, C. G., & Walsh, B. T. (2002). Atypical eating disorders. In C. G. Fairburn, & K. D. Brownell (Eds.), *Eating disorders and obesity: a comprehensive handbook* (2nd ed.) (pp. 171–177). New York: Guilford Press.
- Garfinkel, P. E., Moldofsky, H., & Garner, P. E. (1980). The heterogeneity of anorexia nervosa: bulimia as a distinct subgroup. *Archives of General Psychiatry*, 37, 1036–1040.
- Garner, D. M. (1997). Psychoeducational principles in treatment. In D. M. Garner, & P. E. Garfinkel (Eds.), *Handbook of treatment for eating disorders* (pp. 145–177). New York: Guilford Press.
- Garner, D. M., & Bemis, K. M. (1982). A cognitive-behavioral approach to anorexia nervosa. *Cognitive Therapy and Research*, 6, 123–150.
- Garner, D. M., & Bemis, K. M. (1985). A cognitive-behavioral approach to anorexia nervosa. In D. M. Garner, & P. E. Garfinkel (Eds.), *Handbook of psychotherapy for anorexia nervosa and bulimia* (pp. 107–146). New York: Guilford Press.
- Garner, D. M., Rockert, W., Olmsted, M. P., Johnson, C., & Coscina, D. V. (1985). Psychoeducational principles in the treatment of bulimia and anorexia nervosa. In D. M. Garner, & P. E. Garfinkel (Eds.), *Handbook of psychotherapy for anorexia nervosa and bulimia* (pp. 513–572). New York: Guilford Press.
- Garner, D. M., Vitousek, K. M., & Pike, K. M. (1997). Cognitive-behavioral therapy for anorexia nervosa. In D. M. Garner, & P. E. Garfinkel (Eds.), *Handbook of treatment for eating disorders* (pp. 94–144). New York: Guilford Press.
- Greenberger, D., & Padesky, P. A. (1995). *Mind over mood*. New York: Guilford Press.
- Grilo, C. M. (2002). Binge eating disorder. In C. G. Fairburn, & K. D. Brownell (Eds.), *Eating disorders and obesity: a comprehensive handbook* (2nd ed.) (pp. 178–182). New York: Guilford Press.
- Holderness, C. C., Brooks-Gunn, J., & Warren, M. P. (1994). Co-morbidity of eating disorders and substance abuse: review of the literature. *International Journal of Eating Disorders*, 16, 1–34.
- Hollon, S. D., & Beck, A. T. (1993). Cognitive and cognitive-behavioral therapies. In S. L. Garfield, & A. E. Bergin (Eds.), *Handbook of psychotherapy and behavior change: an empirical analysis* (2nd ed.) (pp. 428–466). New York: Wiley.
- Klerman, G. L., Weissman, M. M., Rounsaville, B. J., & Chevron, E. S. (1984). *Interpersonal psychotherapy of depression*. New York: Basic Books.

- Lock, J., le Grange, D., Agras, W. S., & Dare, C. (2001). *Treatment manual for anorexia nervosa: a family-based approach*. New York: Guilford Press.
- Meyer, C., Waller, G., & Waters, A. (1998). Emotional states and bulimic psychopathology. In H. W. Hoek, J. L. Treasure, & M. A. Katzman (Eds.), *Neurobiology in the treatment of eating disorders* (pp. 271–287). Chichester: Wiley.
- McIntosh, V. V., Bulik, C. M., McKenzie, J. M., Luty, S. E., & Jordan, J. (2000). Interpersonal psychotherapy for anorexia nervosa. *International Journal of Eating Disorders*, 27, 125–139.
- Millar, H. R. (1998). New eating disorder service. *Psychiatric Bulletin*, 22, 751–754.
- Padesky, C. A., & Greenberger, D. (1995). *Clinician's guide to mind over mood*. New York: Guilford Press.
- Paul, T., Schroeter, K., Dahme, B., & Nutzinger, D. O. (2002). Self-injurious behavior in women with eating disorders. *American Journal of Psychiatry*, 159, 408–411.
- Pike, K. M., Loeb, K., & Vitousek, K. (1996). Cognitive-behavioral therapy for anorexia nervosa and bulimia nervosa. In J. K. Thompson (Ed.), *Body image, eating disorders, and obesity* (pp. 253–302). Washington: American Psychological Association.
- Polivy, J., & Herman, C. P. (1993). Etiology of binge eating: psychological mechanisms. In C. G. Fairburn, & G. T. Wilson (Eds.), *Binge eating: nature, assessment and treatment* (pp. 173–205). New York: Guilford Press.
- Ricca, V., Mannucci, E., Mezzani, B., DiBernardo, M., Zucchi, T., Paionni, A., Placidi, G. P. A., Rotella, C. M., & Faravelli, C. (2001). Psychopathological and clinical features of outpatients with an eating disorder not otherwise specified. *Eating and Weight Disorders*, 6, 157–165.
- Rosen, J. C. (1996). Body image assessment and treatment in controlled studies of eating disorders. *International Journal of Eating Disorders*, 20, 331–343.
- Rosen, J. C. (1997). Cognitive-behavioural body image therapy. In D. M. Garner, & P. E. Garfinkel (Eds.), *Handbook of treatment for eating disorders* (pp. 188–201). New York: Guilford Press.
- Russell, G. F. M. (1979). Bulimia nervosa: an ominous variant of anorexia nervosa. *Psychological Medicine*, 9, 429–448.
- Segal, Z. V., Williams, J. M. G., & Teasdale, J. D. (2002). *Mindfulness-based cognitive therapy for depression*. New York: Guilford Press.
- Shafran, R., Cooper, Z., & Fairburn, C. G. (2002). Clinical perfectionism: a cognitive-behavioural analysis. *Behaviour Research and Therapy*, 40, 773–791.
- Steiger, H., Leung, F., & Thibaudeau, J. (1993). Prognostic value of pretreatment social adaptation in bulimia nervosa. *International Journal of Eating Disorders*, 14, 269–276.
- Steiger, H., Gauvin, L., Jabalpurwala, S., Seguin, 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.
- Steinberg, S., Tobin, D., & Johnson, C. (1990). The role of bulimic behaviors in affect regulation: different functions for different subgroups? *International Journal of Eating Disorders*, 9, 51–55.
- Steinhausen, H. -C. (1991). Follow-up studies of anorexia nervosa: a review of four decades of outcome research. *Psychological Medicine*, 21, 447–454.
- Stice, E. (1994). Review of the evidence for a sociocultural model of bulimia nervosa and an exploration of the mechanisms of action. *Clinical Psychology Review*, 14, 633–661.
- Sullivan, P. F., Bulik, C. M., Fear, J. L., & Pickering, A. (1998). Outcome of anorexia nervosa: a case-control study. *American Journal of Psychiatry*, 155, 939–946.
- Waller, G. (2002). The psychology of binge eating. In C. G. Fairburn, & K. D. Brownell (Eds.), *Eating disorders and obesity: a comprehensive handbook* (2nd ed.) (pp. 98–102). New York: Guilford Press.
- Wilson, G. T. (1996). Treatment of bulimia nervosa: when CBT fails. *Behaviour Research and Therapy*, 34, 197–212.
- Wilson, G. T. (1998a). Manual-based treatment and clinical practice. *Clinical Psychology: Science & Practice*, 5, 363–375.
- Wilson, G. T. (1998b). The clinical utility of randomized controlled trials. *International Journal of Eating Disorders*, 24, 13–29.
- Wilson, G. T. (1999a). Rapid response to cognitive behavior therapy. *Clinical Psychology: Science & Practice*, 6, 289–292.
- Wilson, G. T. (1999b). Cognitive behavior therapy for eating disorders: progress and problems. *Behaviour Research and Therapy*, 37, S79–S95.

- Wilson, G. T., & Fairburn, C. G. (2000). The treatment of binge eating disorder. *European Eating Disorders Review*, 8, 351–354.
- Wilson, G. T., & Fairburn, C. G. (2002). Treatments for eating disorders. In P. E. Nathan, & J. M. Gorman (Eds.), *A guide to treatments that work* (2nd ed.) (pp. 559–592). New York: Oxford University Press.
- Wilson, G. T., Fairburn, C. G., & Agras, W. S. (1997). Cognitive-behavioral therapy for bulimia nervosa. In D. M. Garner, & P. Garfinkel (Eds.), *Handbook of treatment for eating disorders* (pp. 67–93). New York: Guilford Press.
- Wilson, G. T., Fairburn, C. G., Agras, W. S., Walsh, B. T., & Kraemer, H. (2002). Cognitive behavior therapy for bulimia nervosa: time course and mechanisms of change. *Journal of Consulting and Clinical Psychology*, 70, 267–274.
- Wonderlich, S. A. (2002). Personality and eating disorders. In C. G. Fairburn, & K. D. Brownell (Eds.), *Eating disorders and obesity: a comprehensive handbook* (2nd ed.) (pp. 204–209). New York: Guilford Press.