# Anxiety and eating disorders understanding the overlap

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# Anxiety and eating disorders: Understanding the overlap

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# CLINICAL PSYCHOLOGY REVIEW

# Anxiety in the eating disorders: Understanding the overlap

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## Abstract

This paper reviews research investigating the comorbidity between eating disorders and anxiety disorders. Whilst there is some inconsistency in the literature, it appears that women with eating disorders have higher rates of anxiety disorders than normal controls. Potential causal relationships between eating disorders and anxiety disorders are outlined, though their relative chronology appears to be somewhat inconsistent. Safety behaviours and cognitive avoidance strategies (i.e., cognitive narrowing and blocking) are suggested as potential mechanisms linking the disorders. A model outlining this hypothesised relationship is developed throughout the review. It is suggested that eating disorders and anxiety disorders might share common aetiological factors, and that these factors can increase an individual's susceptibility to either disorder. Potential implications for the treatment of eating disorders are outlined, and suggestions are made for further research.
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## Contents

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It is difficult to establish accurate prevalence rates for eating disorders, both due to variations in methods used for diagnosis and because much of the research has focused exclusively on the diagnoses of anorexia nervosa and bulimia nervosa. Hoek (1993, 2002) describes how two-stage surveys (using screening measures followed by strict diagnostic interviews) yield lower prevalence rates than earlier surveys based solely on questionnaires. On the basis on these methods, he suggests that the average point prevalence rates amongst females[1] are just 0.28% for anorexia nervosa and approximately 1.0% for bulimia nervosa. However, based on a range of epidemiological studies (including Hoek's, 1993 review), Fairburn and Harrison (2003) suggest slightly higher prevalence figures of 0.7% for anorexia nervosa, and 1–2% for bulimia nervosa. Indeed, these rates are relatively consistent with the American Psychiatric Association's prevalence estimates of 0.5–1% for anorexia nervosa, and 1–3% for bulimia nervosa (American Psychiatric Association, 1994).

Of course, none of these prevalence figures takes into account the diagnosis of 'eating disorder not otherwise specified' (EDNOS; DSM-IV, American Psychiatric Association, 1994), or the equivalent in ICD-10 of 'atypical eating disorders' (World Health Organisation, 1992). These disorders appear to be at least as common in clinical practice as anorexia nervosa and bulimia nervosa combined (Fairburn & Harrison, 2003; Fairburn & Walsh, 2002), and consequently Fairburn and Walsh (2002) suggest that current prevalence rates underestimate the true number of eating disorder cases. Indeed, Grilo (2002) cites evidence that binge eating disorder (one of the EDNOS disorders described in DSM-IV), has prevalence rates of around 2–3% in community samples.

Despite the lack of clarity regarding their prevalence, it is clear that an eating disorder can impact negatively on the sufferer's quality of life. Furthermore, eating disorders have been associated with a high rate of psychiatric comorbidity. For example, Braun, Sunday, and Halmi (1994) reported that 81.9% of their sample of women with eating disorders had at least one Axis I comorbidity, with depression, anxiety, and substance misuse being especially common. Moreover, 69% of this sample met criteria for at least one personality disorder. Since the presence of comorbidities is likely to complicate the formulation and treatment of eating disorders (O'Brien & Vincent, 2003), there is a clinical need to understand more about their prevalence.

Of particular interest in this review are the different manifestations of anxiety found to be comorbid with eating disorders. Although depression is the most frequently diagnosed comorbid disorder (e.g., Braun et al., 1994; Herzog, Keller, Sacks, Yeh, & Lavori, 1992), eating disorder patients have reported that anxiety and anger are more likely to drive binges than depression (Arnow, Kenardy, & Agras, 1992). In addition, anxiety has been associated with further disordered eating behaviours, including vomiting (Carter & Duncan, 1984), laxative abuse (Weltzin, Bulik, McConaha, & Kaye, 1995), and restriction (Chesler, 1995). Moreover, Weltzin et al. (1995) suggest that significant anxiety not

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[1] Like the majority of eating disorders research, most prevalence studies are based on female samples, since only 5–10% of patients in clinical samples are male (Hoek, 1993, 2002). The female pronoun will therefore be used throughout this review to refer to those with eating disorders.

only reduces compliance with therapy, but also often leads to premature termination of treatment. However, it remains the case that the great majority of the evidence for anxiety as a risk factor in the eating disorders comes from retrospective studies (e.g., Jacobi, Hayward, de Zwann, Kraemer, & Agras, 2004), and that almost all the evidence of causal impact of emotional factors comes from experimental studies that have manipulated depression rather than anxiety (e.g., Stice, 2002). Therefore, the potential for understanding the role of anxiety in the development and maintenance of the eating disorders is still to be realised.

In this review of anxiety in the eating disorders, we begin by briefly outlining the general cognitive theory of anxiety. We then discuss the patterns of comorbidity associated with eating disorders and anxiety disorders, and delineate potential temporal relationships between these conditions. We go on to discuss how both safety behaviours and cognitive avoidance strategies might be potential mechanisms linking anxiety and eating pathology, and consider how cognitive avoidance strategies relate to information processing models of anxiety. To conclude the review, we consider both potential implications for the treatment of eating disorders and avenues for future research.

## 1. Cognitive model of anxiety

Beck et al. have proposed a cognitive model of anxiety disorders (Beck, Emery, & Greenberg, 1985), which has been adapted and elaborated by numerous authors, including Beck and Clark (1997). According to the Beck et al. (1985) model, the central problem of anxiety involves the allocation of excessive threat meaning to innocuous situations or stimuli, along with an underestimation of personal coping resources. They propose that this bias results in cognitive, affective, physiological, and behavioural changes in the individual (Beck et al., 1985). Cognitive effects might include fear- and vulnerability-related beliefs and images, difficulty concentrating and reasoning, and hypervigilance for threat. These factors are likely to intensify the threat meaning assigned to the object or situation.

Physiologically, autonomic hyperarousal occurs in preparation for the fight, flight, freeze, or faint response, and consequently the individual may be behaviourally mobilized (to fight or escape). However, this preparation for action may be experienced as aversive physical symptoms (such as palpitations, breathlessness, or dizziness), which further increase the individual's emotional arousal (e.g., Wells, 1997). An alternative physiological reaction suggested by Beck et al. (1985) is that motor responses may be deactivated, resulting in immobilisation and feelings of helplessness and increased vulnerability. In an attempt to prevent the anxiety response outlined above, anxious individuals inhibit risk-taking and maximise safety-seeking and avoidance. However, as with escape, the use of safety behaviours and avoidance strategies prevent the individual from learning that the imagined threat might be exaggerated (e.g., Wells, 1997). Hence, anxiety is maintained.

The above account gives a general description of the cognitive theory of anxiety. However, anxiety is a broad concept incorporating a range of diagnoses, and of course the particular form anxiety takes may be different in different diagnoses. Hence, not all anxiety disorders will be equally associated with eating disorders. The following section will therefore consider the patterns of comorbidity found amongst eating disorders and a range of anxiety disorders.

## 2. Phenomenological associations between anxiety and the eating disorders

In this section, we consider phenomenological patterns of comorbidity between specific anxiety diagnoses and the eating disorders, and the developmental sequencing of those patterns. However, much of the research cited in this review is correlational in nature. It should therefore be remembered that whilst associations between disorders may suggest possible aetiological links, causation cannot be inferred from correlation. In the following section, we consider the causality behind those relationships.

### 2.1. Patterns of comorbidity

In order to understand the nature of associations more clearly, it is important to consider comorbidity from both perspectives — the prevalence of anxiety disorders amongst people with eating disorders, and the prevalence of eating disorders amongst people with anxiety disorders. However, given the relatively low prevalence of eating disorders (American Psychiatric Association, 1994; Fairburn & Harrison, 2003; Hoek, 1993, 2002), there is less research evidence regarding the latter. Even then, the limited evidence relates only to certain anxiety disorders. When reviewing research on comorbidities, O'Brien and Vincent (2003) highlight the importance of considering factors such as sample

size and the presence or absence of comparison groups. These issues will therefore be highlighted in relation to the studies presented below.

### 2.1.1. Comorbidity with generalised anxiety disorder

According to DSM-IV (American Psychiatric Association, 1994), generalised anxiety disorder (GAD) is characterised by excessive anxiety and worry on a variety of topics, occurring on more days than not for at least six months. Some studies have reported high rates of GAD amongst women with eating disorders. For example, Schwalberg, Barlow, Algar, and Howard (1992) report lifetime prevalence rates of 55% in referred women with bulimia nervosa, and 36.4% in obese binge eaters. However, the small sample sizes in this study ($N=20$ and $N=22$ respectively) are likely to affect the accuracy of the prevalence rates. In fact, as noted by Godart, Flament, Perdereau, and Jeammet (2002), there are wide variations in the estimated prevalence rates of GAD across eating disorder studies. For example, Lilenfeld, Kaye, Greeno, Merikangas, Plotnicov, Pollice et al. (1998) report a GAD lifetime prevalence rate of just 13% amongst patients with bulimia nervosa, which was not significantly different to the prevalence rate amongst controls (2%). On the other hand, they report prevalence rates of 31% amongst restrictive anorexics (significantly different to the controls). However, in contrast, Garfinkel, Lin, Goering, Spegg, Goldbloom, Kennedy et al. (1995) found that the lifetime prevalence rate of GAD amongst a community sample of women with bulimia nervosa was significantly different to the rate amongst comparison women (10.9% and 2.5% respectively).

In their review of anxiety comorbidity in the eating disorders, Godart et al. (2002) highlight the heterogeneous nature of the samples, methods, and results of previous research on GAD, and suggest that it is difficult to come to any firm conclusions about prevalence rates. In response, Godart, Flament, Curt, Perdereau, Lang, Venisee et al. (2003) designed a more controlled study, in which a larger sample size was used (271 women with a current eating disorder diagnosis, and 271 matched controls from the community). They investigated a range of DSM-IV comorbid anxiety disorders in this study, including GAD. Godart et al. (2003) reported significantly higher lifetime prevalence rates for GAD amongst referred women with anorexia nervosa compared to controls (48.6% in the restricting subtype compared to 3.6% amongst matched controls, and 45.4% in the binge/purge subtype compared to 10.9% amongst matched controls). They also reported significantly higher rates of GAD amongst women with bulimia nervosa of the purging subtype (32.6% compared to 7% amongst matched controls). Unfortunately, there were too few women with bulimia nervosa of the non-purging subtype to detect differences in the rates of particular anxiety disorders.

Despite the fact that the above findings are not entirely consistent, GAD does appear to be a relatively common comorbidity amongst eating disorder samples. Moreover, the results suggest that GAD is associated with the full range of eating pathologies, and is not just associated with particular behaviours such as purging, bingeing, or restriction. Although many studies do not report on the particular presentation of GAD in women with eating disorders, Schwalberg et al. (1992) state that at least one of the spheres of worry in this group was invariably related to eating, shape, and weight (although of course a diagnosis of GAD cannot be made unless there are also additional areas of worry).

### 2.1.2. Comorbidity with phobic anxiety

According to DSM-IV criteria, a specific phobia refers to marked and excessive fear of a particular object or situation (e.g., heights, animals, receiving an injection). Exposure to this situation consistently provokes an immediate anxiety response, which can take the form of a panic attack. As with generalised anxiety disorder, the literature on comorbidity between eating disorders and simple phobia is somewhat inconsistent.

Bulik, Sullivan, Carter, and Joyce (1996) reported a 30% lifetime prevalence rate for simple phobias in their clinical study of 114 women with bulimia nervosa. However, as there was no control group in this study, it is not clear how this compares to normal controls. Wittchen and Essau (1993) suggest that simple phobia is the most frequent anxiety disorder in community samples, affecting between 8.5% and 25.9% of women. Indeed, Lilenfeld et al. (1998) failed to find a significant difference between prevalence rates in women with bulimia nervosa (19%) and control women (7%), although they did find a significant difference between the control group and women with anorexia nervosa (27%). Conversely, despite finding a difference in the overall prevalence of anxiety disorders, Halmi, Eckert, Marchi, Sampugnaro, Apple, and Cohen (1991) found no significant difference in the lifetime prevalence of simple phobia in women with a history of anorexia nervosa (followed up after ten years) compared to controls. However, in their community study, Garfinkel et al. (1995) found significantly higher lifetime rates of simple phobia both in women with full-syndrome bulimia nervosa (40.0%) and in those with partial-syndrome bulimia nervosa (40.9%) compared to controls (11.4%).

As with GAD, Godart et al. (2002) cite a wide range of prevalence figures, many based on small sample sizes. Overall, the inconsistent results and range of prevalence rates cited (for both clinical and control participants) make it difficult to draw any strong conclusions regarding the comorbidity of eating disorders and simple phobias. It is also not possible to tell from the studies whether any particular phobias (e.g., emetiphobia) are more or less common in women with eating disorders. It is of course possible that phobias related to eating (and associated situations) could be subsumed under the more general heading of social phobia (see later section).

### 2.1.3. Comorbidity with agoraphobia

Agoraphobia is defined in DSM-IV as: "Anxiety about being in places or situations in which help may not be available in the event of having an unexpected or situationally predisposed panic attack". Halmi et al. (1991) report significantly higher lifetime prevalence rates of agoraphobia in their follow up study of women with a history of anorexia (most of whom had shifted diagnosis to EDNOS or bulimia nervosa, or were no longer diagnosable; 14.5% compared to 3.2% amongst controls). Using a community sample, Garfinkel et al. (1995) also found a significantly higher lifetime rate of agoraphobia amongst women with full-syndrome bulimia nervosa (34.5%) compared to controls (7.5%). However, in their review of comorbidity amongst individuals with eating disorders, Godart et al. (2002) report comorbidity rates for agoraphobia from 0% to 34.5%, and suggest that findings are "scarce and contradictory" (p. 267). Following on from this, Godart et al. (2003) report significantly higher lifetime rates of agoraphobia in women with anorexia nervosa compared to matched controls (19.8% in the restricting subtype compared to 2.7% amongst matched controls, and 27.3% in the binge/purge subtype compared to 7.3% amongst matched controls). They also report significantly higher lifetime rates of agoraphobia amongst women with bulimia nervosa of the purging subtype (17.4% compared to 4.7% in matched controls). The non-purging bulimia group was too small to carry out statistical analyses.

Despite some mixed findings, the studies reviewed suggest that agoraphobia is more prevalent in eating disorder samples compared to normal controls, although there is no apparent evidence that particular eating behaviours (restricting, bingeing, or purging) are associated with this anxiety disorder.

### 2.1.4. Comorbidity with social phobia

Social phobia is characterised by a fear of social or performance situations owing to excessive concern over negative evaluation by others (DSM IV; American Psychiatric Association, 1994). A number of studies have demonstrated associations between social phobia and eating disorders. For example, Halmi et al. (1991) found significantly higher lifetime rates of social phobia in their eating disorders group (33.9%) compared to controls (3.2%). Moreover, Godart et al. (2003) observed similarly high lifetime and current rates of social phobia amongst their clients with eating disorders, and reported that social phobia was the most frequent anxiety disorder in bulimia nervosa (lifetime prevalence of 36.0% in the purging subtype compared to 8.1% amongst matched controls, and 36.8% in the non-purging subtype; though this group was too small to detect differences with the controls). Social phobia was also the second most frequent anxiety disorder in anorexia nervosa (lifetime prevalence rates of 37.8% in the restricting subtype compared to 5.4% amongst matched controls, and 41.8% in the binge/purge subtype compared to 12.7% amongst matched controls). Similar results have also been found in community based studies. For example, Garfinkel et al. (1995) found significantly different prevalence rates between individuals with eating difficulties and those without (45.5% in women with full-syndrome bulimia nervosa, 54.5% in women with partial-syndrome bulimia nervosa, and 15.2% amongst controls).

However, not all studies have reported this pattern of results. For example, whilst Bulik, Sullivan, Fear, and Joyce (1997) found a significantly higher rate of social phobia amongst women with bulimia nervosa (30.2%) compared to normal controls (6.1%), they did not find an increased rate amongst women with anorexia nervosa (5.9%). Bulik et al. (1997) acknowledge that this latter finding is inconsistent with other comorbidity studies. By way of explanation, they propose that despite an increased vulnerability, social phobia may not be expressed in women with anorexia nervosa because situations that provoke social anxiety are typically avoided. Consequently, they suggest that the low prevalence rate reported might reflect a lack of social interactions rather than a lack of social anxiety. It might also be worth considering that the two eating disorder groups in this study were selected using quite different methods. Whilst the bulimia nervosa group consisted of consecutive referrals to an outpatient treatment trial (for bulimia nervosa), the anorexia nervosa group were identified and contacted on the basis of a retrospective review of clinical files (of individuals referred to the eating disorders service between 1981 and 1984). Perhaps this group of women with anorexia nervosa was therefore 'atypical' in some way.

As mentioned previously, it can also be helpful to consider rates of eating disorders amongst samples of anxious clients. For example, Wittchen, Stein, and Kessler (1999) investigated the prevalence of social phobia and related comorbidities, using a community sample of 3021 individuals (male and female) between the ages of 14 and 24 years. They found significantly higher rates of eating disorders (mostly EDNOS) among young people who met criteria for generalised social phobia (15%) compared to those who did not (2.6%). However, since they do not specify the EDNOS diagnoses given, it is not possible to consider whether particular kinds of eating pathologies are related. Similarly, Becker, DeViva, and Zayfert (2004) investigated the rates of eating disorders amongst clients presenting at an anxiety clinic. They found that nearly 12% of their sample met criteria for a probable eating disorder (based on a questionnaire measure). Although the rates of probable eating disorder did not differ significantly across the anxiety disorder diagnoses, social phobia had unique variance in the regression analysis.

Therefore, despite some inconsistencies, these studies suggest that social phobia is indeed more prevalent in eating disorders, and that eating disorders might be more prevalent in social phobia cases. However, it is not clear from the studies above how social phobia might be linked to different eating pathologies. However, Garfinkel, Lin, Goering, Spegg, Goldbloom, Kennedy et al. (1996) found significantly higher lifetime rates of social phobia in individuals with bulimia nervosa of the purging subtype (64.7%) compared to those with bulimia nervosa of the non-purging subtype (31.1%). Furthermore, in their dimensional study of social anxiety (a necessary component of social phobia), Hinrichsen, Wright, Waller, and Meyer (2003) investigated the levels of social anxiety across different eating disorder diagnoses, as well as the types of emotion regulation mechanisms typically used. Based on questionnaire responses, they found that social anxiety was higher amongst women with eating disorders (compared to non-clinical controls), particularly those with binge-purge anorexia. Moreover, they found that higher levels of social anxiety were associated with higher levels of bulimic pathology (as measured by questionnaire) in women with bulimia nervosa and non-clinical controls. On the other hand, in the restrictive anorexic group, higher levels of social anxiety were associated with higher levels of dissociation (again measured by questionnaire). However, despite finding the highest levels of social anxiety amongst women with anorexia nervosa of the binge/purge subtype, this diagnosis was not associated with any particular emotion regulation strategies.

### 2.1.5. Comorbidity with obsessive-compulsive pathology

The main feature of obsessive-compulsive disorder (OCD) is the occurrence of persistent and time-consuming obsessions or compulsions (DSM IV; American Psychiatric Association, 1994). Obsessions may be inappropriate and intrusive thoughts, impulses, or images (such as concern over contamination). Compulsions are repetitive behaviours (either overt or covert), whose goal is to prevent or reduce anxiety or distress. In their review of obsessive-compulsive disorder and anorexia nervosa, Serpell, Livingstone, Neiderman, and Lask (2002) conclude that a substantial number of clients with anorexia nervosa display current obsessive and compulsive features, with an even greater proportion meeting OCD criteria during their lifetimes.

Godart et al. (2002) report that OCD has been the most extensively studied anxiety disorder in eating disorder samples, with significantly higher comorbidity rates consistently reported in clinical samples compared to controls. For example, Halmi et al. (1991) report a lifetime prevalence rate of 25.8% amongst women with a history of anorexia (followed up after ten years) compared to 6.5% amongst controls. Unfortunately, due to small sample sizes in this study it was not possible to investigate whether lifetime rates of OCD are different amongst women with different current eating pathologies. However, other research in this area suggests that increased OCD prevalence is more consistently related to restrictive eating pathologies. For example, Godart et al. (2003) found significantly higher lifetime rates of OCD in women with anorexia nervosa compared to controls (24.3% in the restrictive subtype compared to 2.7% amongst matched controls, and 23.6% in the binge/purge subtype compared to 5.4% amongst matched controls). However, they did not find significant differences between women with bulimia nervosa and controls. Whilst they found OCD prevalence rates of 16.7% in women with non-purging bulimia nervosa, this was based on a small sample size ($N = 19$) and was therefore not significantly different to matched controls (5.3%). Among women with bulimia nervosa of the purging subtype, the lifetime prevalence rate for OCD was 9.4%, which was again not significantly different to matched controls (3.5%). Bulik et al. (1997) reported a similar pattern of results, finding a significantly higher prevalence rate for OCD among women with anorexia nervosa (16.2%) compared to women with bulimia nervosa (3.5%), and community controls (2%).

Despite finding a higher rate of eating disorders amongst anxious clients, Becker et al. (2004) did not find that OCD contributed unique variance in their regression analysis. In light of this, they suggest that researchers should consider

that apparent relationships between OCD and eating disorders might be due to other comorbid anxiety disorders, rather than being due to OCD *per se*. Moreover, it is important to consider that whilst comorbidity studies can suggest associations between conditions, they cannot explain why that association exists. In fact, there are a variety of ways in which comorbidities can be explained. These will be explored in the next section of this review.

In summary, it seems that OCD has been more clearly associated with restrictive eating disorder presentations, as opposed to more bulimic presentations. However, the studies reviewed highlight the importance of considering the existence of further comorbidities when considering the likely relationship between eating disorders and anxiety disorders.

### 2.1.6. Conclusion

Whilst the comorbidity literature is certainly not entirely consistent, the evidence reviewed suggests that women with eating disorders do have higher rates of anxiety disorders compared to normal controls. In particular, higher rates of generalised anxiety disorder, social phobia, and agoraphobia appear to be associated with all types of eating pathology. On the other hand, obsessive-compulsive disorder has been more consistently associated with restrictive eating pathologies.

Of course, the majority of studies reviewed were based on clinical samples, which means that comorbidity might be overrepresented. Du Fort, Newman, and Bland (1993) suggest a number of potential reasons for this, including the phenomenon known as Berkson's bias. This bias is seen in the fact that individuals with more than one psychiatric illness are more likely to seek treatment than those with a single diagnosis. In addition, clinicians may be more likely to refer individuals with multiple diagnoses (Feinstein, Walter, & Horowitz, 1986). However, whilst these factors might limit the generalizability of clinical comorbidity studies to community samples, they are likely to give an accurate representation of the levels of comorbidity typically seen in routine clinical practice.

### 2.2. Temporal pattern

Godart et al. (2003) highlight the importance of exploring the nature of the relationship between eating disorders and anxiety disorders. They propose three possible explanations for the comorbidities found between these disorders, suggesting that: (a) anxiety might be a risk factor for eating disorders; (b) anxiety might be secondary to an eating disorder; or (c) the two kinds of disorders may have common shared vulnerability. Prospective studies of 'at risk' populations are difficult to carry out in this area, due to the low base rates of eating disorders (e.g., Serpell et al., 2002). Consequently, research has typically focused on retrospective reports of the temporal relationship between anxiety and eating disorders in order to draw inferences about possible aetiological links.

Where anxiety disorders are comorbid with eating disorders, anxiety is frequently reported to have developed prior to the eating disorder. For example, Godart et al. (2003) found that of those women with comorbid anxiety problems, 47% had at least one anxiety disorder preceding the onset of their eating disorder. In particular, social phobia commonly occurred first (between 51.2% and 71.4% of cases depending on the eating disorder subtype; with higher rates associated with more bulimic pathologies). Similarly, using a large sample of adults with an eating disorder diagnosis, Kaye, Bulik, Thornton, Barbarich, Masters, and Price Foundation Collaborative Group (2004) found that a number of anxiety disorders (obsessive compulsive disorder – OCD; social phobia; specific phobias; generalised anxiety disorder – GAD) typically began in childhood, and usually preceded the onset of the eating disorder. Other anxiety-based disorders (post traumatic stress disorder; panic disorder; agoraphobia) tended to have their origins at about the same age or later than the eating disorder. These findings were supported by a further study of comorbidity with obsessive compulsive disorder (Kaye, Bulik, Thornton, Plotnicov, & Price Foundation Collaborative Investigators, 2006). One potential explanation for these findings is that some forms of childhood anxiety have an aetiological role in the development of eating disorders (as represented schematically in Fig. 1a). For example, Schwalberg et al. (1992) suggest that anxiety about social evaluation might lead to excessive concerns over eating, shape, and weight (all of which are known to be key elements in the pathology of eating disorders; e.g., Fairburn, Cooper, & Shafran, 2003). Accordingly, social anxiety/phobia could be hypothesised to be a potential pathway to, or risk factor for, eating disorders.

In relation to restrictive pathologies in particular, Bulik et al. (1997) found that where OCD was comorbid with anorexia nervosa, most individuals reported that the OCD occurred prior to the onset of the eating disorder. Indeed, Bulik et al. (1997) propose that OCD might increase the probability that an eating disorder is expressed as anorexia
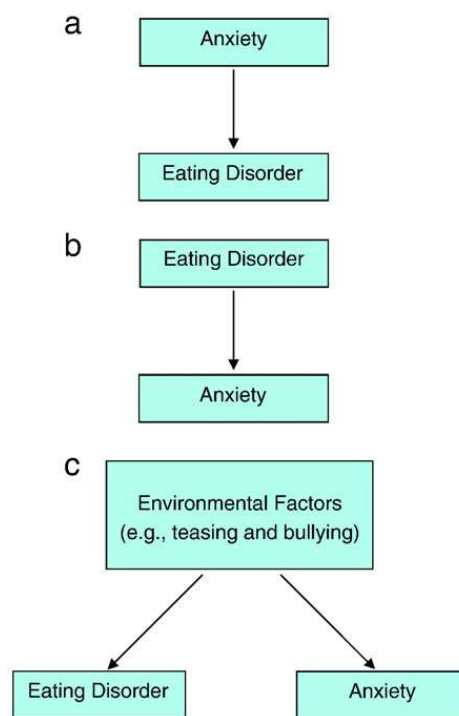
Fig. 1. Possible phenomenological links between anxiety and eating disorders, showing different possible models of risk for comorbidity.

nervosa (though they do not indicate how this effect might occur). Related to this question, Jacobi et al. (2004) suggest that the interaction between anxiety and specific prodromal symptoms of eating disorders needs to be further explored.

Of course, anxiety disorders do not invariably precede the onset of eating disorders. In contrast to the findings discussed above, Godart et al. (2003) found that OCD, along with panic disorder and GAD, more typically appeared simultaneously or after the onset of the eating disorder. Kaye et al. (2004) similarly found that panic disorder and agoraphobia more usually developed subsequently. This leaves the possibility that eating disorders could produce or at least exacerbate anxiety (including OCD and GAD symptoms) in some individuals. This potential relationship between eating disorders and anxiety is represented in Fig. 1b.

Part of this association might be connected to starvation effects. For example, Keys, Brozek, Henschel, Mickelsen, and Taylor (1950) demonstrated that caloric deprivation could cause obsessive-compulsive features in previously asymptomatic individuals. They found that following a prolonged period of food restriction; obsessions and compulsions concerned with the preparation and consumption of food were particularly common. Serpell et al. (2002) suggest that this study provides evidence for the possibility that some of the OCD symptoms observed in eating disorders (particularly anorexia nervosa) might result from malnourishment, rather than being an indication of a 'true' OCD disorder. Pollice, Kaye, Greeno, and Weltzin (1997) also suggest that weight status can impact on general anxiety symptoms. They investigated women with anorexia at different stages of illness and recovery. They found that although anxiety and obsessionality scores remained significantly elevated in women whose weight had been restored (compared to healthy controls), these scores were highest in women who remained underweight.

The evidence reviewed above suggests that the relative chronology between eating disorders and anxiety disorders can vary depending on the anxiety disorder in question. Whilst some anxiety disorders (notably social phobia) frequently precede the onset of the eating disorder, others more commonly occur simultaneously or later (for example GAD). It also appears that whilst OCD features might exist prior to the onset of the eating disorder, they can also occur subsequent to the eating disorder onset (perhaps as a result of malnutrition). However, in interpreting these temporal patterns, it is important to take into account the average age at onset for the disorders (Godart et al., 2002, 2003). For example, whilst social phobia typically starts in childhood, generalised anxiety disorder starts on average at 20 years of age (American Psychiatric Association, 1994). Consequently, Godart et al. (2002, 2003) state that it is unclear whether the relative chronology of anxiety disorders and eating disorders is causative, or whether it merely derives from the natural course of both disorders. Indeed, these average ages at onset are consistent with the finding that social phobia

frequently precedes the eating disorder, whereas generalised eating disorder more typically develops simultaneously or after the eating disorder (Godart et al., 2003).

An alternative explanation for the frequent comorbidity between anxiety and eating disorders is that rather than one causing the other; they both result from a common vulnerability factor (Godart et al., 2003; Wonderlich & Mitchell, 1997). For example, it could be hypothesised that early experiences (e.g., childhood teasing and bullying) could be a risk factor for both disorders (see Fig. 1c). Family studies and multivariate twin studies are two methodologies that can potentially be used to evaluate common cause models such as this (e.g., Lilenfeld, Wonderlich, Riso, Crosby, & Mitchell, 2006). These methodologies will be described further in relation to Fig. 2.

Of course, the three models outlined in Fig. 1 are quite simplistic and suggest that causation is unidirectional. In reality, the connection between eating disorders and anxiety disorders is likely to be more complex and bidirectional. For example, in their transdiagnostic model of the eating disorders, Fairburn et al. (2003) describe how some clients with eating disorders are unable to cope appropriately with intense emotional states such as anxiety. As a result, they engage in "mood modulatory behaviour" (p.517), such as alcohol or drug use, self-harm, or dysfunctional eating behaviours (such as bingeing). However, the persistent use of these behaviours is likely to result in increased negative affect, which further reinforces their use (i.e., according to this model there is likely to be a bidirectional relationship between negative affect and eating behaviours).

Furthermore, Godart et al. (2003) raise the possibility that the relationship between anxiety and eating disorders might be mediated by a mutual relationship with depression (i.e., that depression is responsible for all or some of the association between anxiety and eating disorders). Indeed, Jordan et al. (2003) found that women with anorexia nervosa who were currently depressed were significantly more likely to have social phobia than those without comorbid depression.

Although a detailed investigation of mediator effects is beyond the scope of this review, O'Brien and Vincent (2003) suggest that future research on comorbidities in the eating disorders should involve the explicit testing of both mediator and moderator relationships (the latter being a variable that influences the predictive power of an independent variable). They propose that an understanding of the nature of these relationships has important implications for determining the appropriate focus of treatment. For a thorough description on how to investigate and analyse moderator and mediator effects using multiple regression, see Frazier, Tix, and Barron (2004).

### 2.2.1. Conclusion

Whilst we have outlined the possible temporal relationships between eating disorders and anxiety disorders, the available evidence does not allow clear causal connections to be made. One of the factors preventing this is the absence of prospective studies, which are difficult to undertake due to the low base rates of eating disorders (Serpell et al., 2002). However, despite the uncertainty about any aetiological connections, the research evidence does suggest that the eating disorders are associated with higher rates of anxiety disorders. It is therefore possible to discuss potential mechanisms linking anxiety and eating pathology in individuals who experience both. Such mechanisms will be outlined in the next section of this review.
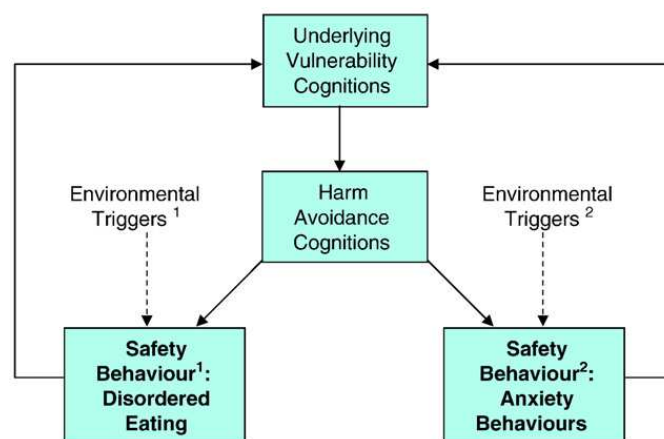


Fig. 2. Safety behaviours as a mechanism of association between anxiety and eating disorders.

## 3. Mechanisms of association

According to cognitive models of anxiety (e.g., Beck & Clark, 1997; Beck et al., 1985), anxiety arises when a situation is viewed as dangerous in some way. Individuals are more susceptible to appraising situations as threatening if they have underlying schemas that represent the world as unsafe and themselves as vulnerable (Beck et al., 1985). The anxiety response itself (which has an effect at the cognitive, affective, physiological, and behavioural levels) can reinforce an individual's view of herself as vulnerable. For example, it may have a negative impact on their functioning, or else be interpreted as a sign of serious physical (or mental) disorder. Furthermore, individuals may inadvertently reinforce their anxiety through the use of safety behaviours and avoidance strategies, which prevent disconfirmation of their belief in catastrophe/vulnerability and may even exacerbate their anxiety symptoms (e.g., Salkovskis, 1991).

A number of behaviours that are commonly associated with eating disorders can also be characterised in terms of safety seeking (e.g., extreme restriction to avoid the fear of massive weight gain). In addition, cognitive avoidance strategies (designed to avoid aversive self awareness and the associated negative affect) have been theoretically linked to dysfunctional eating behaviours. The following sections will therefore describe safety behaviours and cognitive avoidance strategies, and discuss how each could be a mechanism of association between anxiety and eating disorders.

### 3.1. Safety behaviours

Safety behaviours are behaviours that are used by anxious individuals as a way of gaining control and preventing a feared catastrophe (Salkovskis, 1991). However, whilst they may reduce anxiety in the short term, they help to maintain it in the long term. This is because the non-occurrence of the feared outcome is attributed to the use of the safety behaviour, rather than the improbability of the catastrophe (Salkovskis, 1991). Given the high rates of comorbidity between eating disorders and anxiety disorders (described above), women with eating disorders may potentially use a number of non-specific safety behaviours. However, there might also be some safety behaviours that are more particular to the eating disorders, and to the characteristic concerns with loss of control over eating, shape, and weight (e.g., Fairburn & Harrison, 2003). The following sections will therefore outline how cognitive and behavioural rigidity, illness-related behaviours, and body checking might all be examples of safety behaviours in this population.

#### 3.1.1. Cognitive and behavioural rigidity

Women with eating disorders frequently report rigid eating patterns, often with quite ritualistic elements. This may include eating slowly, eating foods in a particular order, or eating only 'safe' foods (with little or no variation). Modifying these eating patterns is often a challenge in therapy, as individuals can be afraid that eating outside of these constraints will result in catastrophic outcomes, such as uncontrolled and rapid weight gain. Rigid daily eating patterns can be evident both in primarily restrictive eaters and in those with bulimic behaviours. In the latter, the individual's intended eating plan may particularly emphasise the need to avoid eating 'forbidden' foods, which are typically foods that are perceived as risky and fattening (e.g., Fairburn, Cooper, & Cooper, 1986). However, it has been hypothesised that attempted abstinence from 'forbidden' foods actually contributes to bingeing. For example, Kales (1990) suggests that when women with eating disorders eat even a small amount of these foods, they often conclude that control over eating has been lost, and hence binge eating ensues. Indeed, using a sample of women with bulimia nervosa, he found that 69% of binges contained foods that these women defined as "forbidden," whereas only 15% of normal eating episodes did.

Of course, rigid and obsessional characteristics in the eating disorders do not always represent safety behaviours. For example, in low weight individuals, rigidity may be a symptom of malnutrition and semi-starvation (Keys et al., 1950; Serpell et al., 2002). However, where rigid behaviours appear to be related to the prevention of a feared catastrophe, then (as in the treatment of anxiety disorders) clients can be encouraged to test out their beliefs by dropping their safety behaviours (e.g., Wells, 1997).

#### 3.1.2. Illness-related behaviours

Gastrointestinal symptoms (such as irritable bowel syndrome and dyspepsia) are common in both anorexia nervosa (e.g., Waldholtz & Andersen, 1990) and bulimia nervosa (e.g., Chami, Andersen, Crowell, Schuster, & Whitehead, 1995). These symptoms are typically considered to be physiological consequences of disordered eating behaviours,

such as restriction, vomiting, and laxative misuse (e.g., Beumont, Russell, & Touyz, 1993). However, as with cognitive and behavioural rigidity, the concept of safety-seeking might be relevant to these behaviours. Bethune, Gompels, and Spickett (1999) cite three case studies in which individuals undertook strict low carbohydrate diets in response to a perceived carbohydrate allergy. They suggest that the most likely explanation for the symptoms was in fact abnormal glucose metabolism secondary to the carbohydrate restriction itself. The avoidance of carbohydrates might therefore be hypothesised to be a form of safety behaviour, designed to prevent a feared consequence (gastrointestinal discomfort). However, as with safety behaviours generally (e.g., Salkovskis, 1991), the low carbohydrate diet apparently maintained and even exacerbated the symptoms experienced. Of course, whilst Bethune et al.s' (1999) study is consistent with this explanation, it does not examine it explicitly. Consequently further research (examining the thoughts and beliefs driving these behaviours) is needed. Nonetheless, where food intolerances and allergies are reported, it may be pertinent for clinicians to consider whether some form of safety behaviour is involved in their aetiology and maintenance. Again, where this applies, the most suitable treatment is likely to involve testing out the consequences of dropping the identified safety behaviour/s.

### 3.1.3. Body checking and avoidance

Body checking is another potential safety behaviour that is known to be common amongst women with eating disorders (e.g., Cooper, Todd, & Wells, 2000; Shafran, Fairburn, Robinson, & Lask, 2004). Fairburn et al. (2003) suggest that body checking stems from the over-evaluation of eating, shape, and weight typically seen in the eating disorders. Individuals who engage in this process examine their body using a variety of methods, including repeated weighing, scrutinizing specific body parts (with or without the aid of a mirror), judging their shape and weight using the fit of clothes, or seeking reassurance from others (Rosen, 1997). Mountford, Haase, and Waller (2006) suggest that a range of beliefs drive such body checking. They found evidence of beliefs that body checking helps the individual maintain control over their eating and weight, beliefs that checking directly decreases anxiety (as a form of reassurance), and beliefs that not checking results in a feared catastrophe (as is typical with safety behaviours). Consequently, Mountford et al. (2006) suggest that it is simplistic to suggest that there is only one explanation for checking behaviours, though it appears that at times it functions as a safety behaviour.

In addition to body checking, individuals may engage in avoidance behaviours. Shafran et al. (2004) found that many individuals alternate between body checking and avoidance, the latter of which involves refusing to be weighed, covering mirrors, and wearing baggy clothes to disguise their shape. As with checking behaviours, avoidance may be related to a range of beliefs, including those related to averting a feared catastrophe. However, where they function as safety behaviours, both checking and avoidance are likely to prevent individuals from disconfirming their fears (e.g., Salkovskis, 1991). This means that anxiety (including that related to eating, shape, and weight concerns) is likely to be maintained in the long term.

### 3.1.4. Summary of the role of safety behaviours

Within the cognitive-behavioural framework for anxiety, individuals with underlying vulnerability cognitions are seen as more likely to appraise situations as threatening. Consequently, they are more likely to use safety behaviours and avoidance strategies. A number of behaviours commonly seen in the eating disorders can also be characterised in terms of safety seeking. In this review, cognitive and behavioural rigidity, illness related behaviours, and body checking (and avoidance) have all been highlighted as potential examples of safety behaviours. Whilst these behaviours might be driven by a variety of beliefs, it appears that averting feared catastrophes (a characteristic of safety behaviours) is one of their possible roles. It has therefore been suggested that safety behaviours (driven by underlying vulnerability cognitions) might be a potential mechanism of association between anxiety and eating disorders. This is illustrated in Fig. 2.

The model depicted in Fig. 2 suggests that underlying vulnerability cognitions result in cognitions relating to harm avoidance. These are the cognitions that drive safety behaviours. However, it is suggested that triggers in the environment determine the way in which these behaviours manifest. Given one set of environmental circumstances, the cognitions may manifest as disordered eating behaviours, but given a different set, they may manifest as part of an anxiety disorder. Safety behaviours are also seen as maintaining the disorder (eating disorder, anxiety disorder, or both) because the non-occurrence of the feared outcome is attributed to the use of the safety behaviour rather than the improbability of the feared event. This would lead to vulnerability cognitions being reinforced and maintained, leading to further use of safety behaviours.

### 3.2. Cognitive avoidance strategies

Cognitive avoidance strategies are another potential mechanism for the association between anxiety disorders and eating disorders. These strategies function either directly or indirectly to reduce awareness of negative cognitions and the associated negative affect (e.g., anxiety or depression). Two main avoidance strategies have been described in the literature: 'cognitive narrowing' and 'blocking'. These strategies have been associated with a number of impulsive behaviours, including self-harm (e.g., Suyemoto, 1998) and substance misuse (e.g., Steele & Josephs, 1988). However, in relation to the eating disorders, they have been particularly associated with bingeing and vomiting. In the following section, we begin by outlining Heatherton and Baumeister's (1991) escape from awareness model, which relates the process of cognitive narrowing to binge eating. We then go on to describe the blocking models of Lacey (1986) and Root and Fallon (1989), and discuss how this strategy might be relevant to bulimic behaviours. The construct of dissociation will then be discussed in relation to these cognitive strategies.

It should be noted that because the eating disorder models for cognitive avoidance have been explicitly developed to account for binge eating, they do not relate to restrictive attitudes and behaviours. However, it may be that synonymous processes are associated with restrictive eating pathologies. This is therefore potentially an area that would benefit from further research and theory development.

#### 3.2.1. Cognitive narrowing

According to Heatherton and Baumeister's (1991) escape from awareness model, bingeing occurs as a consequence of 'cognitive narrowing', an intentional strategy employed to escape from aversive self-awareness and the associated negative affect. In support of this model, Heatherton and Baumeister (1991) cite evidence that bulimics have unrealistically high standards or ideals, both in relation to bodily thinness (e.g., Powers, Schulman, Gleghorn, & Prange, 1987), and in relation to performance and achievement (e.g., Butterfield & Leclair, 1988). Heatherton and Baumeister (1991) suggest that these women experience aversive self-awareness when they perceive that they have failed to live up to their demanding ideals. In order to escape from the associated emotional distress (e.g., anxiety and depression), it is hypothesised that the cognitive strategy of 'narrowing' is employed. This strategy involves focusing attention on the immediate and present stimulus environment and engaging in low-level or concrete thinking. Consequently, self-awareness remains at a relatively low level, and negative perceptions of the self are not processed in any depth (so negative emotions can be temporarily avoided). However, a further result of this shift to low levels of thinking is that higher-level cognitive functions (such as reasoning and inhibition) are lost. To illustrate the hypothesised effect on inhibitions, Heatherton and Baumeister give the example of cheating in an exam. They suggest that at a low level of meaning, cheating can be reduced to making marks on a page (a far more tolerable act). Accordingly, internal obstacles to this action are removed, making the individual more willing to carry it out. In a similar way, cognitive narrowing is said to result in restraints against eating being reduced or removed from awareness, so that bingeing occurs despite the binge-eater's abhorrence of the behaviour.

Indirect support for the connection between binge eating and reduced self-awareness comes from the finding that instructions to monitor eating appear to enhance dietary restraint and reduce the likelihood of disinhibited eating amongst dieters (e.g., Pecsok & Fremouw, 1988). In addition, alcohol consumption (which is known to reduce self-awareness and behavioural inhibition) often precedes bingeing (e.g., Abraham & Beumont, 1982). There is also some evidence to support the hypothesised change to lower-level thinking accompanying cognitive narrowing. For example, dichotomous cognitions have been found to be prominent among both bulimics and subclinical binge eaters prior to binge episodes (e.g., Lingswiler, Crowther, & Stephens, 1989). However, Heatherton and Baumeister (1991) acknowledge that there is some ambiguity in the evidence. Indeed, while binge eating has often been associated with reductions in anxiety (e.g., Fairburn et al., 1986), Heatherton and Baumeister suggest that it is not clear whether binge eating results from this cognitive narrowing, or whether bingeing is itself able to facilitate the avoidance of negative cognitions and emotions. The latter would perhaps be more consistent with the 'blocking' models described by Lacey (1986) and by Root and Fallon (1989).

#### 3.2.2. Blocking

In contrast to the cognitive narrowing model, blocking models depict binge eating as a direct method for reducing aversive self-awareness. For example, Lacey (1986) proposes that one of the functions of binge eating is to moderate or 'block' negative emotional states. He highlights a number of predisposing factors that might lead people to use eating in

this way, including difficult family and peer relationships, a reliance on external standards for judging self-worth, and family preoccupation with appearance, weight, and eating. Lacey (1986) suggests that these kinds of early experiences typically lead to inadequate adult coping skills, including impaired interpersonal relationships and feelings of ineffectiveness and lack of control. In this context, negative life events such as sexual conflict, changes in life circumstances, or loss, result in excessive self-reflection and self-criticism. Because their attention is already focused on food (as a result of both familial and cultural pressures for thinness), the manipulation of food becomes a convenient defence mechanism. He proposes that in the light of external stressors, these women initially instigate dieting to try to increase their self-worth by altering their body shape. However, as a result of this restriction they become more vulnerable to binge eating, and consequently to vomiting. Despite finding these behaviours distasteful, bulimics may continue to engage in them because they discover that they can be used both to fill an emotional void (e.g., associated with boredom or loneliness) and to reduce emotional arousal (such as anxiety or distress). Indeed, women with bulimia nervosa have reported that bingeing and vomiting can be used to "anaesthetize" negative emotional states (Vanderlinden & Vandereycken, 1997). Given their lack of alternative coping skills, Lacey (1986) suggests that bulimic women may therefore come to rely on bingeing and vomiting as ways of moderating their emotions.

Root and Fallon's (1989) model has many similarities to that proposed by Lacey (1986), though it primarily considers bulimic individuals who are victims of abuse. Amongst other things, they suggest that bingeing is used as a method of dealing with stress and tension, since these individuals lack sufficient alternative coping strategies. They also suggest that bingeing can be used to temporarily anaesthetize powerful negative emotions and block out distressing memories (particularly of abuse). Thus both blocking models see bingeing as allowing the individual temporary relief from aversive self-awareness, in the context of deficient alternative coping strategies and feelings of lack of control.

### 3.2.3. Links with dissociation

Both cognitive narrowing and blocking are potentially related to the construct of dissociation. Dissociation is described in DSM-IV as: "a disruption in the usually integrated functions of consciousness, memory, identity, or perception of the environment" (American Psychiatric Association, 1994). For example, Schulman (1991) compared the experience of bingeing to dissociation, and suggested that both function to block out painful emotions and experiences. Binge eating and dissociation might therefore be alternative means of achieving the same end (i.e., emotional blocking). On the other hand, Demitrack, Putnam, Brewerton, Brandt, and Gold (1990) propose that dissociative experiences lead to a narrowing of cognitive focus and the exclusion of important (often conflicting) information from consciousness. Meyer, Waller, and Waters (1998) suggest that this style of thinking can lead to behaviours that would usually be inhibited (e.g., eating) becoming disinhibited. As they point out, this has obvious overlaps with the 'escape from awareness' model proposed by Heatherton and Baumeister (1991). Indeed, Heatherton and Baumeister themselves make the connection between cognitive narrowing and dissociation. They cite research from Abraham and Beumont (1982), which found that 75% of bulimics reported feelings of depersonalization and derealization (i.e., aspects of dissociation) during binges. In addition, 72% of the women in this study reported experiencing a reduction in negative mood states as a result of the binge. Taken together, these findings suggest that dissociation might be an important construct to consider when investigating the processes of 'blocking' and 'cognitive narrowing' described above.

### 3.2.4. Conclusion

In summary, binge eating may occur as a result of the disinhibition accompanying cognitive narrowing (a strategy employed to escape from aversive self-awareness and the associated negative affect). Conversely, bingeing may be more directly involved in the reduction of self-awareness by providing a blocking process. Both these strategies may have connections with the construct of dissociation. Indeed, whilst these models describe different cognitive processes, they are not incompatible with one another. In fact, McManus and Waller (1995) suggest that cognitive narrowing might be particularly important in the initial onset of binge eating (along with dietary restriction), but once the blocking effects of bingeing are discovered, this process might be more important in explaining its continued use. Of course, there may also be some individual differences in the relative importance of these processes in driving binge eating. Fig. 3 demonstrates how these cognitive avoidance strategies might potentially connect eating disorders and anxiety disorders in the context of specific cognitions.

In this model, disordered eating and anxiety behaviours are each seen as being associated with specific underlying cognitions (i.e., related to eating, weight, and shape or to immediate threat cognitions). The use of cognitive narrowing
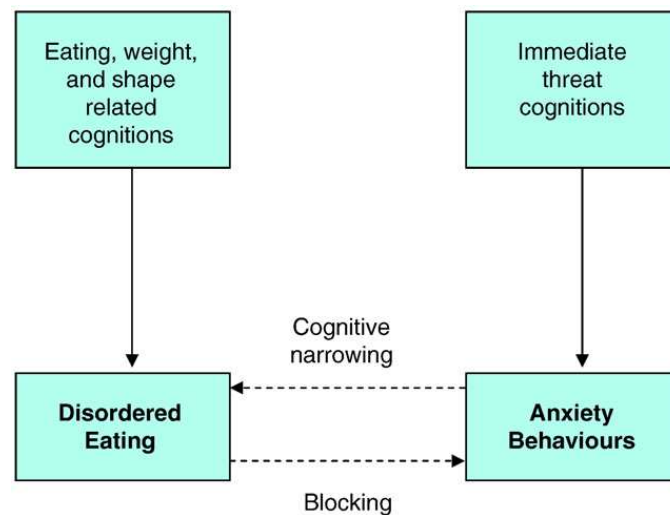
Fig. 3. Cognitive avoidance strategies as a mechanism of association between anxiety and eating disorders.

is seen as being driven by attempts to reduce the experience of anxiety, and disordered eating follows as a consequence (i.e., as a result of reduced inhibitions). Conversely, blocking starts with disordered eating, since these behaviours are directly used to block out the experience of anxiety. Fairburn et al. (2003) noted that the use of various blocking behaviours (e.g., substance misuse, self-harm, or bingeing) is likely to be reinforced by the reduction in anxiety they bring about. However, the underlying cognitions (i.e., related to eating, weight, and shape or to vulnerability to harm) are not actually challenged. As such, it can be hypothesised that where these cognitive avoidance strategies are used, the connection between anxiety and disordered eating will be maintained in the long term.

Of course, whilst the models for cognitive narrowing and blocking have been developed to account for bulimic eating behaviours (i.e., bingeing and vomiting), it is also possible that similar processes apply to restriction. For example, in a sample of women with anorexia nervosa (restricting subtype), Hinrichsen et al. (2003) found that higher levels of social anxiety were associated with higher levels of dissociation (a construct which, as described above, has been related to cognitive narrowing and blocking). Investigating how these cognitive strategies might apply to restrictive eating behaviours might therefore be an important area for further research and theory development.

## 4. Aetiological determinants of the eating-anxiety link

Inherent in these models is the principle that having an anxiety disorder potentially increases the likelihood of having a comorbid eating disorder (and vice versa). This is because both are hypothesised to be related to the same aetiological factors (underlying harm avoidance cognitions). As indicated in relation to Fig. 1c, family study designs are one way to test for the presence of common aetiological factors (see Lilenfeld et al., 2006 for a detailed description of family studies and related methodologies). To test for shared familial risk factors, a four-group family design would ideally be employed (eating disorder probands with anxiety disorders, eating disorder probands without anxiety disorders, non-eating disorder probands with anxiety disorders, and non-eating disorder probands without anxiety disorders — i.e., non-clinical controls). Assuming that eating disorders and anxiety disorders share a common aetiology, elevated rates of both disorders would be expected among the relatives of the first three groups (compared to the non-clinical controls).

Unfortunately, to our knowledge, a study using these four groups (with balanced samples in each) has yet to be completed. However, there are studies that have investigated the patterns of anxiety disorders amongst relatives of eating disorder probands. Results from these studies currently yield mixed results. For example, Lilenfeld et al. (1998) found that compared to controls, relatives of both anorexia and bulimia nervosa probands had significantly increased rates of GAD and panic disorder. Furthermore, relatives of anorexia nervosa probands had significantly increased rates of social phobia and OCD. However, although they found "some suggestion" of shared familial risk between social phobia and anorexia nervosa, they report finding no evidence of shared causation between OCD and eating disorders (i.e., the rate of OCD was only elevated amongst the relatives of eating disorder probands who themselves had OCD). However, Lilenfeld et al. concede that their relatively small sample size (particularly in the anorexia nervosa group) may have limited the power of their analyses.

Using a similar study design, Wade, Bulik, Prescott, and Kendler (2004) found evidence of shared familial risk factors between GAD and bulimia nervosa. However, they found this to be sex-specific (i.e., the relationship between bulimia nervosa in one sibling and GAD in the other was only evident in female-male twin pairs). They indicate that, using this design, they were unable to comment on the nature of shared risk factors (i.e., whether they are genetic, environmental, or a complex combination of both).

Multivariate twin studies (which compare the patterns of comorbidity between monozygotic and dizygotic twins) are another way in which common cause models can be investigated. Using this method, Kendler et al. (1995) found evidence for shared genetic vulnerability between bulimia nervosa and phobias and panic disorder (but not GAD). This type of twin study can also be used to estimate the extent to which shared and individual-specific environmental factors affect the risk for particular disorders. However, in order to identify specific factors involved in the aetiology of disorders, sophisticated, longitudinal, prospective research (which is both costly and time consuming) is necessary (Lilenfeld et al., 2006).

## 5. Relationship to models of anxiety

Heatherton and Baumeister (1991), Lacey (1986), and Root and Fallon (1989) all assert that bulimic behaviours function either directly or indirectly to reduce awareness of aversive cognitions and emotions. It therefore seems pertinent to consider how women with eating disorders might process these cognitions and emotions. The information processing model of anxiety proposed by Beck and Clark (1997) has recently been related to women with bulimia (e.g., Ainsworth, Waller, & Kennedy, 2002). As such, we will begin by outlining the three stages of this model. We then discuss the evidence relating to attentional bias pertaining to both anxiety and eating disorders, before moving on to consider the concept of schema avoidance.

### 5.1. Beck and Clark's information processing model of anxiety

Beck and Clark (1997) hypothesize that the cognitive-affective-physiological-behavioural pattern of anxiety arises from a three-stage information processing sequence. Moreover, they state that it is the tendency of this system to attribute threat inappropriately to innocuous stimuli that is at the heart of anxiety disorders.

According to this model, the initial stage of threat processing involves the 'orienting mode' (Beck, 1996). This stage generally occurs outside of conscious awareness, and involves the rapid and involuntary processing of information. The function of this mode is to identify stimuli and assign attentional priority. Based on the findings of subliminal studies, Mathews and Macleod (1994) suggest that this pre-attentive analysis of meaning may only classify stimuli as threat-related or not.

The second stage of processing involves the activation of a 'primal mode', a cluster of schemas designed to fulfill basic evolutionary goals (e.g., safety, sociability, procreation). As a result of this focus on survival, primal modes tend to be inflexible and rigid. Once they are activated, these modes take up much of the attentional resource, thus reducing the capacity for more constructive or reflective modes of thinking. However, whilst processing at this stage can be rapid and involuntary (and can thus occur outside of conscious awareness), individuals will be aware of the products of the primary threat appraisal. One consequence of this process is the narrowing of cognitive processing, which leads to biases and inaccuracies in thinking (such as rigid and dichotomous thinking, with an intolerance for ambiguity). In addition, negative automatic thoughts (related to threat and danger) occur at this stage of processing. The cognitive avoidance strategies outlined in Fig. 3 would be examples of strategies employed at this stage of processing.

The final stage of processing is 'secondary elaboration'. Information processing at this stage is typically slow, effortful, and schema-driven. However, the primal mode remains active. Schemas representing the individual's current concerns and personal issues are activated at this stage, and threat information is evaluated within this context. In addition, individuals begin to evaluate their own resources for coping using the 'metacognitive mode' (a process that allows them to think about thinking). Beck and Clark (1997) suggest three possible outcomes of an individual's reflection (or failure of reflection) at this stage. The first is that anxiety increases because a more realistic appraisal of the situation is blocked and the primal mode continues to be dominant. Second, anxiety can decrease if the individual downgrades the likelihood or severity of threat and upgrades their ability to cope. Finally, anxiety can be reduced if the individual engages in defensive behaviours prompted by the primal mode, for example escape or avoidance. Ainsworth et al. (2002) suggest that avoidance is likely to occur both at behavioural and cognitive levels. Therefore, cognitive

narrowing and blocking (as outlined in Fig. 3), along with dissociation can all be considered examples of cognitive avoidance strategies. In contrast, safety behaviours (as outlined in Fig. 2) can be considered to be behavioural forms of avoidance (Beck & Clark, 1997).

### 5.1.1. Attentional bias

Beck and Clark's (1997) model asserts that the function of the early stages of processing is to assign attentional or processing priority to incoming stimuli. As such, attentional biases are potentially important in understanding anxiety and other pathologies. Attentional bias research has frequently utilised the Stroop colour naming task and the visual dot probe task. The Stroop colour naming task involves naming the colour of the ink in which a word is printed, whilst ignoring the meaning of the word. Research studies using this technique have consistently demonstrated that anxious individuals take longer to colour-name emotionally threatening words compared to neutral words, suggesting that participants have difficulty ignoring the emotional content of the stimuli (see Mathews & Macleod, 1994). In contrast, the dot probe task involves the simultaneous presentation of two words (one below the other) onto a computer screen. The words disappear, and a dot probe appears in the spatial location of one of the words. Participants are required to press a button when the dot is detected, and the response latency is recorded. Previous research has found speeded detection latencies for probes replacing negative (compared to neutral) words. This suggests that anxious individuals have an attentional bias towards emotionally negative stimuli (see Mathews & Macleod, 1994).

In their review of information processing biases in eating disorders, Lee and Shafran (2004) conclude that Stroop research has generally indicated that women with eating disorders take longer to colour-name eating-, shape-, and weight-related words (suggesting they have difficulty ignoring the emotional content of these words). However, they note that a number of criticisms have been made against the Stroop task, including the fact that most research has focused on negatively valenced words. In light of these criticisms, Rieger et al. (1998) used a visual dot probe task consisting of both positively and negatively valenced body-, food-, and shape-related words. They found that women with eating disorders tended to direct attention away from words connoting thinness, but directed their attention towards words consistent with fatness. This was interpreted as suggesting that women with eating disorders attend to information consistent with their fear of weight gain, but ignore information that counters this fear (similar to the construct of 'schema avoidance' — see below). Moreover, there is evidence of attentional biases unrelated to eating, shape, and weight amongst women with eating disorders. For example, McManus, Waller, and Chadwick (1996) used the Stroop task to investigate the impact of five different forms of threat. They found that whilst women with bulimia nervosa were significantly slower to colour-name all forms of threat, this interference effect was particularly pronounced for self-generated ego threats (i.e., self criticism). They report that these results provide indirect support for Heatherton and Baumeister's (1991) model, which asserts that aversive self-awareness can trigger bulimic behaviours. This finding can therefore be taken as indirect evidence for the model outlined in Fig. 3.

### 5.1.2. Schema avoidance

Ainsworth et al. (2002) point out that according to Beck and Clark's (1997) model, the avoidance (both cognitive and behavioural) that occurs in the latter stages of information processing is important in the maintenance of anxiety. Ainsworth and colleagues further suggest that in order to understand this avoidance, it is helpful to consider the schema-focused cognitive-behavioural model proposed by Young (1994). Young describes schemas as "extremely stable and enduring themes." He suggests that maladaptive schemas are developed as a result of negative experiences during childhood, and once they are formed, they influence the way in which events and experiences are interpreted. Young suggests that the enduring nature of these schemas can be explained in terms of three schema processes — schema maintenance; schema compensation; and schema avoidance. Schema maintenance involves accentuating information that is consistent with the schema and minimising information that is inconsistent. As a result, the maladaptive schema is reinforced. Conversely, schema compensation involves overcompensating for maladaptive schemas (i.e., acting in ways that are opposite to that predicted by the schema). However, this process leads to maintenance rather than the removal of the underlying schema. Finally, schema avoidance refers to behaviours and cognitive strategies designed to reduce the activation of schemas and the associated negative affect. Again, this does not result in any challenges being made to the schema content.

Ainsworth et al. (2002) suggest that cognitive narrowing (Heatherton and Baumeister, 1991), blocking (Lacey, 1986; Root & Fallon, 1989) and dissociation are all examples of schema avoidance strategies. Thus, bulimic behaviours such as bingeing might be associated with early maladaptive schemas (i.e., they may occur either as a

consequence of attempts to avoid triggering the schemas, or else are utilised to block the intolerable cognitions and emotions resulting from the schemas).

### 5.2. Conclusion

The initial two stages of anxiety processing involve assigning attentional priority to incoming information, and making an initial (and rapid) appraisal of threat. Research has demonstrated that anxious individuals have an attentional bias towards threatening information. In addition, women with eating disorders have been found to have an attentional bias towards words consistent with their fear of weight gain, as well as towards ego-threat words.

The final processing stage in anxiety is more reflective and consequently involves a more realistic appraisal of the situation. However, this reflective process can be blocked by the activation of more primal responses such as cognitive or behavioural avoidance. It has been suggested that the concept of schema avoidance is helpful to the understanding of cognitive avoidance strategies. Specifically, it is suggested that these strategies might function to avoid triggering maladaptive schemas and the associated negative affect. However, whilst employing cognitive avoidance strategies might allow a temporary reprieve from negative affect, they ultimately do nothing to diminish the underlying maladaptive schemas.

## 6. Final model of the association between anxiety and the eating disorders

Fig. 4 is an amalgamation of Figs. 2 and 3. It also indicates the hypothesised childhood origins of underlying vulnerability cognitions (which exist at the schema level). The key elements are: the schema-level *underlying*
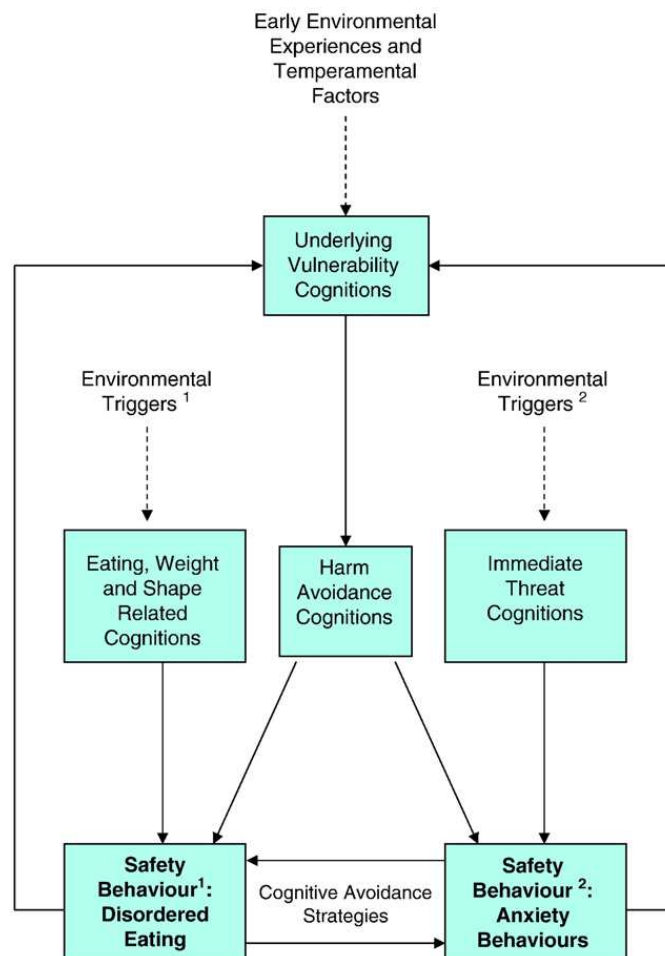


Fig. 4. Model of the potential mechanisms connecting anxiety and eating disorders.

*vulnerability cognitions*, driven largely by early experiences and individual temperament; the resultant *harm avoidance cognitions*, which are not pathological in themselves, but which constitute a vulnerability factor in the presence of environmental triggers; the *cognitions relating to eating, weight, and shape* that are driven by relevant environmental triggers (e.g., teasing about appearance); and the *cognitions relating to immediate threat* (e.g., an interpersonal threat). This is a shared transmission model, where the harm avoidance cognitions drive both the eating and the anxiety symptoms (in the context of the more immediately accessible vulnerability or eating, weight, and shape cognitions), rather than a model where anxiety *per se* drives the eating pathology or vice versa. Thus, the harm avoidance cognitions are not specific to either the eating or the anxiety, but can contribute to either or both sets of symptomatic behaviours. According to the model, the mechanisms of association between eating disorders and anxiety disorders can include safety behaviours, and also cognitive avoidance strategies.

In this model, triggers in the environment determine the form that safety behaviours take. Whilst safety behaviours may initially result in a reduction in negative affect, they ultimately reinforce the underlying vulnerability cognitions that drive them. In addition, since cognitive avoidance strategies (i.e., cognitive narrowing and blocking) can be utilised to avoid triggering underlying vulnerability cognitions and resultant negative affect, their use is ultimately self-reinforcing. Because the individual avoids experiencing negative emotions, they do not practice distress tolerance. Moreover, the harm avoidance cognitions underlying the use of these strategies (i.e., beliefs that negative emotions cannot be tolerated) are not challenged.

The model suggests that eating disorders and anxiety disorders share common aetiological factors. As a result, once the vicious cycles involved in either disorder have been activated, the individual's susceptibility for symptoms related to the other disorder (both categorically and dimensionally) is also increased. This assertion is consistent with research from Kaye et al. (2004). They found that anxiety scores on self-report assessments tended to be highest amongst individuals who had both a lifetime anxiety diagnosis and a current eating disorder. Anxiety scores were somewhat lower in those who had either a current eating disorder or a lifetime anxiety disorder diagnosis. However, they also found that self-rated anxiety was higher in individuals who had recovered from an eating disorder (compared to controls) even if these individuals had never met diagnostic criteria for an anxiety disorder. This suggests that anxiety levels are higher in individuals with eating disorders, even when DSM-IV diagnostic criteria are not met.

In addition, Kaye et al. (2004) report that individuals whose eating disorder was remitting were less likely to have a history of anxiety disorders compared to individuals with current eating disorder symptoms. Although they acknowledged that this could reflect a bias in recall, they interpret this finding as potentially indicating that not having an anxiety disorder is moderately associated with recovery from an eating disorder. This might suggest indirect support for the idea outlined above — that once the vicious cycles involved in either disorder have been activated, susceptibility to symptoms related to the other is also increased.

Finally, Keel, Klump, Miller, McGue, and Iacono (2005) used a discordant monozygotic twin design to investigate the possibility of shared transmission between eating disorders and anxiety disorders. They found that in twins not concordant for eating pathology, the non-eating-disordered twin was more than twice as likely to suffer from an anxiety disorder compared to controls. This increased risk was not due to anxiety in the eating-disordered twin — thereby suggesting a common underlying diathesis between anxiety and eating disorders, and lending support to the model outlined in Fig. 4.

The model presented in this paper could potentially be further developed to account for factors such as the attentional biases for threat seen in both eating disorders and anxiety. The mechanisms accounting for variations in comorbidity between eating disorders and particular anxiety disorders could also be investigated further.

## 7. Implications for the treatment of the eating disorders

A number of authors have highlighted the importance of considering comorbidity in the formulation and treatment of eating disorders (e.g., Braun et al., 1994; O'Brien & Vincent, 2003; Serpell et al., 2002). However, little appears to have been written about the way in which anxiety (or indeed any other) comorbidity affects the focus of treatment. The model presented in this paper suggests that where there is comorbidity, eating disorders and anxiety disorders may share common underlying factors. As such, effective treatment should involve targeting the underlying vulnerability cognitions, the harm avoidance cognitions, and the safety behaviours. If these are not dealt with effectively, treatment is likely to be less successful and the risk of relapse (for either disorder) is likely to remain high. Indeed, Weltzin et al. (1995) report that in their clinical experience, significant anxiety reduces compliance with attempts to normalise eating habits, and often leads to drop-out from treatment.

Within a cognitive-behavioural framework, treatment for safety behaviours (whether associated with an anxiety disorder or eating disorder) will involve using a range of behavioural experiments (e.g., Wells, 1997). The aim of these experiments is to test out the individual's catastrophic predictions regarding their non-use of safety behaviours. In relation to underlying vulnerability cognitions, the aim of treatment is to encourage individuals to elaborate and reflect upon their threat-related cognitions (Beck & Clark, 1997). Beck and Clark suggest that this can be achieved through a combination of behavioural exercises and verbal hypothesis testing.

It is also important to recognise the powerful affect modulation function of cognitive avoidance strategies. In 'blocking', the individual uses binge eating as a way of reducing intolerable affect. In contrast, in 'cognitive narrowing', binge eating occurs as a consequence of attempts to avoid negative affect. This suggests that where either of these strategies is used, the underlying emotions need to be targeted in order to reduce binge eating. In the first instance, this might involve increasing the individual's capacity for distress tolerance. Secondly, therapy might involve dealing with cognitions and their link with emotions. For example, beliefs regarding the consequences of experiencing negative emotions (such as anxiety or anger) can be investigated and tested.

## 8. Suggestions for future research

The comorbidity research reviewed has focused on prevalence rates of anxiety disorders amongst different eating disorder diagnoses. Given the recent emphasis on transdiagnostic approaches to the eating disorders (Fairburn et al., 2003), it might therefore be helpful for comorbidity research explicitly to investigate the various presentations of anxiety associated with particular eating behaviours (irrespective of diagnosis). It is also notable, to date, that the constructs of cognitive narrowing and blocking have not been applied to restrictive pathologies. Understanding more about how these (or similar) strategies might apply to the range of eating disorder presentations would be beneficial in the formulation and treatment of cases.

Further research might also look at testing and extending the model presented in this review. For example, the model could be extended to account for factors such as attentional biases for threat (seen in both anxiety disorders and eating disorders). It could also be modified to account for the variations in comorbidity between eating disorders and particular anxiety disorders.

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