Anxiety Disorders and Eating Disorders: A review of their relationship

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Historically, the comparative phenomenology of anxiety and eating disorders has been examined from three perspectives. First, parallels in symptomatology such as the similarity between binging and purging in bulimia or eating-related rituals in anorexia and compulsive behaviour in obsessive compulsive disorder, or between the fear of negative evaluation in both eating disorders and social phobia, have suggested to some a similar underlying psychopathology with symptom expression in different domains. Second, patterns of comorbidity and family history have been studied in both clinical and epidemiological samples of women with eating disorders and have suggested elevated rates of anxiety disorders in both eating disordered probands and their family members. Third, both behavioural and pharmacological treatments known to be effective in the anxiety disorders have been applied successfully to the treatment of eating disorders (e.g. exposure with response prevention and fluoxetine for bulimia). In the absence of prospective studies, these three lines of evidence converge to suggest a modest association between eating and anxiety disorders. The present paper reviews and integrates evidence from the above three perspectives in an attempt to determine the nature of the relation between anxiety and eating disorders.

The comparative phenomenology of anxiety and eating disorders has been examined from several perspectives. First, parallels in symptomatology between the eating and anxiety disorders have suggested a similar underlying psychopathology and possibly pathophysiology. Second, patterns of comorbidity in both clinical and epidemiologic samples of women with eating disorders reveal elevated rates of anxiety disorders in individuals with eating disorders. Similarly, eating disorders are overrepresented in clinical samples of individuals with primary anxiety disorders. Third, both behavioural and pharmacological treatments known to be effective in the anxiety disorders have been applied successfully to the treatment of eating disorders (e.g. fluoxetine and exposure with response prevention for bulimia). The present paper reviews and integrates evidence from these three perspectives in an attempt to determine the nature of the relation between anxiety and eating disorders.

Types of Anxiety in Eating Disorders

Anxiety has been implicated as a central factor in the pathogenesis and maintenance of anorexia and bulimia nervosa. In addition to the core observable anxieties about weight and food, two aspects of anxiety, namely fear of negative evaluation and obsessionality, have been addressed both theoretically and empirically as potentially defining features of the eating disorders.

Fears or anxieties about weight gain are pathognomonic of anorexia nervosa. Crisp coined the term "weight phobia" to describe the almost phobic avoidance of high calorie foods and body weight seen in women with anorexia nervosa (Crisp, 1967). The diagnostic criteria for anorexia nervosa highlight "an intense fear of gaining weight or becoming fat, even though underweight" and in bulimia nervosa"self-evaluation is unduly influenced by body shape and weight" (American Psychiatric Association, 1994). These core features have been conceptualised alternately as a function of fears of negative evaluation by the self and by others or as manifestations of obsessional traits.

Fear of negative evaluation.

In both anorexia and bulimia nervosa, women are fearful of weight gain and intensely self-critical; however, this negative self-evaluation also generalises to fears of negative evaluation by others. Not only do these women fear negative evaluations about their appearance, but their social anxieties generalise to settings unrelated to food and eating as well.

Early studies examining the construct of social anxiety in women with anorexia nervosa suggested that between 33 and 80% of subjects were either socially anxious or shy (King, 1963; Morgan & Russell, 1975; Rothenberg, 1988; Rowland, 1970; Warren, 1968). The majority of these studies did not utilise either formal diagnostic criteria or

psychometrically sound instruments. Crisp and Baht (1982) assessed several dimensions of anxiety in individuals with the restricting type of anorexia nervosa (i.e., no binging and purging) and anorectics who also binged and purged. They found higher levels of anxiety in the women who binged and purged, higher levels of social anxiety in women who purged, and lower levels of anxiety associated with lower weight in women with anorexia. They hypothesised that the relation between low weight and lessened anxiety reflected the sense of control attained from being at low weight (and not having to face the phobic stimulus, namely fat).

More recent studies utilising standardised instruments to measure the construct of social anxiety have clarified the nature of social anxiety in women with eating disorders. For example, Bulik, Beidel, Duchmann, Weltzin and Kaye (1991) compared the responses of 23 women with anorexia nervosa, 54 women with bulimia nervosa, 50 college undergraduates, and 43 women with social phobia on the Social Phobia and Anxiety Inventory (Turner, Beidel, Dancu, & Stanley, 1989). The women with eating disorders endorsed clinically significant social fears that were equal in intensity to women with social phobia. The social fears reported in women with anorexia and bulimia were not limited to social fears of eating and drinking in public but extended to more generalised social situations. Results of this investigation suggested that not only do anorectic and bulimic women score high on social phobia scales, but that their general non-social anxiety is also higher than either women with social phobia or controls.

Striegel-Moore, Silberstein, & Rodin (1993) compared responses of women with bulimia nervosa to a sample of women with high scores on the Eating Attitudes Test (Garner, Olmsted, Bohr, & Garfinkel, 1982) and to controls

Table 1: Studies of Obsessive-Compulsive Traits in Women with Anorexia and Bulimia Nervosa

Authors & Date	Subjects	Diagnostic Criteria & Design	Instruments	Results and Conclusions				
Smart et al.,1976	AN (22) Obessionals Healthy controls	Russell Criteria In- and Outpatients	LOI		*Symptom*	*Trait*	*Resistance*	*Interference*
				AN	21.0	9.1	17.2	20.0
				Obs	33.3	11.0	36.0	36.7
				Cont	11.4	5.1	7.3	3.8
				AN significantly > controls AN State Trait Resistance < obsessionals AN Trait = obsessionals				
Strober, 1980	AN (22) MAD (22) PD (22)	Feighner criteria In patients	LOI		*Symptom*	*Trait*	*Resistance*	*Interference*
				AN	16.6	9.9	18.4	14.0
				MAD	17.7	6.0	12.2	9.2
				PD	10.4	5.4	7.0	3.9
				AN vs MAD	<.05	<.001	ns	ns
				AN vs PD	<.02	<.001	<.01	<.01
Solyom et al., 1982	AN (15) OCD (14)	"Standard Criteria" DSM III Consecutive referrals	LOI		*Symptom*	*Trait*	*Resistance*	*Interference*
				AN	19.3	9.1	27.4	14.9
				OC	27.7	10.9	32.0	35.8
			p		ns	ns	ns	ns
Channon et al., 1985	AN (45)	Diagnostic criteria unknown Inpatient- admission - discharge 1 yr follow up	MOC		*Admission*	*D/C*	*F.U.*	*p*
				AN	9.9	7.7	7.9	ns
				Scores on MOC similar to non-neurotic controls				
Bulik et al., 1991	BN (20) OCD (20)	DSM III-R BN inpatients OCD in and outpatients	MMPI SCI-90-R BDI	No difference between BN and OCD women on anxiety or OC measures. High frequency of 2-7 elevations on MMPI in both groups.				
Fahy, et al., 1990	AN (29) BN (44) BN-hx-AN (23)	DAM-III-R Outpatients	MOC	No difference in OC scores across ED subgroups or in comparison to healthy controls.				
Hsu, 1992	AN (16)	DSM-III-R Follow-up study	MOC		*Recovered*	*Nonrecovered*	*p*	
				AN	3.8 + 3.5	9.7 + 5.9	<.03	
Kaye, et al., 1992	AN (19) Healthy controls (14)	DSM-III-R In- and outpatients	Y-BOCS	AN Controls	22 + 5 3 + 3 No difference between bulimic anorectics and restrainers		p < .001	

on the Self-Consciousness Scale (Fenigstein, Scheier, & Buss, 1975). Women with bulimia nervosa scored significantly higher on subscales of public self-consciousness and social anxiety than controls. The authors concluded that social self-concerns are integral to the eating disorders; however, their design did not include a second pathological control group to determine if the effect was specific to bulimia nervosa or a nonspecific effect of psychopathology.

In sum, studies examining social anxiety from a dimensional perspective have found high levels of both social and non-social anxiety symptoms in women with anorexia and bulimia nervosa.

Obsessionality

The second manner in which anxiety has been implicated in the core behaviours in anorexia and bulimia nervosa highlights the obsessional and compulsive nature of the disorders.

Anorexia nervosa. Anorexia nervosa is marked by obsessional preoccupation with food and weight, ritualistic eating behaviours, obsessive exercise and other behaviours that parallel those seen in individuals with obsessive-compulsive disorder. In 1939, Palmer and Jones first suggested that anorexia nervosa may be a manifestation of obsessive-compulsive disorder. Later, DuBois (1949) attempted to re-name the disorder "compulsion neurosis with cachexia." Rothenberg (1986) called eating disorders the "modern obsessive compulsive syndrome" and provided some evidence that, together with disturbance in affect, obsessive-compulsive symptoms were the most frequently reported symptoms or disorders in studies of women with anorexia nervosa.

Several investigations have examined the nature of obsessional thoughts and behaviour in women with anorexia in comparison to women with OCD. These studies are summarised in Table 1. The vast majority of studies have assessed obsessional symptomatology during the acute phase of the illness using either clinical observation or instruments such as the Leyton Obsessionality Inventory (LOI) (Bulik, Beidel, Duchmann, Kaye, & Weltzin, 1992; Smart, Beumont, & George, 1976; Solyom, Freeman, & Miles, 1982; Strober, 1980), the Maudsley Obsessive-Compulsive Inventory (MOC) (Channon & DeSilva, 1985; Fahy, 1991; Hsu, Crisp, & Callender, 1992; Weiss & Ebert, 1987) or the Yale-Brown Obsessive Compulsive Scale (Y-BOCS) (Kaye, Weltzin, Hsu, Bulik, McConaha, & Sobkiewicz, 1992).

Overall, studies using the LOI suggest high scores on obsessionality in women with anorexia during the acute phase of the illness. Strober (1980) suggested that although obsessional symptoms decreased after weight restoration, obsessional trait scores remained elevated. Studies utilising the MOC have been less consistent with some authors reporting increases in MOC scores in patients with current anorexia (Channon & DeSilva, 1985; Hsu, et al., 1992) and others reporting no elevation over healthy controls (Fahy, 1991; Weiss & Ebert, 1987).

Findings have varied according to instruments used. Overall, retrospective clinical information suggests that women with anorexia nervosa have premorbid obsessional traits which become exacerbated during the acute phase of the illness, possibly secondary to starvation. Outcome studies indicate that those who remain ill, retain high obsessionality scores, whereas those who recover from anorexia nervosa approach scores of healthy controls (Hsu, et al.,1992).

Bulimia nervosa. The nature of the obsessionality and compulsive behaviour in bulimia nervosa is conceptualised somewhat differently than in anorexia. In more severe cases of bulimia nervosa, preoccupations with food and weight and binge-purge activities become all-encompassing. Rosen and Leitenberg (1982) outlined the "anxiety reduction model" of bulimia nervosa. By comparing bulimia with OCD, they forwarded the idea that vomiting is an escape - avoidance behavior similar in function to hand washing in OCD. According to this model, eating (especially binging) elicits anxiety in the bulimic woman due to fear of weight gain. Subsequent vomiting then negatively reinforces binge-eating by reducing the fear of weight gain. Once vomiting has become established as an escape response it becomes the "driving force" that sustains binge-eating. Although vomiting is viewed as being central to the maintenance of bulimic behavior, the authors do not deny either the pleasurable aspects of binge-eating nor its role in relieving distressing feelings. They claim, however, that relief from these negative feelings can only be obtained when binge-eating is combined with vomiting (Rosen & Leitenberg, 1985). It is not clear how the positively reinforcing aspects of behaviors such as binging are accommodated within this framework.[1]

This conceptualisation of bulimia nervosa has prompted much research examining the nature of obsessionality in women with bulimia nervosa. Steere, Butler, & Cooper (1990) compared women with bulimia to women with generalised anxiety disorder using the Present State Examination. Bulimic women scored significantly higher on obsessional ideas and ruminations and specific phobias whereas women with GAD scored higher on other scores of anxiety. Obsessionality was the strongest variable

1 Rosen and Leitenberg's "anxiety reduction model of bulimia" has been the target of much debate. First, the model's focus on the centrality of vomiting fails to account for three conditions: the nature of binge-eating in early phases of bulimia nervosa when the woman will often binge eat for a considerable period before initiating vomiting; for those individuals who binge-eat but do not purge, and for those individuals who continue to binge-eat after cessation of vomiting behaviour (Carter & Bulik,1994). In addition the model does not pay sufficient attention to cues that precede binge-eating and to the power of binge-eating as both a positive and negative reinforcer as central maintaining factors of bulimia. A second important difference between bulimic and OCD behaviour is that the individual with OCD actively avoids situations in which they would be likely to encounter the feared stimulus. The bulimic woman, in contrast, via cravings or urges to binge, actively engages in binge-eating which results in the anxious state of fullness which she then counteracts by purging.

Table 2: Comorbidity of Eating and Anxiety Disorders

Authors	Subjects	Design & Diagnostic Criteria	Diagnostic Instrument	Group	Results
Hudson, et al., 1983	AN (15) B (48) AB (25)	In- and Out-patients	DIS	AN B AB	*Agoraphobia* 13% 10% 24% *Panic* 38% 39% 44% *Simple* 25% 12% 4% *OCD* 69% 24% 44% *Any anxiety disorder* 75% 47% 56%
Hudson, et al., 1987	Active Bulimia (51) Remitted Bulimia(19) Major Depression(29) Non psychiatric (28) controls	DSM-III Outpatients	DIS	B (total) Dep Cont	*Panic & Agoraphobia* 17% 42% 14% *OCD* 33% 25% 7% *Any anxiety disorder* 43% 50% 21%
Laessle et al., 1987	AN (13) BN (13) AB (26)	DSM-III In or Outpatients	CIDI German version)	AN B A & B	*Agoraphobia* 0% 8% 15% *Simple* 15% 40% 19% *Social* 23% 31% 50% *OCD* 15% 8% 15% *Any anxiety disorder* 31% 62% 57%
Powers et al., 1988	BN (30)	DSM III-R Volunteers treatment study	SCID-P	BN	*Panic* 3% *Simple* 20% *Social* 16.7% *OCD* 3% *GAD* 10%
Schwalberg et al., 1992	BN (20) Obese bingers (22) Social Phobia (20) Panic d/o (20)	DSM-III-R Outpatients	ADIS-R	BN OB SP Panic	*Panic* 0% 4.5% 0% 100% *Simple* 10% 13% 20% 15% *Social* 45% 36.4% 100% 25% *OCD* 15% 4.5% 5% 5% *GAD* 55% 36.4% 45% 25%
Fornari et al., 1992	AN (24) BN (21) AB (18)	DSM-III-R In- and Outpatients?	SADS-L	A BN AB	*OCD* 25% 72.9% 66%
Bossert-Zaudig et al., 1993	BN (24)	DSM-III-R Inpatients	SCID-P	BN	*Panic* 4.2% *Simple* 4.2% *Social* 4.2% *OCD* 4.2% *Panic & Agoraphobia* 4.2% *AWOPD* 8.3% Presence of anxiety disorder associated with worse outcome
Brewerton et al., 1993	BN (59)	DSM-III-R Outpatients	SCID	BN	*Social* 17%
Braun et al., 1994	AN-R (34) AN-B (22) BN (31) BN-hx-AN (18)	DSM-III-R Consecutive Inpatients	SCID	AN-R AN-B BN BN-hx-AN	*Panic* 12% 14% 10% 0% *Simple* 0% 14% 0% 6% *Social* 3% 14% 16% 28% *OCD* 21% 18% 13% 22% *GAD* 6% 0% 0% 0% *Any anxiety disorder* 35.3% 40.9% 29% 50%
Thiel et al., 1995	Mixed AN & BN (93)	DSM-III-R Inpatients	Y-BOCS	AN & BN	*OCD* 37%
Sullivan et al., 1995	AN (32) Random controls(50)	DSM-III-R Follow-up study & electoral roll control group	DIGS	AN Controls	*Overanxious* 50% 0% *Separation Anxiety d/o* 22% 1.75%
Bulik et al., 1995	BN (114)	DSM-III-R Consecutive admissions to outpatient clinical trial	SCID I	BN	*Panic* 10% *Simple* 30% *Social* 30% *Over-anxious* 27% *Separation Anxiety d/o* 10% *OCD* 4% 92% of subjects had anxiety disorder onset prior to BN
Deep et al., 1995	AN (24) weight-recovered	DSM-III-R Recruited by advertising	SADS-L modified	AN	*Simple* 25% *Social* 42% *Over anxious* 21% *Separation Anxiety d/o* 4% *OCD* 63% *GAD* 17% *Any anxiety d/o* 75%
Brewerton et al. 1995	BN (59)	DSM-II-R Consecutive referrals	SCID	BN	*Panic* 10% *Social* 17% *GAD* 12% *Any anxiety disorder* 6%

in discriminating between the two groups; however, the authors contend that the high scores on obsessionality were a reflection of obsessions about food and weight rather than non-food and body related obsessions.

Bulik et al. (1992) compared responses on the Minnesota Multiphasic Personality Inventory (Hathaway & McKinley, 1951), Symptom Checklist-90-Revised (Derogatis, 1983) and the Beck Depression Inventory (Beck, Ward, Mendelson, Mock, & Erlbaugh, 1961) between 20 women with bulimia nervosa and 20 women with OCD. Bulimic and OCD women showed no significant differences on their MMPI profiles, with the majority of women in both groups showing clinically significant elevations on scales 2 (Depression) and 7 (Psychasthenia). Scale 7 elevations suggest anxiety and tension as well as obsessions, compulsions, ruminations, perfectionism and self-critical thoughts.

A related study compared obsessionality between restrictor anorectics and women with bulimia nervosa. The restrictor anorectics scored significantly higher on the symptom measure of obsessionality on the LOI than the bulimic sample. There were no differences however on the Trait, Resistance, or Interference measures (Bulik, unpublished manuscript).

These data suggest that the presence of actual obsessional behaviors and thoughts may be more prevalent in restrictor anorectic women which may or may not be related to emaciation itself, but that both anorectic and bulimic women exhibit significant and comparable levels of trait obsessionality which is lower than that reported by women with OCD, but markedly higher than healthy controls.

Overview of Types of Anxiety and Eating Disorders

Psychometric studies focusing on dimensional characteristics of anxiety lend considerable evidence to the conceptualisation of anxiety as central to anorexia and bulimia nervosa. The pitfalls of attempting to base an aetiological model on such data are multifold. First, no study has provided a premorbid measure of either social anxiety or obsessionality in women who later develop anorexia or bulimia nervosa. Retrospective clinical reports of premorbid personality may be biased by the salience of symptoms (i.e. obsessionality) exhibited during the acute phase of the illness. Second, given that women tend to suffer from eating disorders for several years prior to seeking treatment, the anxiety scales may in part be tapping the effects, rather than the causes, of having an eating disorder. Third, starvation-induced obsessional behaviour is well-documented (Keyes, Brozek, & Henschel, 1959). Data which suggest normalisation of anxiety scores after recovery from anorexia indicate that the observed obsessionality may be a state rather than a trait phenomenon. Longitudinal studies currently underway which first assess high-risk children prior to the age of risk for developing eating disorders may aid in identifying whether observed anxious traits predate the onset of eating disorders and whether they predict vulnerability to eating disorders in a high-risk group.

Comorbidity of Anxiety and Eating Disorders

Clinical studies

In addition to psychometric studies of anxiety in women with eating disorders, studies of comorbidity have also been conducted. Estimates of rates of anxiety disorders in women with eating disorders have varied widely as a function of sampling strategy and diagnostic instruments used. Table 2 presents a summary of a selection of the major clinical studies.

Schwalberg, Barlow, Alger, & Howard (1992) compared 20 women with bulimia, 22 obese binge-eaters, 20 social phobics and 20 individuals with panic disorder using a structured interview for anxiety disorders (ADIS-R; DiNardo, Barlow, Cerny, Vermilyea, Vermilyea, Himadi, et al., 1986). They found that 75% of women with bulimia had an additional lifetime diagnosis of an anxiety disorder. The most frequent comorbid anxiety diagnoses were generalized anxiety disorder (55%) and social phobia (45%). Comparing dates of onset, 59% of the women with comorbid bulimia and anxiety reported the age of onset of the anxiety disorder to predate the onset of the eating disorder. This study is particularly valuable in that it included both anxious control groups as well as a non-bulimic eating-disordered group. The early onset of anxiety disorders suggested a possible pathway into bulimia nervosa, although the sample size was small and required replication.

We have recently examined comorbidity of anxiety disorders and bulimia nervosa in a sample of 114 women participating in a cognitive-behavioural treatment trial for bulimia to determine whether anxiety disorders tend to pre-date or follow the onset of an eating disorder (Bulik, Sullivan, Carter, & Joyce, submitted). We extended the study of Schwalberg et al. by including structured diagnoses of childhood anxiety disorders as well (overanxious disorder and separation anxiety disorder). Of the 114 women with current bulimia nervosa, 27% suffered from childhood overanxious disorder and 10% from childhood separation anxiety disorder. In addition, 10% had panic disorder, 2% agoraphobia without panic disorder, 30% social phobia, 30% simple phobia and 4% obsessive-compulsive disorder. Overall, 64% of bulimic women had any anxiety disorder. Ninety-two percent of women had the onset of the anxiety disorder prior to the onset of bulimia nervosa. The rates of comorbidity reported in this study were markedly higher than those reported in the National Comorbidity Study for women in the United States which were 5% panic disorder, 15.5% social phobia, and 15.7% simple phobia (Kessler, McGonagle, Zhao, Nelson, Hughes, Eshleman, et al., 1994).

Addressing the same issues in women with anorexia nervosa, initial analyses of a follow-up study of all women treated for anorexia nervosa in Christchurch between 1980-1984 revealed that 22% of the 32 cases studied thus far suffered from separation anxiety disorder and 50% cases from overanxious disorder prior to the development of anorexia nervosa (Sullivan, Bulik, & Fear, in progress). These rates compare to 1.75% for separation anxiety disorder and 0% for overanxious disorder for a randomly selected sample of community controls. All of these women experienced symptoms of a childhood anxiety disorder prior

to the onset of anorexia nervosa possibly suggesting an anxiety mediated pathway into anorexia nervosa. As the methodology for this study is directly comparable to Bulik (Bulik, et al., submitted), a direct comparison of childhood onset anxiety can be made between women with anorexia and bulimia nervosa. Women with anorexia nervosa display higher rates of overanxious disorder and separation anxiety disorder than do women with bulimia nervosa who themselves have markedly higher rates of these disorders than random controls.

Braun, Sunday, & Halmi (1994) also compared comorbidity of anxiety disorders across subtypes of eating disorders and found that more women with concurrent anorexia and bulimia (in comparison to restrictor anorectics, normal weight bulimics, and bulimics with a history of anorexia nervosa) tended to have an anxiety disorder as their first psychiatric disorder. Thirty-seven percent of the entire sample had some lifetime anxiety disorder and 18.1% had obsessive-compulsive disorder. Rates of OCD did not differ significantly across diagnostic subgroups. The three bulimia groups tended to have higher rates of social phobia (18.3%) than the restrictor group (2.9%). Brewerton, Lydiard, Herzog, Brotman, O'Neil, & Ballenger (1995) also found a 36% rate of any anxiety disorder in a sample of 59 bulimic women with 71% showing the onset of anxiety disorders before the onset of bulimia.

Finally, Deep, Nagy, Weltzin, Rao, & Kaye (1995) found that 75% of a sample of 24 long-term, weight-recovered anorectics reported a lifetime history of any anxiety disorder. Eleven of the fourteen subjects with childhood anxiety disorders reported the onset of an anxiety disorder prior to the onset of anorexia nervosa. The authors suggest that the presence of childhood anxiety disorders may herald the first behavioural expression of a biologic vulnerability to develop anorexia nervosa.

Epidemiological studies.

The aforementioned studies reveal a broad range of estimates of comorbid anxiety disorders. All of the above studies may overestimate the true comorbidity of eating and anxiety disorders as they all examined clinically referred samples. Clinical samples can reveal significant associations that do not exist in the community because of differential referral. Epidemiological studies provide estimates of comorbidity that are not confounded by treatment-seeking. These differences were illustrated by finding higher comorbidity of affective disorders and substance-use disorders in bulimic women sampled in a clinic than bulimic women identified epidemiologically (Bushnell, Wells, McKenzie, Hornblow, Oakley-Browne, & Joyce, 1994). Unfortunately, this study did not include anxiety disorders diagnoses in the interview conducted with the subjects recruited from the clinic.

Walters and Kendler (1995) explored epidemiologic characteristics and risk factors of anorexia nervosa as part of a large population-based study of 2,163 female twins. They examined patterns of comorbidity across three levels of severity of subclinical and clinical anorexia nervosa by altering diagnostic inclusion criteria. All three groups showed significant comorbidity with bulimia nervosa and major depression. Odds ratios indicated a significant association between a computer defined narrow group and generalised anxiety disorder, between the clinically defined narrow group and alcoholism and phobias, and between the clinically defined broad group and alcoholism, phobias and panic disorder. Thus, the presence of anorexia nervosa in either subclinical or clinical forms significantly increased the risk of also suffering from a comorbid anxiety disorder of some sort. Only the precise nature of the anxiety disorder differed according to the restrictiveness of the definition of caseness for anorexia nervosa.

In the same twin study, Kendler, MacLean, Neale, Kessler, Heath, & Eaves (1991) examined comorbidity of anorexia, affective disorders and anxiety disorders in 123 probands with broadly defined bulimia nervosa. Rates of comorbidity were: major depression (51.2%), phobias (42.3%), alcoholism (15.5%), generalised anxiety disorder (11.4%), anorexia nervosa (9.8%), and panic disorder (8.9%). Odds ratios were elevated for anorexia, alcoholism, panic disorder, GAD, phobias and affective disorder.

Kendler, Walters, Neale, Kessler, Heath, & Eaves (1995) have also analysed the data from this study examining the multivariate structure of the genetic relationship among phobias, GAD, panic disorder, bulimia nervosa, major depression and alcoholism. In this analysis, they provided statistical evidence for two genetic factors. One factor indicated a relation between major depression and GAD. The other factor loaded heavily on phobia, panic disorder and bulimia nervosa suggesting genetic factors common to these three disorders. These findings suggest first that the anxiety disorders are genetically heterogeneous, with GAD loading with major depression and panic and phobias loading together with bulimia nervosa. Second, these analyses place bulimia within the genetic 'family' of phobias and panic. Kendler, et al. (in press) highlight that the "acute, short-lived or even paroxysmal" nature of the three disorders (e.g. panic attacks, binging and purging) loaded heavily on this factor.

This series of papers by Kendler et al. has provided the best estimate of comorbidity and of genetic factors in the eating and anxiety disorders. The studies are not without limitation. Most importantly, the relatively low base rates of the eating disorders in the twin sample necessitated the use of broad definitions of the disorders. This may have led to the inclusion of individuals who would not be considered cases in the clinic and consequently diluted the severity and comorbidity estimates. Other limitations of the study are succinctly outlined by Kendler, et al. (1995); however the contributions of these papers far outweigh their limitations.

Rates of eating disorders in anxiety disorders samples. Several studies have attempted to confirm the relation between eating and anxiety disorders by examining rates of eating disorders in individuals with anxiety disorders. Case reports and small case series have addressed the rates of eating disorders in samples of women with obsessive compulsive disorder. Joffe and Swinson (1987) found no evidence of elevated scores on the Eating Attitudes Test in 16 patients with OCD. Fahy, Osacar, & Marks (1993) found prevalence rate of 11% for anorexia nervosa in a group of

105 female patients with OCD. They noted that patients with early onset OCD may be at increased risk for developing eating disorders. Similarly, Noshirvani, Kasvikis, Marks, Tsakiris, & Monteiro (1991) found a rate of anorexia of 12% in 170 female OCD patients in comparison to 0% in 137 male OCD patients. Rubenstein, Pigott, L'Heureux, Hill, & Murphy (1992) also found a 12.9% prevalence rate of anorexia nervosa in 62 patients with OCD which increased to 17.7% when the criteria were widened to include subthreshold anorexia or bulimia. Somewhat higher estimates were found by Tamburrino, Kaufman, & Hertzer (1994) who found a history of an eating disorder in 43% of 31 women with OCD. Structured interviews were not used to determine the presence of eating disorders in this study. The rates reported in these studies are markedly higher than the prevalence of anorexia nervosa in women (0.2%) reported in epidemiological studies (Wells, Bushnell, Hornblow, Joyce, & Oakley-Browne, 1989). Summarising across studies, with the exception of Tamburrino et al., the rates of anorexia in individuals with OCD are approximately 12% which is significantly greater than that expected in the general population, but represents only a minority of individuals with OCD.

Overview of Comorbidity of Anxiety and Eating Disorders

Combining information from clinical samples of women with eating disorders, epidemiologic studies and studies of anxiety disorders patients, a certain degree of clarity emerges regarding the relation between eating and anxiety disorders. First, anxiety disorders are frequent comorbid conditions in women presenting for treatment with anorexia and bulimia nervosa. Second, in a clear majority of individuals with comorbid eating and anxiety disorders, the anxiety disorder occurred first, possibly suggesting an anxiety pathway in the genesis of eating disorders. Third, epidemiologic data confirm clinical findings and reveal a genetic association between bulimia nervosa, panic and phobias thus supporting and extending clinic-based research. Fourth, eating disorders are overrepresented in clinical samples of individuals with OCD; however, it is unclear whether this overrepresentation is unique to OCD samples or would also be likely to be observed in women presenting with other primary Axis I disorders.

Treatment

The proven efficacy of a given treatment for a disorder does not necessarily reveal a causal pathway. If two distinct disorders respond to the same treatment, only limited inferences can be made about the similarity of the underlying pathogenesis of those disorders. This situation exists between the eating and anxiety disorders as two interventions—one pharmacological and one behavioural—produce substantial gains in the treatment of both disorders.

Pharmacologic treatment for anorexia nervosa.

Brain serotonin has been implicated in the modulation of appetite, mood, personality variables and neuroendocrine function (Kaye, Weltzin, & Hsu, 1993). Kaye, Gwirtsman, George, & Ebert (in press) have found elevated 5-HIAA, the major serotonin metabolite, in cerebrospinal fluid (CSF) in long-term weight-recovered anorectics. Elevated CSF 5-HIAA may reflect increased serotonergic activity. Serotonergic abnormalities have also been implicated in OCD (Insel, Mueller, Alterman, Linnoila, & Murphy, 1985; Zohar & Insel, 1987; Zohar, Mueller, Insel, Zohar-Kadouch, & Murphy, 1987).

Several studies have shown the considerable efficacy of serotonergic drugs in the treatment of OCD (Thoren, Asberg, Cronholm, Jornestedt, & Traskman, 1980; Turner, Jacob, Beidel, & Himmelhoch, 1985; Volavka, Neziroglu, & Yaryura-Tobias, 1985). Likewise, the effect of selective serotonin reuptake inhibitors has also been studied in anorexia nervosa. Kaye, et al. (1993) review several studies which have suggested that women with anorexia treated with serotonin-specific medication have an improved response to refeeding programmes (Crisp, Lacey, & Crutchfield, 1987; Gwirtsman, Guze, Yager, & Gainsley,1990; Halmi, Eckert, LaDu, & Cohen, 1986). We extended those findings (Kaye, Weltzin, Hsu, & Bulik, 1991) by administering fluoxetine in an open trial to 31 anorectics during hospitalisation and throughout one year of follow-up. We found that 29/31 patients remained at or above 85% average body weight throughout the follow-up interval. Response was judged to be good in ten, partial in 17 and poor in four participants. It also appeared that women with a restricting pattern of anorexia responded better than bulimic anorectics to fluoxetine treatment. Obviously, placebo controlled double-blind trials are necessary to confirm the relevance of this open-trial to the treatment of anorexia nervosa.

Pharmacologic treatment for bulimia nervosa.

More controlled investigations are available for the treatment of bulimia nervosa with serotonin-specific medications. The fluoxetine trial for bulimia nervosa, (Fluoxetine Bulimia Nervosa Collaborative Study Group, 1992), found that half of the subjects who received 60 mg of fluoxetine showed a ≥75% decrease in the frequency of binging and ≥60% decrease in the frequency of vomiting. Although the changes were significant, they must be considered along with the high dropout rate (31%). In addition, although rates of binging and purging were decreased significantly, many individuals were still bulimic (binging/purging twice per week) at the end of treatment. Thus, fluoxetine does not cure bulimia in most, but may assist in reducing the frequency of the core symptoms.

Behavioural interventions with bulimia: Exposure with response prevention.

As mentioned previously, Rosen and Leitenberg have posited an anxiety reduction model of bulimia nervosa wherein purging serves as an anxiety reducer by decreasing the anxiety produced by binging. The treatment which has arisen from this model, exposure with response prevention, is the behavioural treatment of choice for OCD and other anxiety disorders such as panic (Barlow, 1989; Marks, 1987; Minichello, Baer, & Jenike, 1988). Originally adapted from the flooding procedure (Malleson, 1959; Meyer, 1966), ERP is the repeated or prolonged presentation of aversive or

appetitive stimuli (either imaginally or in vivo), combined with the prevention of avoidance responses until the capacity of the stimuli to elicit anxiety or other unwanted effects have diminished or ceased (Powell, Gray, Bradley, Kasvikis, Strang, & Barratt, 1990). Rosen and Leitenberg's anxiety reduction model provided the impetus for applying ERP to bulimia nervosa (Leitenberg, Gross, Peterson, & Rosen, 1984; Leitenberg & Rosen, 1988; Leitenberg, Rosen, Gross, Nudelman, & Vara, 1988; Rosen & Leitenberg, 1985; Rosen & Leitenberg, 1988).

Their model emphasises that treatment should focus on reducing the frequency of vomiting rather than binging. Exposure treatment therefore involves exposing the bulimic woman to cues associated with vomiting and then preventing that response. For example, the bulimic woman is required to eat as much of a "forbidden" or high-risk food as she can until she has a strong urge to vomit (exposure), and then to refrain from doing so (response prevention). The patient is instructed not to vomit for at least 2.5 hours following exposure. The therapist stays with the patient until the urge to vomit has passed. This type of ERP is often referred to as P-ERP or ERP to pre-purge cues.

Alternative formulations and treatments based on ERP principles have been forwarded by Schmidt & Marks (1988; 1989). They implement exposure techniques for bulimia nervosa at an earlier link in the behavioral chain by focusing on cues that prompt binge-eating, rather than cues that precede purging. They suggest that binge-eating may function as an escape-avoidance response from unwanted affective states. Their treatment procedure involves instructing patients to eat a small amount of a binge food ("an appetizer") in order to trigger an urge to binge (exposure) but then resist the urge to do so (response prevention). The subject is encouraged to smell and touch the food and concentrate on the feelings and fears it evokes. This is often referred to as B-ERP or ERP to pre-binge cues.

An alternative conceptualization of B-ERP, using a classical conditioning analysis was forwarded by Jansen, van den Hout, deLoof, Zandbergen, & Griez (1989). Consumption of binge food was conceptualised as an unconditioned stimulus (UCS), and stimuli specifically associated with binge-eating as a conditioned stimulus (CS) in bulimia nervosa. They hypothesized that repeated or prolonged exposure to the CS in the absence of the UCS would lead to the extinction of "craving".[2]

A key advantage of both these rationales for the use of B-ERP is that they trace the origins of bulimic behavior further back in the behavioral chain. However both pay insufficient attention to the positively reinforcing aspects (or conditioned appetitive responses), involved in binge eating.

Effectiveness of ERP for bulimia nervosa.

A number of studies have reported the effectiveness of P-ERP in the context of cognitive-behavioural therapy in reducing binging and purging in bulimic women (Agras, Schneider, Arnow, Raeburn, & Telch, 1989; Giles, Young, & Young, 1985; Gray & Hoage,1990; Jansen, et al., 1989; Leitenberg, et al., 1988; Rosen & Leitenberg, 1982). For a complete review of the efficacy of ERP in bulimia nervosa see Carter and Bulik (1994). The data are conflicting (Agras, et al., 1989; Leitenberg & Rosen, 1989) as P-ERP has been reported to enhance (Wilson, Rossiter, Kleifeld, & Lindholm, 1986), to have no effect (Wilson, Eldridge, Smith, & Niles, 1991) and to detract (Agras, et al., 1989) from the efficacy of cognitive-behavioural therapy.

Three studies have reported the effective use of B-ERP (Jansen, et al., 1989; Schmidt & Marks, 1988; 1989) in the treatment of bulimia nervosa. All studies evaluating B-ERP have involved small samples, and potentially involved cross-contamination of procedures as they involved subjects being asked to eat small amounts of binge foods which is the same procedure often used in P-ERP.

Exposure techniques have been implemented in a variety of ways in the treatment of bulimia nervosa, and the significance of these variations remains unknown (Wilson, 1988). Few systematic examinations of these variables have been conducted. Little is known regarding the optimal implementation of exposure techniques with bulimia nervosa.

We are currently conducting a clinical trial comparing the efficacy of P-ERP, B-ERP and a relaxation control condition in the treatment of bulimia nervosa (Bulik, Sullivan, Carter, & Joyce, in progress). One hundred and fifty women are being treated with a core of cognitive therapy which is then supplemented with one of the aforementioned behavioural treatments. This trial should eventually be able to answer: 1) does ERP add to the efficacy of cognitive therapy for bulimia nervosa; 2) is B-ERP more effective than P- ERP or visa versa; and 3) can we predict for whom B-ERP or P-ERP may be most appropriate.

Overview of Treatment

This brief account suggests a role for selective serotonin reuptake inhibitors in the treatment of anorexia and bulimia nervosa. The precise targeted symptom of these drugs is unclear. In anorexia nervosa, they enhance response to refeeding and assist with weight maintenance. In bulimia nervosa they reduce the frequency of binging and purging but rarely effect abstinence. Data suggest there is no difference in efficacy of response between bulimic women with and without current depression (Fluoxetine Bulimia Nervosa Collaborative Study Group, 1992). Studies have not differentiated participants on measures of anxiety to determine whether these drugs may be superior in treating women with anxious or obsessional traits.

The efficacy of ERP in treating both OCD and bulimia nervosa can be interpreted in several ways. First, the acquisition of both OCD and bulimia nervosa in an individual may involve conditioning trials. Only through repeated exposure and response prevention can the conditioning be "undone". This interpretation does not

2 The term "craving" is utilized here as it was originally used in the text. The concept of craving is currently under review as it tends to be an ambiguous term that is defined differently across a range of individuals (Pickens, 1992). Henceforth, we will refer to the urge to binge rather than the craving for binge foods.

require inferences about underlying pathogenic mechanisms and can apply to any disorder or behaviour in which conditioning plays a role. Second, OCD and bulimia may be different manifestations of one core biological or cognitive vulnerability and only environmental- specific events contribute to determining the expression of the vulnerability as OCD or bulimia. This interpretation is consistent with the serotonergic theories of bulimia and OCD. This explanation could account for the efficacy of both pharmacological and behavioural treatments for both disorders. Third, the efficacy of ERP in bulimia nervosa may tell us nothing about the relation between the two disorders. This argument is compelling in that ERP has also been applied successfully to treatment of alcoholism and opiate dependence (Childress, McClellan, & O'Brien, 1986; Drummond, Cooper, & Glautier, 1990; McClellan, Childress, Ehrmann, & O'Brien, 1986; O'Brien, Childress, McClellan, & Ehrman, 1990; Rankin, Hodgson, & Stockwell, 1983) both of which are distinct in their aetiology and clinical manifestation from OCD and bulimia nervosa.

Given that the most accurate interpretation of the treatment issue is unknown, overall, results from the treatment trials provide the least compelling evidence for the relation between eating and anxiety disorders.

Conclusion

Research over the past decade has been dominated by studies of the relation between eating and affective disorders. This focus has obscured what may be a more fundamental relation, and one which may be of aetiological significance, namely the relation between eating and anxiety disorders.

The diagnostic categories of anorexia and bulimia nervosa are heterogeneous. Anxiety may not be part of the clinical picture of some women with these disorders; however, clinical and epidemiological evidence converge to suggest that a sizeable portion of women with anorexia and bulimia nervosa present with comorbid anxiety and that the anxiety disorder often occurred first.

Given these findings, anxiety disorders should be included in a pool of potential risk factors or antecedents to the development of eating disorders. Young children, especially girls who experience childhood onset anxiety disorders may therefore be at increased risk to those without anxiety disorders of developing a later eating disorder. The magnitude of risk is tempered by the base rate of the disorders in the community. The mechanism through which anxiety may lead to an eating disorder is unknown.

Several hypotheses emerge. First, recent research has suggested two types of childhood social anxiety. The first, or fearful type, refers to those children who display increased physiological arousal and fear of novel situations. The second form is a more self-conscious social anxiety and is marked by greater self-focused attention and concerns about performance (Bruch et al., 1988). Although speculative, it is conceivable that girls in this second category who are also exposed to an emphasis on thinness and appearance could witness the transfer of early self-conscious concerns about performance and behaviour to concerns about weight and appearance which could then prompt dieting and other disordered eating behaviours.

Second, early obsessional tendencies observed by some groups as common in women with eating disorders, coupled with exposure to emphasis on thinness and appearance, could lead to the obsessional and ritualistic nature of the behaviours associated with anorexia and possibly bulimia nervosa. These premorbid tendencies could then be exacerbated by acute starvation, combining to reveal the clinical picture of obsessionality in these women.

Alternatively, a third dimension, such as temperament, could be a heritable characteristic which itself predisposes to the development of both eating and anxiety disorders. This hypothesis requires more complicated longitudinal designs which are capable of addressing developmental aspects of psychopathology.

Several conclusions emerge from this review. First, anxiety should be assessed routinely when assessing eating disorders. The presence of comorbid anxiety disorders may complicate the course of treatment and may suggest alternative interventions. To date no one has examined the effect of having an untreated anxiety disorder on treatment outcome in anorexia or bulimia. Anxiety disorders are often overlooked in clinical settings. Second, anxiety disorders should not be overlooked in children. Children who exhibit acting out behaviours are usually the target of attention and intervention, whereas the shy and withdrawn child receives less attention because she or he does not cause disruption. From Kagan's work (Biederman, Rosenbaum, Bolduc-Murphy, Faraone, Chaloff, Hirshfeld, et al., 1993), we know that behavioural inhibition during childhood predisposes to later anxiety disorders. Our work suggests that childhood anxiety may place children at risk for eating disorders as well. Finally, we are in the process of conducting a longitudinal study of the offspring of women with eating disorders which may shed light on the significance of anxiety in childhood and vulnerability to eating disorders in a high-risk group.

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