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What is the association between obsessive–compulsive disorder and eating disorders?

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ABSTRACT

Because eating disorders (EDs) and obsessive compulsive disorder (OCD) co-occur at high rates and can have functionally similar clinical presentations, it has been suggested that both constructs might be part of a common spectrum of disorders. Identifying the relationship between EDs and OCD may lead to the discovery of important shared core disease processes and/or mechanisms for maintenance. The objective of this paper is to understand the relationship between EDs and OCD by systematically reviewing epidemiological, longitudinal and family studies guided by five models of comorbidity posited by Klein and Riso (1993) and others. Though this literature is relatively small, the preponderance of evidence from these studies largely suggests that OCD/ED co-occur because of a shared etiological relationship. Limitations to extant literature, and suggestions for future research are discussed.

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1. Introduction

Comorbidity among mental disorders has important diagnostic, clinical, and research implications, and raises questions about the validity of diagnostic categories and the true nature of comorbidity (Widiger & Clark, 2000). Understanding how comorbidity occurs and clarifying the relationship between two disorders may answer questions about diagnostic boundaries, mechanisms of psychopathology and common etiology. Additionally, the presence of comorbidity often affects treatment outcome and relapse risk (Carter, Blackmore, Sutandar-Pinnock, & Woodside, 2004; Milos, Spindler, Ruggiero, Klaghofer, & Schnyder, 2002; Pike, 1998), and therefore understanding how and why comorbidity occurs is essential to development of more targeted treatments.

Recently, research efforts have been directed towards investigating the purported link between eating disorders and obsessive compulsive disorder (Hudson, Pope, Yurgelun-Todd, Jonas, & Frankenburg, 1987; Lilenfeld et al., 1998). The DSM-IV (APA, 1994) categorizes three disorders under the umbrella of 'eating disorders' — anorexia nervosa (AN), bulimia nervosa (BN), and eating disorder-not otherwise specified (EDNOS), and lists a fourth condition in the appendix, binge eating disorder (BED). The eating disorders (EDs) are heterogeneous in symptom manifestation, but all have common dysfunctional thoughts and behaviors associated with food and body image on varying dimensions.

Conversely, obsessive compulsive disorder (OCD) is categorized in the DSM-IV as an anxiety disorder, and hallmarks of the disease include obsessions, defined as recurrent, intrusive unwanted thoughts and images, and compulsions, which are performed in an effort to reduce the obsession-related anxiety (APA, 1994). Individuals with OCD often have different obsessions and compulsions, such as fear of contamination/excessive handwashing or aggressive urges/excessive checking.

Although EDs and OCD seem disparate, further examination reveals similar cognitive, behavioral, and personality characteristics. Each condition has the cognitive feature of repetitive thoughts and preoccupations about a certain feared stimulus (e.g. EDs: food/body image/weight preoccupation; OCD: obsessive preoccupation with symmetry, contamination, etc) of which is usually followed by some negative affect (e.g. anxiety/fear: Buree, Papageorgis, & Hare, 1990; Rachman, 1997). For both EDs and OCD, this is then followed by compensatory behaviors (EDs: restriction, binge/purge, compulsive exercise; OCD: handwashing/checking/other compulsion) aimed at reducing the negative affect (Rachman & Hodgson, 1980; Schupak-Neuberg & Nemeroff, 1993), and anxiety more specifically (Bulik, 1995; Bulik, Sullivan, Fear, & Joyce, 1997; Godart, Flament, Perdereau, & Jeammet, 2002; Tynes, White, & Steketee, 1990).

Recently, contention among researchers has risen about how to best diagnostically classify the disorders. Because of the relationship of anxiety in ED development and maintenance, it has been suggested that EDs may be better categorized as an anxiety disorder in a transdiagnostic model of psychopathology (Fairburn, Cooper, & Shafran, 2003; Waller & Pallister, 2008). Conversely, evidence from behavioral, genetic, neuroimaging, and treatment response studies collectively suggest that OCD is distinguishable from other anxiety disorders (see Bartz & Hollander, 2006, for review), and subsequently, categorization of OCD as an anxiety disorder has been questioned. One solution to the diagnostic controversies associated with EDs and OCD is to list them under a spectrum of disorders in which phenotypically disparate disorders share common characteristics, such as perfectionistic tendencies, or obsessive and compulsive characteristics (e.g. Obsessive Compulsive spectrum, see Hollander, 1993; Hollander, Friedberg, Wasserman, Yeh, & Iyengar, 2005; McElroy, Phillips, & Keck, 1994).

Identifying the relationship between EDs and OCD may lead to discovery of important shared core disease processes and/or mechanisms for maintenance. Additionally, examination of the relation between these conditions could have important implications in the further study of diagnosis and subsequent treatment of both EDs and OCD. The goal of this paper is to use the approach put forth by Klein and Riso (1993; also see, Neale & Kendler, 1995, and Rhee, Hewitt, Corley, & Willcutt, 2005) as a basis to review the literature on the relation between EDs and OCD. Klein and Riso describe models that explain why two psychiatric disorders co-occur, and describe patterns of results that can be used to support or refute each model. Because methodological limitations of the literature make testing of the original models difficult, we have consolidated some of the models. In the next section, we discuss each of these five models.

2. Models of comorbidity

2.1. Comorbidity is due to base rates and sampling issues

The first set of explanations of comorbidity considers chance co-occurrence and the characteristics of the sample as explanations of comorbidity. The Chance Model attributes the co-occurrence of two disorders to random chance operationalized by multiplying the base rate, or prevalence, of each disorder (Klein & Riso, 1993). Thus, if disorder A has a prevalence of 10% and disorder B has a prevalence of 20% than if the disorders co-occur by chance, the prevalence rate for comorbid A/B should be 10%×20% or 2%.

The Sampling Bias Model posits that comorbidity may be a function of employing clinical samples, which thereby increase the likelihood of co-occurrence of disorders. Berkson (1946) noted this exact phenomenon and speculated that individuals who have multiple disorders are more likely to be referred for treatment than those with single disorders (i.e., 'Berkson's bias'). As such, the co-occurrence of OCD and EDs in clinical samples may be due to the severity and number of illnesses that characterize clinical populations, and thus overestimate the natural co-occurrence of the disorders.

2.2. Explanations of comorbidity based on atypicality

The Atypicality Model posits that comorbidity is an atypical form of one or both disorders. This atypicality can occur because of several reasons. For example, atypicality can arise because one disorder (e.g., AN) may be a part, or manifestation, of the other (e.g., OCD), and thus the two disorders may artifactually co-occur.

Other examples of atypicality include multiformity, in which the comorbid condition is actually an atypical form of one of the disorders, despite having phenomenological features of the other disorder. In this respect, it may be that the comorbid condition is a more severe form of one of the pure disorders. Finally atypicality may arise due to heterogeneity of a comorbid condition, such that some individuals with comorbid ED/OCD may have atypical forms of ED and other individuals with comorbid ED/OCD may have atypical forms of OCD.

Though Klein and Riso (1993) suggest that these explanations are distinct and can be tested separately, they require methodological designs that have not been used by researchers to date in the OCD and

ED comorbidity literatures. Nevertheless, the explanations above can be assumed under a broader Atypicality Model, in a best effort to test the Klein and Riso models.

2.2.1. Comorbid condition is a distinct condition

The Independent Disorder Model posits that the comorbid condition is a third, independent disorder that is distinct from either of the pure disorders. Thus, while the comorbid condition may appear to have similar symptoms from each disorder, etiologically, the comorbid condition is distinct.

2.2.2. Comorbidity occurs because of etiological relationships

The final explanation of comorbidity, the Etiological Relationship Model, attributes etiological commonalities as the underlying mechanism for occurrence of comorbidity. One form of this model suggests that the pure and comorbid conditions are at different phases or alternative expressions of the same disorder, and thus should share similar etiological processes. A second form of this model suggests that comorbidity occurs because one disorder is a risk factor for another.

Klein and Riso (1993) and others (Neale & Kendler, 1995) have outlined patterns of results for epidemiological, longitudinal, and family studies that would denote one particular model as providing a better fit of the data than another. For example, if comorbid OCD/ED is a third independent disorder of both OCD and ED, then results yielded from such studies should coalesce into a different pattern than if OCD and EDs co-occur because of an etiological relationship between the two. This review will systematically examine the literature on the comorbidity between EDs and OCD under the rubric of the five described models in order to understand the nature of their relationship. Given the strong support that OCD is distinct from other Anxiety Disorders (Bartz & Hollander, 2006), this review will focus on studies reporting OCD/ED comorbidity specifically, rather than anxiety disorders more broadly. EDs will be restricted to AN and BN, as EDNOS individuals are often excluded from OCD/ED studies. Of note, some studies reported AN and BN together, rather than separately, therefore the abbreviation "ED" is used in this review when the study reported BN and AN together. Additionally, subtypes (AN-restricting type vs. AN-binge/purge type) were often not distinguished, but we note the few studies that do.

3. Review of comorbidity studies

The goal of this section is to determine which, if any of the five models, are supported or refuted collectively by the literature, and thus provide the clearest explanation of why EDs and OCD co-occur.

4. Epidemiological studies

The first two models, the Chance Model and the Sampling Bias Model can only be tested by examining prevalence rates of comorbidity (Klein & Riso, 1993). Results from these large scale studies can determine whether the rate of comorbidity of the two disorders is higher than the chance rate. We separately review studies of samples recruited from clinics and the community as community epidemiological studies allow one to rule out the Sampling Bias Model (comorbidity is due to the possibility that those seeking treatment are more likely to have comorbid disorders).

4.1. Prevalence rates of EDs and OCD in the community

First, we will determine what the chance comorbidity rate in the community would be for OCD/ED based on prevalence estimates from community epidemiological studies of EDs and OCD. Lifetime prevalence of anorexia nervosa (AN) is approximately 0.5–0.9% and the 12-month prevalence for anorexia nervosa (AN) is approximately 0.3% (Favaro, Ferrara, & Santonastaso, 2004; Hoek & van Hoeken, 2003; Hudson, Hiripi, Pope, & Kessler, 2007, Steinhausen, Winkler, & Meier, 1997). A recent study found that the lifetime and 12-month prevalence of Bulimia Nervosa (BN) is 1.5% and 0.5%, respectively (Hudson et al., 2007) which is similar to earlier population-based surveys (Bushnell, Wells, Hornblow, Oakley-Browne, & Joyce, 1990; Garfinkel et al., 1995; Kendler et al., 1991; Steinhausen et al., 1997).

In the recent National Comorbidity Survey-replication (NCS-R), the lifetime and 12-month prevalence of OCD was estimated to be 2.3%, and 1.2%, respectively (Ruscio, Stein, Chiu, & Kessler, in press). These rates are commensurate with previous results (Bebbington, 1998).

4.1.1. Comorbidity rate if ED and OCD co-occur by chance

From these rates, chance estimates of 12-month comorbidity can be calculated as 0.0036% (0.3%×1.2%) for AN/OCD comorbidity, and 0.006% (0.5%×1.2%) for BN/OCD comorbidity, and lifetime comorbidity rates as 0.021% for AN/OCD and 0.035% for BN/OCD.

4.1.2. Prevalence rates of comorbid ED/OCD in the community

This next section examines whether the actual prevalence rates of comorbid OCD/ED in the community exceed our estimated comorbidity rates based on the Chance Model. Hudson et al. (2007) examined comorbidity rates of disorders in individuals with EDs from the NCS-R population discussed previously and found that 17.4% of BN individuals had a lifetime diagnosis of OCD. The group reported 0% lifetime prevalence of OCD in AN individuals. In the Zurich community-based epidemiological cohort study, Angst et al. (2004) reported that 14.3% of individuals with OCD had a lifetime diagnosis of BN, though rates of AN were not reported. Three other community studies (Grabe et al., 2001; Lewinsohn, Streigel-Moore, & Seeley, 2000; Rapoport et al., 2000) found no lifetime cases of ED among individuals with OCD or OCD cases among ED individuals. Of note however, these findings may be limited by small samples ($N=17$ for Grabe et al; $N=19$ for Lewinsohn et al; $N=35$ for Rapoport et al). Unfortunately, other large-scale community-based epidemiological studies (e.g., the original NCS, Epidemiologic Catchment Area Survey, Great Smoky Mountain Study) either only reported OCD or only reported EDs, and thus comorbidity was not reported. Similarly, other community studies examining comorbidity in individuals with EDs have reported high rates of anxiety disorder and ED comorbidity, but did not specifically assess OCD (Garfinkel et al., 1995, 1996).

In sum, the community epidemiological studies largely suggest that BN and OCD do not occur due to chance. Unfortunately, due to non-reporting or 0% prevalence of AN in community samples, AN/OCD comorbidity due to chance cannot be ruled out as a potential explanation.

4.2. Clinical population comorbidity studies

The bulk of studies examining comorbidity between EDs and OCD have been conducted with samples recruited from clinics, rather than the community. Although these studies cannot rule out the Sampling Bias Model, they can be used to compare comorbidity rates in clinical populations to chance rates estimates.

4.2.1. Prevalence of OCD in ED populations

In general, studies examining comorbidity in individuals with EDs have shown extremely varying rates of comorbidity between the eating disorder types. Specifically, the lifetime prevalence rates of OCD in AN individuals has ranged from 0 to 69% and in BN individuals ranged from 0 to 43% (see Godart et al., 2002; Swinbourne & Touyz, 2007 for reviews). For AN subtypes, some studies with clinical populations have reported higher OCD comorbidity rates in individuals with anorexia nervosa-binge/purge subtype (AN-BP) versus anorexia nervosa-restricting subtype (AN-R) (Fornari et al., 1992; Halmi et al., 2003; Salbach-Andrae et al., 2008; Speranza et al., 2001),

whereas others did not find significant differences between the subtypes (Godart et al., 2006; Kaye, Bulik, Thornton, Barbarich, Masters, & Price Foundation Collaborative Group, 2004; Laessle, Wittchen, Fichter, & Pirke, 1989).

4.2.2. Rates of EDs in OCD populations

Studies with clinical populations have also examined EDs in clinical samples of OCD and these rates varied, though less than rates of OCD in ED populations. The lifetime prevalence rates of AN in OCD individuals have ranged from 3 to 17% and current prevalence rate was 0–2.4% (du Toit, van Kradenburg, Niehaus, & Stein, 2001; LaSalle et al., 2004; Pinto et al., 2006; Rubenstein, Pigott, L'Heureus, Hill, & Murphy, 1992). The prevalence of BN in OCD individuals has been documented less in the literature than AN, though lifetime prevalence ranged between 3.1 and 10% and current rates reported were between 1 and 3.5% (du Toit et al., 2001; LaSalle et al., 2004; Pinto et al., 2006; Rubenstein et al., 1992).

4.3. Summary of epidemiological studies in clinical populations

In summary, the preponderance of studies in clinical populations report comorbidity rates of AN/OCD and BN/OCD that are considerably higher than chance, and suggest that these comorbidities do not occur due to chance.

Inconsistently, community studies appear to support a higher prevalence of BN/OCD comorbidity than AN/OCD, and data from clinical populations support higher rates of AN/OCD than BN/OCD. There are several reasons why results from community and clinical studies are discrepant. First, this may be due to the low prevalence rate of individuals with AN (see Swinbourne & Touyz, 2007), making comorbidity rates of AN/OCD less reliable. Second, many of the clinical samples that had very high rates of AN/OCD were assessed using DSM-III criteria, before obsessions and compulsions related to food were excluded from OCD diagnoses in the DSM-III-R (Fornari et al., 1992; Godart et al., 2002). Third, it may be that clinical samples include more severely underweight anorexics than in the community, and severe starvation and malnutrition has been shown to be associated with increased obsessional tendencies (Keys, Brozek, Henschel, Mickelson, & Taylor, 1950). Lastly, discrepancies may exist due to other methodological differences between community and clinical studies (see Swinbourne & Touyz, 2007 for discussion).

5. Longitudinal and family studies

While the Chance and Sampling Bias models can be tested with epidemiological studies, the Atypicality, Independent Disorder and Etiological Relationship models can be examined by evaluating the results and patterns of longitudinal and family studies. Klein and Riso (1993) state that in order to definitively differentiate models, follow-up studies should ideally be prospective and include three groups at baseline: pure forms of the disorders in interest (e.g. OCD and ED), and a third comorbid group (with both OCD and ED). Unfortunately, the majority of longitudinal studies of OCD/ED comorbidity utilize retrospective designs. Additionally, because of the low prevalence rates of EDs, the number of prospective studies examining comorbidity is small (Serpell, Livingstone, Neiderman, & Lask, 2002) and none examine all three groups together, and instead examine one pure disorder and a comorbid group. Thus, the following review of longitudinal studies will examine the relative support for various models, but cannot definitively rule-out any as no study has employed the requisite three group design.

5.1. Longitudinal study patterns

Klein and Riso (1993) have identified distinct patterns of results from longitudinal studies that would support various comorbidity explanations by examining the diagnostic stability of the pure disorders and the comorbid disorder over time from baseline to follow-up. If the Atypicality Model were true, results would illustrate one of two patterns:

1) one of the pure disorders (e.g., ED) remains stable over time, but the other pure disorder (e.g., OCD) and the comorbid disorder (OCD/ED) at follow-up are either the same pure disorder (e.g. OCD) or comorbid disorder (e.g. OCD/ED), but neither develop into the other pure disorder (e.g., ED);
2) each pure disorder would show diagnostic stability over time, but only the comorbid group would become either of the pure disorders (ED or OCD) or continue to exhibit the comorbidity.

If the Independent Disorder Model were true, the pattern of results would illustrate that over time EDs, OCD, and comorbidity would show diagnostic stability and not develop into one of the other groups.

If the Etiological Relationship Model were true, two patterns may emerge:

1) the first would illustrate diagnostic instability, or individuals with either pure disorder or the comorbid disorder would exhibit either of the pure disorders or the comorbid disorder (e.g. OCD, ED, or OCD/ED) at follow-up. This may also indicate that the comorbid disorder emerges as different phases of the same underlying condition.
2) the second pattern under the Etiological Relationship Model that may emerge is similar to the first form of the Atypicality Model listed above, in that one of the pure disorders (ED) would exhibit diagnostic stability over time, but in this model, individuals with the other pure disorder (e.g. OCD) or the comorbid disorder (OCD/ED) can exhibit either of the pure disorders or the comorbid disorder at follow-up (i.e. OCD, ED, or OCD/ED). This specifically suggests that one disorder is a risk factor of the other (in this example that OCD is a risk factor for EDs).

By utilizing these patterns, we hope to identify the explanations of comorbidity that are supported by the literature.

5.1.1. Longitudinal prospective studies

Several ED prospective studies examine ED comorbidity with anxiety disorders, but few report OCD separately, or only report comorbidity at follow-up, and not at baseline. The study that most directly tests the models in question is a study by Rastam, Wentz and colleagues in which 51 individuals with AN and 51 matched controls were assessed three times over an 18-year period (Rastam, Gillberg, & Gillberg, 1996; Wentz, Gillberg, Gillberg, & Rastam, 2001; Wentz, Gillberg, Anckarsa, Gillberg, & Rastam, 2009). At baseline, 27.5% of AN individuals had comorbid OCD, in which OCD occurred earlier in most of the cases. At follow-up, the vast majority of individuals with AN continued to exhibit OCD, even after recovery of AN. This is consistent with studies that found that BN individuals continue to report obsessive–compulsive symptoms, even after recovery (Morgan, Wolfe, Metzger, & Jimerson, 2007; von Ranson, Kaye, Weltzin, Rao, & Matsunaga, 1999). Collectively, these results suggest partial support for the Atypicality and Etiological Relationship models, but do not support the Independent Disorder Model.

5.1.2. Longitudinal retrospective studies

The preponderance of studies examining the chronology of onset of EDs and OCD using retrospective studies have illustrated both patterns of results that support the Etiological Relationship Model. The first supported pattern illustrates that the age of onset of OCD usually occurs prior to the onset of AN and develops on average 5.4 years earlier (Bulik et al., 1997; de Mathis et al., 2008; Thornton & Russell, 1997). These results suggests that OCD is a risk factor for AN, and thus supports the Etiological Relationship Model.

The second pattern that lends support to an Etiological Relationship Model indicates that the disorders tend not to show diagnostic

stability. For example, studies have illustrated that onset of OCD can occur prior, simultaneously, and occasionally following the onset of EDs (Godart, Flament, Lecrubier, & Jeammetodart et al., 2000; Godart et al., 2003; Kaye et al., 2004; Milos et al., 2002; Speranza et al., 2001). These studies indicate that the pure disorders are not diagnostically stable over time, and that OCD, EDs and the comorbid condition emerge as different phases of the same disorder, thus giving additional support to the Etiological Relationship Model.

In sum, longitudinal studies examining OCD/ED comorbidity suggest that the disorders co-occur due to an underlying etiological relationship (see Table 1 for summary). This conclusion, however, is tempered by several methodological limitations of the extant literature. First, most longitudinal studies examine OCD/ED comorbidity are retrospective, and are thus susceptible to biases of memory recall regarding onset ages. Second, as mentioned above, none of the studies followed all three groups of disorders (ED only, OCD only, and comorbid OCD/ED) that Klein and Riso strongly encourage. However, in spite of these limitations, results from longitudinal studies lend support towards the Etiological Relationship Model of comorbidity and, more specifically, that OCD and EDs are different phases of the same underlying condition or that OCD is a risk factor for the development of an eating disorder.

5.2. Family and twin study patterns

Family studies have traditionally been employed to determine whether the co-occurrence of disorders is due to independent, partially overlapping or identical familial risk factors or familial liability. As is the case for longitudinal studies, Klein and Riso (1993) have suggested patterns of results from family studies which support the models of comorbidity. Klein and Riso recommend that family studies of comorbidity should include four groups of probands: pure form of each disorder, the comorbid condition, and controls with neither of the pure disorders. Additionally, Klein and Riso recommend that family studies of comorbidity need to report the familial rates of each pure disorders and the comorbid condition. Thus, the following review will report familial rates of EDs, OCD, and OCD/ED comorbidity in probands with EDs, OCD, and OCD/EDs.

Though a number of studies have examined the familial transmission of disorders in their homotypic probands and relatives, i.e. ED in

Table 1
Summary of longitudinal studies.

Study	Sample	Results	Model(s) supported
Longitudinal studies			
Bulik et al. (1997)	AN ($N=68$) BN ($N=118$) MDD ($N=56$) Controls ($N=98$)	33% developed OCD prior to AN, 17% developed OCD simultaneously with AN, and 50% developed OCD after AN	Etiological Relationship (OCD is a risk factor)
de Mathis et al. (2008)	OCD ($N=330$)	Age of onset of OCD predicted future EDs	Etiological Relationship (OCD is a risk factor)
Godart et al. (2000)	AN-R ($N=29$) BN-P ($N=34$)	OCD occurred prior (33%), simultaneously (17%) or after (50%) ED age of onset in those with OCD/ED	Etiological Relationship (alternate expression)
Godart et al. (2003)	AN-R ($N=111$) AN-BP ($N=55$) BN-P ($N=86$) BN-NP ($N=19$) Controls ($N=271$)	OCD occurred simultaneously or after the onset in AN-R (61%) and AN-BP (53.4%)	Etiological Relationship (alternate expression)
Kaye et al. (2004)	AN ($N=97$) BN ($N=282$) AN/BN ($N=293$)	62% of those who had comorbid OCD developed OCD prior to ED and 38% developed OCD simultaneously or after ED onset	Etiological Relationship (alternate expression)
Milos et al. (2002)	AN ($N=84$) BN ($N=153$)	ED onset more often preceded OCD onset	Etiological Relationship (alternate expression)
Morgan et al. (2007)	Recovered BN ($N=21$) BN ($N=25$) Controls ($N=28$)	OC scores were significantly higher in BN and BN-Recovered vs. controls	Atypicality Etiological Relationship (alternate expression and OCD is a risk factor)
(Rastam et al., 1996; Wentz et al., 2001, 2009)	AN ($N=51$) Controls ($N=51$)	OCD preceded AN in majority of cases, and then continued even after recovery from AN	Atypicality Etiological Relationship (alternate expression and OCD is a risk factor)
Speranza et al. (2001)	AN-R ($N=44$) AN-BP ($N=14$) BN-P ($N=23$) BN-NP ($N=8$) Controls ($N=89$)	OCD occurred prior to ED (AN and BN not separately reported) in 65% of cases, occurred simultaneously in 17% of cases, and occurred after ED onset in 17% of cases.	Etiological Relationship (alternate expression)
Thornton and Russell (1997)	DSM-IIIR AN ($N=35$) BN ($N=33$)	OCD preceded ED in 86% of cases	Etiological Relationship (OCD is a risk factor)
von Ranson et al. (1999)	Recovered BN ($N=29$) BN ($N=31$) Controls ($N=19$)	OC scores were significantly higher in BN and BN-Recovered vs. controls	Atypicality Etiological Relationship (alternate expression and OCD is a risk factor)

Note. OCD = obsessive compulsive disorder, ED = eating disorder, OCD/ED = comorbid OCD and ED, C = controls, AN = anorexia nervosa, AN-BP = anorexia nervosa-binge/purge subtype, AN-R = anorexia nervosa-restricting subtype, BN = bulimia nervosa, BN-P = bulimia nervosa-purging subtype, BN-NP = bulimia nervosa-non-purging subtype, vs. = versus.

ED proband relatives, OCD in OCD proband relatives, fewer family studies have examined heterotypic transmission, i.e., OCD in the relatives of ED proband (e.g., Bellodi et al., 2001; Lilenfeld et al., 1998; Strober, Freeman, Lampert, & Diamond, 2007), and EDs in the relatives of OCD probands (Bienvenu et al., 2000; Black, Goldstein, Noyes, & Blum, 1994; Black, Noyes, Goldstein, & Blum, 1992). Analogous to the longitudinal studies, no study employed the ideal design of all four proband and relative groups.

5.2.1. Family studies of comorbid probands

To date, one study has compared rates of EDs and OCD in relatives of probands with ED, comorbid ED/OCD, and control probands. Although a pure OCD proband group is not included, this study comes closest to Klein and Riso's suggestion of four groups. Bellodi et al. (2001) found that rates of OCD in ED proband relatives were similar to rates in ED/OCD proband relatives, but both were significantly higher than rates of OCD in control relatives. Additionally, results revealed that familial rates of EDs in pure ED, comorbid ED/OCD, and controls were all similar. This pattern of results supports the Etiological Relationship Model, and, more specifically, that OCD is a risk factor for the development of EDs.

5.2.2. Family studies of ED probands

Family studies of ED probands have found that the relatives of AN and BN probands had significantly higher rates of EDs than relatives of control probands (Lilenfeld et al., 1997, 1998; Strober et al., 2007; Strober, Freeman, Lampert, Diamond, & Kaye, 2000; Strober, Freeman, Lampert, Diamond, & Kaye, 2001, though see e.g. Grigoroiu-Serbanescu, Magureanu, Milea, Dobrescu, & Marisnescu, 2003; Wentz-Nilsson, Gillberg, & Rastam, 1998). Two of those studies assessed OCD in ED probands, and found that relatives of AN and BN probands had significantly higher rates of OCD than relatives of control probands (Lilenfeld et al., 1997, 1998; Strober et al., 2007). Neither of these studies, however, examined the familial rates of comorbid ED/OCD. Nevertheless, the pattern of results from these studies suggest that the two disorders share a common etiological factor; therefore lending support to the Etiological Relationship Model.

5.2.3. Family studies of OCD probands

Family studies of OCD probands have supported different models than family studies of AN probands. Black and colleagues (1992, 1994) found that rates of OCD in the relatives of OCD probands were significantly higher than rates of OCD in control relatives, but rates of EDs in OCD proband relatives were similar to that of control relatives. These findings were replicated in subsequent studies (Bienvenu et al., 2000; Nestadt et al., 2000). These results do not identify one specific model of support, but rather lend support to the Atypicality, Independent Disorder, and Etiological Relationship models. This discrepancy from ED proband studies may be due to small sample size, and thus low power to detect any differences between groups.

Collectively, similar to the longitudinal studies, family studies of OCD and ED comorbidity largely supported the Etiological Relationship Model (see Table 2 for summary). Although the family studies of OCD probands did not show specificity for this model, the results of the vast majority of the ED and comorbid OCD/ED family studies did. This conclusion, however, must be taken with caution as most of these studies did not examine relatives of more than one pure disorder

Table 2
Summary of family studies.

Study	Sample	Results	Model(s) supported
Family studies			
Bellodi et al. (2001)	AN-R probands ($N=38$) AN-BP probands ($N=46$) BN probands ($N=52$) Control probands ($N=72$)	Rates of EDs and OCD/EDs were higher in ED and OCD/ED proband relatives than control relatives	Etiological Relationship (OCD is a risk factor)
Black et al. (1992, 1994)	OCD ($N=32$) OCD relatives ($N=120$) Controls ($N=33$) Control relatives ($N=129$)	Rates of OCD were higher in OCD relatives, but rates of EDs were similar in both groups	Atypicality Etiological Relationship Independent Disorder
Bienvenu et al. (2000)	OCD probands ($N=80$) OCD relatives ($N=343$) Controls ($N=73$) Control relatives ($N=300$)	Rates of OCD were higher in OCD relatives, but rates of EDs were similar in both groups	Atypicality Etiological Relationship Independent Disorder
Lilenfeld et al. (1997)	BN probands ($N=177$) Control probands ($N=190$)	Rates of EDs and OCD were significantly higher in BN relatives vs. control relatives	Etiological Relationship (alternate expression)
Lilenfeld et al. (1998)	AN-R probands ($N=26$) AN-R relatives ($N=93$) BN probands ($N=47$) BN relatives ($N=177$) Control probands ($N=44$) Control relatives ($N=190$)	Rates of EDs in AN-R and BN relatives vs. controls and rates of OCD were significantly higher in AN-R relatives vs. control relatives	Etiological Relationship (alternate expression)
Nestadt et al. (2000)	OCD proband families ($N=80$; total subj. $N=423$) Control proband families ($N=73$; total subj. $N=373$)	EDs were not significantly associated with OCD families	Atypicality Etiological Relationship Independent Disorder
Strober et al. (2000, 2007)	AN-R probands ($N=152$) AN-R relatives ($N=574$) Control probands ($N=181$) Control relatives ($N=647$)	AN-R relatives had a higher rate of AN and OCD than control relatives	Etiological Relationship (alternate expression)

Note. OCD = obsessive compulsive disorder, ED = eating disorder, OCD/ED = comorbid OCD and ED, C = controls, AN = anorexia nervosa, AN-BP = anorexia nervosa-binge/purge subtype, AN-R = anorexia nervosa-restricting subtype, BN = bulimia nervosa, BN-P = bulimia nervosa-purging subtype, BN-NP = bulimia nervosa-non-purging subtype, vs. = versus.

proband and controls, let alone Klein and Riso's recommendation of examining familial rates of four groups of probands.

5.2.4. Twin studies

Though family studies can help elucidate patterns of familial transmission, they cannot address whether these patterns are due to genetic or environmental factors (Kendler, 2005). Twin studies, however, can address this question. A review of the literature suggests that no twin study has examined the comorbidity of EDs and OCD specifically, though several twin studies have examined the comorbidity of ED and anxiety disorders. These studies suggest at least partially shared genetic transmission of EDs and anxiety disorders (Keel, Klump, Miller, McGue, & Iacono, 2005; Kendler et al., 1995; Rowe, Pickles, Simonoff, Bulik, & Silberg, 2002; Silberg & Bulik, 2005). For example, in a multivariate twin analysis, Kendler et al. (1995) examined the comorbidity between several disorders and found that a common genetic factor explained the comorbidity of BN, Specific Phobia and Panic Disorder, while familial–environmental factors only influenced liability to BN. Additionally, Silberg and Bulik (2005) used multivariate twin analyses to determine liabilities in juvenile twin girls, and model-fitting revealed a common genetic factor among symptoms of eating pathology, separation anxiety disorder, and overanxious disorder throughout development. Although neither of these studies specifically examined OCD, results do suggest that the comorbidity of anxiety disorders and EDs arises from a common underlying genetic factor.

6. Summary and conclusions

Researchers and clinicians have long hypothesized a relation between eating disorders and OCD (Hudson et al., 1987; Lilenfeld et al., 1998). Using Klein and Riso's (1993) models of comorbidity as a guide, this review has attempted to systematically examine which models of comorbidity are supported by epidemiological, longitudinal, and family studies.

Epidemiological studies suggest that comorbidity between EDs and OCD does not arise from chance or sampling bias. This conclusion, however, is supported more by the findings for BN/OCD association than the AN/OCD association, likely due to the lower prevalence rate for AN in the population (Hudson et al., 2007).

Longitudinal studies largely support the Etiological Relationship Model of comorbidity. This model subsumes several sub-models such as comorbidity being alternate expressions or different phases of the same underlying condition or that OCD is a risk factor for the development of an eating disorder (Klein & Riso, 1993). Interestingly, our review of family studies also supported the Etiological Relationship Model of comorbidity of OCD and EDs.

6.1. Personality dimensions shared between OCD and ED

Although concluding that there is a common etiological relationship between OCD and EDs is critical to understanding these conditions, what might be the exact nature of this shared etiology? One possibility is that personality characteristics, such as impulsivity and perfectionism, may be common dispositional tendencies that underlie (or mediate) the comorbidity of OCD and EDs. Indeed, numerous studies have shown that these characteristics are core personality characteristics of individuals with OCD and EDs (Shafran & Mansell, 2001; Summerfeldt, Hood, Antony, Richter, & Swinson, 2004; Wade, Tiggemann, Bulik, Fairburn, Wray, & Martin, 2008; Wonderlich, Connolly, & Stice, 2004). Though a full discussion of the relationship between these personality tendencies and OCD and EDs is beyond the scope of this review, below we discuss how aspects of impulsivity and perfectionism may underlie commonalities between OCD and EDs.

Impulsivity may be more specific to the relation between OCD and binge–purge tendencies, rather than the restricting tendencies. Research has shown that impulsivity is linked to OCD (Ettelt et al., 2007; Summerfeldt et al., 2004) and individuals with BN and AN-BP have exhibited higher impulsive traits than AN-R individuals (Claes, Vandereycken, & Vertommen, 2002; DaCosta & Halmi, 1992; Fahy & Eisler, 1993). However, different facets of impulsivity, such as externally-related impulsive behaviors (e.g. theft, reckless driving) and "urgency", may be more related to binge–purge tendencies than other facets of impulsivity (Fischer, Smith, & Anderson, 2003; Peñas-Lledó, Vaz, Ramos, & Waller, 2002). Recently, Gay, Rochat, Billieux, d'Acremont, and Van der Linden (2008) posited that "urgency" may be the facet of impulsivity that is related to compulsive behaviors in OCD, and thus may be the specific impulsivity facet that is shared between these two disorders. Future studies are needed, however, to clarify impulsivity's role in the relation between OCD and BN.

In addition to impulsivity, perfectionism has also been argued to be a core personality trait in both EDs and OCD. Tolin, Worhunsky, and Maltby (2006) found that perfectionism is more strongly endorsed in OCD patients than other anxiety disorders, and has been argued to be a risk factor for the development of OCD by the Obsessive Compulsive Cognitions Working Group (1997, 2005). Individuals with AN and BN are also consistently characterized as perfectionistic (Kaye, 2008), and AN and BN individuals tend to have higher scores on perfectionism scales than controls (Lilenfeld et al., 2000; Wade et al., 2008). Like impulsivity, this personality trait may reflect a shared underlying vulnerability for the development and maintenance of these disorders.

However, analogous to studies on impulsivity, different facets of perfectionism, may be more specific to certain ED subtypes. Specifically, studies have examined perfectionism facets such as concern over mistakes and doubts about actions (Bulik et al., 2003; Minarik & Ahrens, 1996). In a study of female twins, higher 'concern over mistakes' was only associated with AN and BN, and not other disorders, such as anxiety disorders and depression. 'Doubts about actions' was only a predictor of BN (Bulik et al., 2003). Frost and Steketee (1997) compared facets of perfectionism in OCD patients and controls and found that OCD patients had significantly higher scores on both 'concern over mistakes' and 'doubts about actions'. It is thus possible that 'concerns over mistakes' underlies the comorbidity of all EDs and OCD, but 'doubts about actions' only underlies the comorbidity between BN and OCD. Future studies are therefore needed to clarify the facets of perfectionism that may be shared between OCD and EDs.

It is also possible that perfectionism and impulsivity may be interacting mediating factors in the etiology of OCD and ED. Perfectionism has been suggested to regulate impulsive behavior in individuals with OCD (Ettelt et al., 2007), and therefore may act as a protective factor in individuals with higher impulsivity. In EDs, perfectionism has been suggested to be a risk factor for AN only when other risk factors, such as low self-efficacy, are present (Wade et al., 2008). Thus, perfectionism's role in the maintenance of ED and OCD behavior may vary, especially when impulsive tendencies are present. In addition, the interaction of these two traits may distinguish AN and BN. While exploration of shared factors includes other constructs besides perfectionism and impulsivity, further research examining these relationships is essential to our understanding of ED/OCD comorbidity.

6.2. Diagnostic implications

The results of this review also have important diagnostic implications. Given the similar nature of EDs and OCD phenotypically (and as reviewed here, etiologically), including EDs as part of an OC spectrum may be warranted (Bartz & Hollander, 2006; Treasure, 2006). This commonality, however, may only be true for certain obsessive–compulsive characteristics. For example, Hasler et al. (2005) reported that EDs are only associated with contamination obsessions and cleaning rituals and not other clusters of OC symptoms

(Hasler et al., 2005). Other studies have also reported this finding, particularly in those with binge/purge behaviors, but also found high co-occurrence of aggressive and symmetry symptoms in EDs (Halmi et al., 2003; Matsunaga et al., 1999). In short, instead of relying on DSM diagnoses, it may be more useful to examine broader OC dimensions in order to examine comorbidity more comprehensively.

Our review of the literature heeds obvious limitations in understanding how and why these disorders co-occur. First, the low prevalence rates of both disorders prevent a thorough examination of the comorbidity between EDs and OCD. Though a large part of the clinical ED/OCD literature examines AN comorbidity with OCD and reports higher rates than BN, the most recent U.S. community epidemiological study (NCS-R) did not report any cases of current AN (Hudson et al., 2007), and thus it was impossible to examine comorbidity rates in AN individuals. Additionally, older studies that used the 3rd edition of the DSM (APA, 1980) are not able to examine subtypes of EDs. While the ED and OCD populations are admittedly small, collaboration between researchers studying the psychopathology of these disorders in their respective fields may aid in the collection of data and increase the ability to determine whether specific ED features (binge/purge vs. restricting) have differential associations with OCD.

Second, longitudinal and family studies of OCD/ED comorbidity revealed additional limitations. In order to definitively differentiate these models of comorbidity, Klein and Riso (1993) and others (Neale & Kendler, 1995) recommend that longitudinal studies employ two pure disorder groups and a comorbid group, and that family studies employ all three patient proband groups plus a control proband group. As previously mentioned, none of the reviewed studies utilized the complete set of groups. Therefore, although models were partially supported, none can be completely supported. Although this is likely difficult given the low prevalence rates of the disorders in question, the understanding of comorbidity will be incomplete until these designs are utilized. Again, collaboration among researchers of both fields could result in a more complete examination of comorbidity.

In sum, despite these limitations, the extant evidence suggests that EDs and OCD may share common, underlying etiological factors which may lead to their high co-occurrence. Future research is needed to examine the specific nature of this shared etiology, though some studies suggest that it may be at least partially due to some combination of the personality traits — impulsivity and perfectionism. Collaboration between researchers may allow a more detailed examination of the relationship of these low prevalent, but debilitating conditions.

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