

Eating Disorders in Obsessive-Compulsive Disorder:  
Prevalence and Effect on Treatment Outcome

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### **Authors Note**

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**Abstract**

The aim of the present study was to examine the prevalence of eating disorder symptoms in patients with obsessive-compulsive disorder (OCD). Additional aims were to assess whether having comorbid eating disorders could influence the treatment outcome for OCD, and if symptoms of eating disorders were reduced after treatment for OCD. The sample consisted of 93 patients with a primary diagnosis of OCD. The patients underwent assessment with the Yale-Brown Obsessive-Compulsive Scale, Beck Depression Inventory, and Eating Disorder Inventory both prior to and after treatment. First, the analysis showed that the sample of OCD patients had higher prevalence of eating disorders than a population of physically active students. Moreover, the women in the sample had significantly more symptoms of eating disorders than the men. Correlational analysis showed that eating disorders did not affect the treatment outcome for OCD; the patients generally had a significant improvement of OCD symptoms. On the other hand, symptoms of eating disorders were not significantly reduced after treatment. Summarized, this study concludes that there is a high prevalence of eating disorder symptoms among patients with OCD. It further shows that comorbid eating disorders does not hinder the effect of treatment for OCD. However, as the symptoms of eating disorders persist after such treatment, an implication of the present study is that these symptoms may need closer attention.

*Keywords:* comorbidity, obsessive-compulsive disorder, eating disorder, cognitive-behavioural therapy, exposure and response prevention (ERP)

The importance of studies on both the prevalence of comorbidity, as well as the effect of treatment for people who have comorbid psychiatric disorders, is stressed in the research literature (Kendall & Clarkin, 1992). Several authors point out a connection between obsessive-compulsive disorder (OCD) and eating disorders. It has been suggested that there is an overlap between the two psychiatric diagnosis, and this generates questions about possible common neurobiological, genetic, and psychological elements (Serpell, Livingstone, Neiderman, & Lask, 2002). The primary focus in this study is symptoms of eating disorders in patients with OCD. Although more research has been done on symptoms of OCD in patients with eating disorders (Godart, Flament, Perdereau, & Jeammet, 2002), there are also some studies with the same focus as the present study (see Appendix for an overview over these studies).

Some of these studies are based on small sample sizes, however most of the research indicates that patients with OCD have a higher prevalence of eating disorders than what is found in the normal population. Research on this topic has been brought forward for approximately 70 years (Hsu, Kaye, & Weltzin, 1993), and it supports a connection between eating disorders and OCD. The importance of studying patients with this type of comorbidity is underlined by the pronounced tendency for these patients to have yet other comorbid conditions, such as depression and anxiety, as well as a higher prevalence of suicide attempts (Sallet et al., 2010).

### **Obsessive-Compulsive Disorder and Eating Disorders**

OCD is a psychiatric disorder characterized mainly by repeated obsessions and compulsions (American Psychiatric Association, 2000). Obsessions are perceived as intrusive and inappropriate, and can for instance be persistent thoughts, ideas, or images that cause anxiety or distress. To suppress or neutralize such obsessions, the patients often feel a strong need to carry out compulsions, such as repetitive behaviours or mental acts. Some of the most common obsessions are about contagion, killing a loved one, or doubts about for instance if he or she remembered to lock the door. Compulsions can for example be to repeatedly clean hands or to check if the door is locked. In the DSM-IV it is underlined that these symptoms have to be severe enough to take more than one hour a day, or cause great distress or impairment. Onset of OCD is most often seen in adolescence or early adulthood, however it may also develop in childhood (American Psychiatric Association, 2000). A review article indicated that lifetime prevalence for OCD in community samples was between 0.7% and

1.6%, and that 12-month prevalence was between 0.3% and 1.0% (Norwegian Institute of Public Health, 2009).

Some activities related to eating disorders, for instance over-exercising, have been characterized as compulsive. However, patients with eating disorders usually obtain pleasure from such activities and, since their underlying goals are related to shape and weight, they often do not have a strong desire to resist them. Hence, these actions are not considered to be compulsions as defined in the DSM-IV. Patients with OCD often experience the obsessions and compulsions as unwanted and alien to themselves (American Psychiatric Association, 2000). In other words, patients with eating disorders usually have egosyntonic symptoms, while patients with OCD usually have egodystonic obsessions (Serpell et al., 2002). This distinction is important to keep in mind when discussing the comorbidity of OCD and eating disorders.

The American Psychiatric Association (1994) describes three diagnostic categories of eating disorders in the DSM IV; anorexia nervosa, bulimia nervosa, and eating disorders not otherwise specified (EDNOS). The three diagnoses have similarities, but also some distinctions. For the purpose of the present study, where the focus is on eating disorders in general, most emphasis will be on the description of these general tendencies. However, briefly explained, patients with anorexia have an extreme preoccupation with body shape, and go through extreme measures not to gain weight. For a patient to be diagnosed with anorexia, he or she has to weigh less than 85% of what is expected. Patients with bulimia go through recurrent episodes of binge eating and compensatory behaviours (such as purging, using laxatives, or exercising excessively). EDNOS is a diagnostic category that is used to describe patients who do not meet the criteria for a specific eating disorder, but who have many of the same problems (American Psychiatric Association, 1994).

Vitousek and Gray (2007) point out that common for the different eating disorders is a belief that weight and body shape symbolizes a person's worth. These researchers also describe that these beliefs contribute to an exaggerated focus on food intake and energy consumption. Moreover, the consequences of eating disorders are often critical, and can lead to disruptions in psychological and physical health. Especially for patients with anorexia the convictions about weight and food intake are difficult to alter. In addition, they carry out belief-consistent behaviours by undereating and use compensatory behaviours such as overexercising and purging (Vitousek & Gray, 2007). Taken together, these factors contribute to the seriousness of anorexia nervosa.

The onset of eating disorders is typically around midteenage years (Fairburn, 2003). Among females, the lifetime prevalence rate for anorexia is approximately 0.5%, and it seems as if the occurrence of anorexia has increased in the last decades (American Psychiatric Association, 2000). The DSM-IV also points out that there is usually found a higher prevalence for subthreshold anorexia, i.e., patients with the diagnosis EDNOS. For men the lifetime prevalence is about one-tenth compared to that of females. Bulimia affects approximately 1-3% of the population (American Psychiatric Association, 2000). In the DSM-IV it is pointed out that males represent a small portion of these cases as well, approximately one-tenth of the occurrence rate of that in females. Furthermore, there is a much lower prevalence rate of eating disorders in underdeveloped countries than in industrialized countries (Vitousek & Gray, 2007).

A study, comparing obsessions and compulsions in patients with anorexia and OCD, found that patients with anorexia often had symptoms that dealt with symmetry, exactness, ordering, and arranging (Bastiani et al., 1996). Patients with OCD, on the other hand, more often had mixed compulsions concerning aggression, checking, cleaning, and repeating sexual and somatic obsessions. Obsessions that appeared to be similar in patients with eating disorders and patients with OCD were about religion, symmetry, and hoarding or saving. Further, counting compulsions also seemed to be common in both groups of patients. Bastiani et al. (1996) point out that, despite this research, clinicians do not seem to believe such a link exists between anorexia and OCD. The authors speculate whether this might be due to the fact that patients with anorexia lack many of the OCD symptoms commonly seen in patients diagnosed with OCD.

Findings of biological similarities further support the link between anorexia and OCD. Dysfunction in hypothalamic serotonin (5-HT) has been implicated in both groups of patients (Enoch et al., 1998). The neurobiological research on hypothalamic serotonin (5-HT) has found a possible link between behavioural traits common in both anorexia and OCD, for example perfectionism or obsessionality, and the 5-HT<sub>2A</sub>-1438G/A promoter polymorphism, or some closely linked variant (Enoch et al., 1998). It is assumed that the functioning of the 5-HT<sub>2A</sub> receptor contributes to eating behaviours and anxiety, however the exact function is unknown.

In early family research of patients with anorexia, the primary focus was on the presence of affective disorders in family members, rather than the presence of OCD (Serpell et al., 2002). However, Lilenfeld et al. (1998) conducted a controlled family study and found an increased risk of clinically subthreshold forms of eating disorders, major depressive

disorder, and OCD in relatives of anorexic and bulimic probands. The authors concluded that these disorders were unlikely to share a common cause with eating disorders, however they discussed the possibility that a specific familial risk factor for anorexia may be obsessional personality traits.

### **Eating Disorders in Patients With Obsessive-Compulsive Disorder**

Summarized from studies on eating disorders in patients with obsessive-compulsive disorder, the lifetime prevalence rate of anorexia in patients with OCD had a range from 3.1% to 26%, and point prevalence was between 0% and 3%. Regarding bulimia in patients with OCD, the lifetime prevalence rate ranged from 3% to 9.6%, while the point frequency was from 0% to 3.5% (see Appendix for an overview over these studies.). There seems to be some difference in prevalence of eating disorders in OCD depending on which measurements were used. There was only one study that used MINI International Neuropsychiatric Interview Plus (M.I.N.I Plus), and the prevalence in this study was lower than in most of the other studies. Research that used the Structured Clinical Interview I for DSM-IV (SCID-I) also indicated relatively low prevalence, while higher prevalence was found in a study that used a survey including history of eating disorders. However, the last-mentioned study did not specify how they measured eating disorders. Relatively high incidences were also found in two studies that reviewed case records retrospectively for history of eating disorders.

It is thus possible that differences in how eating disorders are measured can explain the discrepancy between these studies. The two studies that reviewed case records retrospectively (Fahy et al., 1993; Kasvikis et al., 1986) were both conducted at the Bethem-Maudsley Hospital. It is pointed out that their high estimations of prevalence may be due to them taking unusually comprehensive case histories for all patients (Kasvikis et al., 1986). Furthermore, it is important to stress that the studies that report the highest incidences have collected data retrospectively, and that they have not used well-established diagnostic tools.

One study from India did not find any OCD patients with comorbid eating disorders (Prabu et al., 2012). A possible explanation for this conspicuous finding can be found in a cross-cultural literature review, which pointed out that there were generally low reports of eating disorders in Asian populations (Miller & Pumariega, 2001). Research on Indian schoolgirls found that 29% scored higher than the recommended thresholds on the *Eating Attitudes Test* (King & Bhugra, as cited in Miller & Pumariega). However, after closer scrutinisation, these researchers noted that cultural and linguistic factors may have led to misinterpretation of some of the questions. When they looked closer at the questions

specifically addressing abnormal eating behaviour, only a very small number of girls answered positively.

### **Treatment of Comorbid Obsessive-Compulsive Disorder and Eating Disorders**

The literature regarding treatment outcome for OCD patients with comorbid eating disorders is scarce. Some clinicians hypothesise that "... treatment of OCD is generally not very effective in those who are nutritionally compromised" (Woodside & Staab, 2006, p. 659). They further assume that treatment with exposure and response prevention, and other cognitive therapeutic methods, are unlikely to have significant effect on patients that are nutritionally compromised.

There have been conducted studies on other conditions that are often comorbid with OCD, and whether such comorbidity can influence OCD-treatment. A substantial amount of research has been done on comorbid depression; nonetheless, the findings here are inconsistent (Keeyley, Storch, Merlo, Geffken, 2008). It has been suggested that not distinguishing between different severity levels of depression may be the reason for this inconsistency, and that it is merely severe depression that complicates response to exposure and response prevention therapy (ERP) for OCD (Abramowitz, 2004). These speculations were confirmed in a study (Abramowitz, Franklin, Street, Kozak, & Foa, 2000), nevertheless it was also indicated that even severely depressed patients also had moderate treatment gains. Research has so far also confirmed that severe depression influences the long-term effects of OCD-treatment (Abramowitz & Foa, 2000; Steketee, Chambless, & Tran, 2001).

In addition, studies have been conducted to assess whether yet other comorbid disorders influence the treatment outcome for OCD patients treated with ERP. Neither comorbid tic disorder in adolescents (Himle, Fischer, Van Etten, Janeck, & Hanna, 2003) nor comorbid anxiety disorder in children (Storch et al., 2008) seems to impede the treatment response, but limited research has been conducted on these two comorbid disorders. Relatively more research has looked at comorbid personality disorders (Keeley et al., 2008), and found evidence that these, especially schizotypal personality disorder, hinder the treatment of OCD (Fricke et al., 2006; Minichello, Baer, & Jenike, 1987; Moritz et al., 2004).

To our knowledge, there have not yet been conducted any studies on the effect of comorbid eating disorders on the treatment outcome for OCD. Further, no studies seem to have investigated whether ERP-treatment for OCD can affect symptoms of eating disorders. However, one study on a multimodal treatment for comorbid OCD and eating disorders has been conducted. The study examined 56 inpatients, diagnosed with OCD and a comorbid



eating disorder, before and after treatment (Simpson et al., 2013). To assess symptom levels of these disorders they used the Yale-Brown Obsessive-Compulsive Scale (Y-BOCS) and the Eating Disorder Inventory (EDI). The treatment the patients were given was a multimodal approach, called the Comorbid Program, which was based on cognitive-behavioural therapy. The treatment included a supervised eating plan, administration of medications, and social support. In addition, the approach used ERP-techniques to address both OCD and eating disorders. This means that, as well as focusing on calorie intake, the program targeted feared situations related to eating. First, the patients graded their fears by making a hierarchy of feared eating situations. Thereafter, with assistance from the therapists, they were gradually exposed to the increasingly difficult steps. Additionally, the eating plan was composed as an ERP-hierarchy, and the patients did ERP-exercises related to body shape. The results showed significant reductions in symptoms of OCD and eating disorders, both yielding high effect sizes ( $d= 1.57$  and  $d= .85$ , respectively). Depression scores were also significantly reduced (Simpson et al., 2013).

Although there are no studies with the same focus as the present study, some research has been conducted to look at the effect of comorbid OCD on the treatment outcome for eating disorders (Olatunji, Tart, Shewmaker, Wall, & Smiths, 2010; Thiel, Züger, Jacoby, & Shcübler, 1998). In one study, 254 eating disorder patients with OCD and 254 eating disorder patients without OCD, were assessed before and after treatment for eating disorders (Olatunji et al., 2010). The goal was to examine whether improvement in symptoms of OCD would influence the symptoms of eating disorders. The results indicated that treatment of eating disorders could lead to a reduction of OCD symptoms. Moreover, the results suggested that the improvements in symptoms of eating disorders fully accounted for the observed reduction in symptoms of OCD, whereas the improvements in symptoms of OCD only partly accounted for the reduction of symptoms of eating disorders. It is crucial to note that the design of this study does not rule out alternative explanations of the mediational findings. One alternative explanation is that some of the treatment elements might specifically have addressed the patients' obsessional behaviour, without being specifically directed towards eating disorders or OCD per se, and thus both disorders are influenced. Further, it is possible that some of the elements in the study were non-specific, and therefore could influence the symptoms of many disorders that are comorbid with eating disorders, for instance depression. The researchers emphasize that further investigation on specific treatments for specific disorders are needed to rule out these alternative explanations (Olatunji et al., 2010).

Other researchers (Lewin, Menzel, Strober, 2013) also underline the need for further studies on treatment of comorbid anorexia and OCD. They note that treatment, as well as presentation, prognosis, and assessment, is complicated when these two conditions co-occur. Undernourishment and low weight can impede the capability for information processing and also hinder the efficacy of psychotherapy (Bulik, Berkman, Brownley, Sedway, & Lohr, 2007). It is therefore interesting to look into whether patients with eating disorder symptoms can utilize, and have good effect of, ERP-treatment for OCD.

### **Objective and Hypothesis**

As we can see there are many factors connecting OCD and eating disorders, some psychological, others neurobiological and genetic. Taken together, the links between the disorders underline the necessity of more research on this topic. More specifically, seeing as the prevalence numbers vary a lot between studies, it is important to further assess the prevalence of eating disorders in patients with OCD. In addition, it is also necessary to study treatment for patients with this type of comorbidity. The patients in the present study underwent cognitive therapy with ERP, and the study addressed whether having comorbid eating disorders would affect the treatment for OCD patients.

Individual cognitive therapy with ERP is widely considered to be the treatment of choice for OCD (Foa et al., 2005; Franklin, Abramowitz, Kozak, Levitt & Foa, 2000; Franklin & Foa, 2002). In this treatment approach, compulsions are considered to reduce anxiety induced through obsessive ideation (Vitousek & Gray, 2007). Compulsions are therefore seen as safety behaviours, which can be either covert or overt. The approach uses repeated exposure to obsessional cues combined with prevention of compulsive behaviours, with the aim of habituating to the anxiety response caused by obsessions and to dispose of safety behaviours. In addition, the ERP treatment may include cognitive techniques, and this has been found to be useful to prevent relapse (Hiss, Foa & Kozak, 1994). Group ERP treatment seems to be an effective treatment for OCD, however more studies are required to compare the effect of group and individual treatment formats (Jónsson & Hougaard, 2009).

Summarized, there are three hypotheses in this study: (1) Regarding prevalence, the hypothesis is that there is a higher prevalence of eating disorders in patients with OCD than what is usually found in the normal population; (2) Considering treatment for comorbid eating disorders and OCD, the hypothesis is that having comorbid eating disorders will result in poorer treatment outcome for OCD patients; (3) Seeing as no interventions for eating

disorders were given, it is hypothesised that the symptoms of eating disorders will remain stable from the beginning to the end of treatment.

## **Method**

### **Participants**

In this study there were 93 treatment completers. Among this sample 64 patients were from Sørlandet Hospital in the South of Norway, while the remaining 29 were patients at St. Olavs Hospital in the Centre of Norway. The participants were recruited through referrals from various psychiatric outpatient clinics and general practitioners. After being given a description of the study, all of the participants gave their written informed consent. The study has been approved by Regional Ethics Committee for research with human subjects.

The patients were assessed with SCID-I (First, Spitzer, Gibbon, & Williams, 1995), and the main inclusion criterion was that they had a primary diagnosis of OCD. In addition, the participants had to score 16 or higher on the Y-BOCS (Goodman et al., 1989a, 1989b). Furthermore, only patients between age 18 and 65 were included. Exclusion criteria were current alcohol or drug abuse/ dependence, psychotic disorder, high risk of suicide, and general assessment of functioning below 50. Patients were also excluded from the study if they were taking benzodiazepine medication, and if they had been involved in exposure therapy during the last six months. In a diagnosis-specific group treatment in a community clinic these exclusion criteria would normally be used, and therefore they were utilized as exclusion criteria in this study.

The characteristics of the final sample are presented in Table 1. The reason for dividing the sample into men and women is, as mentioned, that eating disorders are found more often in women than in men (American Psychiatric Association, 2000). As the table shows, there were also some significant demographic differences between women and men. This applied to age, with men being significantly older than women in the sample, and to onset age of OCD, where men had a significantly higher average age compared to women. Furthermore, a significantly higher proportion of men in the sample used SSRIs, compared to women. There were no significant gender differences regarding duration of OCD, civil status, employment status, or for any of the comorbid disorders. Concerning civil status, there was a slight tendency that more women than men in the sample were married or cohabitating, this was however not a significant difference.

Table 1

*Pre-Treatment Status on Demographic and Diagnostic Variables*

	Women (n = 67)	Men (n = 26)	Total (n = 93)	<i>p</i>
	<i>M (SD)</i>	<i>M (SD)</i>	<i>M (SD)</i>	
Age	32.7 (10.8)	38.5 (12.6)	34.4 (11.6)	.029
Onset age of OCD	17.8 (8.5)	22.6 (11.6)	19.1 (9.7)	.037
Duration of OCD in years	14.7 (11.1)	16.7 (11.4)	15.2 (11.1)	.457
	%	%	%	
Married/ cohabiting	70.3	50.0	64.0	.095
Using SSRI	40.6	70.8	49	.014
Employment status				.613
Employed	33.3	41.7	35.6	
Unemployed	44.4	37.5	42.5	
Student	17.5	16.7	17.2	
Part-time employee	4.8	4.2	4.6	
Panic/ agoraphobia	18.8	25.0	20.5	.523
Social anxiety disorder	25.0	12.5	21.6	.209
Generalized anxiety disorder	18.8	20.8	19.3	.828
Specific phobia	12.5	16.7	13.6	.617
PTSD	9.4	4.2	8.0	.427
Depressive disorder <sup>a</sup>	37.1	43.5	37.5	.626

*Note.* The *p*-values show the comparison between men and women. OCD = obsessive-compulsive disorder; PTSD = Post-traumatic stress disorder; SSRI: Selective serotonin

<sup>a</sup> Includes dysthymia, major depression and recurrent depression.

### **Treatment and Therapists**

All the participants underwent ERP-treatment for OCD, however 66 patients received the treatment in a group format, and 27 patients underwent an individual form of the treatment. No interventions were aimed at reducing symptoms related to eating disorders.

The groups in the ERP-group therapy consisted of approximately six participants. The program stretched over twelve weeks, and the groups met once a week for sessions that lasted 2.5 hours. Three and twelve months after treatment the participants met again for follow-up meetings. The behavioural group therapy was based on a Norwegian translation of the manual developed by Krone, Himle, and Nesse (1991). Psycho-education and ERP, both in-vivo and home-based, were the two main components of the treatment program. The therapists

conducting the group therapy were one psychologist and one psychiatrist, with respectively ten and eight year's therapy experience with various psychiatric conditions.

During the first six sessions, psycho-education was given for approximately 20-40 minutes each time. The topics for the psycho-education were: the nature of OCD and the principles of behavioural therapy; causes of the disorder; other treatments available for OCD; family life; specialized techniques that make the behaviour therapy effective; and lifestyles and OCD. The remaining time in these six first sessions was devoted to ERP. In the final six sessions, nearly all the time was dedicated to in-vivo ERP exercises and planning of homework. However, the last session focused especially on termination of the therapy and maintenance.

The participants were given a workbook at the beginning of treatment, and it was essential that the participants used this book systematically. The workbook consisted of educational material, forms for self-completed behavioural analysis, and forms for recording behavioural exercises. In addition, as homework, the participants were to record Subjective Units of Discomfort (SUDS) related to the specific homework exercises they were assigned. An important goal for the therapists was to ensure that each group participant had individualized treatment exercises based on behavioural ERP. In the later sessions, patients were to design their own homework assignments. In-vivo exercises were conducted in the sessions to assist participants when they were initiating new difficult assignments. Due to limited time available, approximately half of the group sessions included in-vivo exercises for every group member, except for a few patients, for whom it was difficult to find suitable exposures exercises.

Family members, or other significant persons, were invited to participate in the group after the fourth session. During the family session, the family members were taught about the nature of OCD, behavioural therapy for the disorder, and they were given specific recommendations regarding family involvement in OCD and its treatment (see Krone et al., 1991 for a more detailed description of the treatment program).

The individual ERP treatments were based on Kozak and Foa (1997), and contained the same central features as in the group format. Psychologists, psychiatrists, and a psychiatric nurse conducted the therapy. In summary, the treatment elements were: psycho-education about OCD and the treatment model; construction of a symptom hierarchy; ERP, both in-vivo and as homework; and finally maintenance relapse prevention. A mean amount of 15 individual sessions were given. The individual treatment sessions lasted for 90 minutes, and each patient had two sessions per week.

## Measures

The different assessment instruments were administered before and directly after treatment.

**Symptoms of obsessive-compulsive disorder.** To assess patients for symptoms of OCD, trained independent evaluators administered Y-BOCS (Goodman et al., 1989a, 1989b). Y-BOCS is a structured clinician-rated interview containing 10 items. Each item is answered on a scale from 0 (no symptoms) to 4 (extreme symptoms), with the total range being 0 to 40. The interview also gives separate subtotals for the severity of obsessions and compulsions, with five items assessing obsessions and five items assessing compulsions (Goodman et al., 1989a, 1989b). In the present study only total scores were used. The pre-treatment Y-BOCS had a low alpha value (.69), probably due to restriction of range, while the post-treatment Y-BOCS showed a good internal consistency (alpha value= .89).

**Symptoms of eating disorders.** To measure symptoms of eating disorders, the EDI-I (Garner, Olmstead, & Polivy, 1983) was used. This inventory is a self-report measure, which consists of 64 items presented in a Likert scale format with six alternatives ranging from “always” to “never”. The EDI is designed to assess psychological and behavioural traits common in anorexia and bulimia (Garner et al., 1983). Although it should not be used as a diagnostic instrument, the inventory can obtain important information about clinical status and response to treatment when administered at different points in time (Garner, 1990).

The EDI is a multiscale measure, and includes eight subscales: EDI 1 – *Drive for Thinness*; EDI 2 – *Bulimia Nervosa*; EDI 3 – *Body Dissatisfaction*; EDI 4 – *Ineffectiveness*; EDI 5 – *Perfectionism*; EDI 6 – *Interpersonal Distrust*; EDI 7 – *Interoceptive Awareness*; and EDI 8 – *Maturity Fears*. The researchers behind the development of the EDI have established reliability (internal consistency) for all subscales and several indices of validity. In the current study we used the first three subscales and the total EDI index when describing the prevalence of eating disorders in patients with OCD prior to treatment. We also looked at the same EDI subscales when assessing symptoms of eating disorders after treatment of OCD.

The reason for only using the three first subscales, in addition to the total EDI score, is that these three subscales assess attitudes and behaviours related directly to body shape and eating (Garner et al., 1983). Furthermore, as the same authors point out, patients with anorexia and bulimia score significantly higher than normal controls on these subscales. The remaining subscales assess symptoms that are common in many psychiatric disorders (Pigott et al., 1991). However, these researchers (Pigott et al., 1991) also view the subscale *Interoceptive Awareness* as specifically measuring eating disorders, but the present study has

not focused on this subscale. This is due to the consensus, as summarized by Nevoen, Clinton, and Norring (2006), that the first three subscales can discriminate between patients with eating disorders and psychiatric controls, while the remaining subscales yield more ambiguous evidence. The pre-treatment EDI showed good internal consistency (Cronbach's alpha: Total EDI= .95; *Drive for Thinness* = .82; *Bulimia Nervosa* = .83; *Body Dissatisfaction*= .93).

**Symptoms of depression.** As a measure of depression, the Beck Depression Inventory (BDI) (Beck, Rush, Shaw, & Emery, 1979) was administered. The BDI is a widely used measure that has proved to be reliable and valid in assessing severity of depression in both clinical and non-clinical populations (Beck, Steer, & Garbin, 1988). The questionnaire consists of 21 items that are descriptions of symptoms and attitudes, for instance "sense of failure" and "guilt feeling". The items are to be rated on a scale from 0 to 3 in terms of intensity. The Cronbach's alpha for the pre-treatment BDI was .92, and thus the BDI showed good internal consistency.

### Overview of the Data Analysis

To find the prevalence for eating disorders among patients with OCD we used already established cut-off scores for three of the EDI subscales and the total EDI score. We looked at the prevalence separate for men and women, as well as the total percentage. The patients in our sample were also compared to a sample of physically active students, from a study conducted by Kjelsås and Augestad (2004), on the different EDI scores. The effect sizes between the samples were calculated with *Cohen's d* with pooled standard deviations.

The first subscale we looked at was *Drive For Thinness*, and for this subscale we used a cut-off at 15 points or higher ( $\geq 15$ ). This cut-off score is based on the mean score on the subscale *Drive for Thinness* for patients with anorexia nervosa (Torstveit, Rosenvinge, & Sundgot-Borgen, 2008), and can therefore be considered a conservative cut-off. The other subscale we looked at was *Bulimia Nervosa*. In this case we also used a cut-off point that has been set by others (Meltzer et al., 2001). In accordance with this, the cut-off was set to 5 or higher ( $\geq 5$ ). As a third measure of the prevalence for eating disorders among patients with OCD, we looked at the subscale *Body Dissatisfaction*. Here the cut-off was set to 14 points or higher ( $\geq 14$ ). This cut-off is also seen as relatively conservative since it is based on the mean scores of patients with anorexia nervosa (Torstveit et al., 2008). In addition to the subscales, we also looked at the total EDI score. A common cut-off for the total EDI score is 40 points

or higher ( $\geq 40$ ) (see for instance Augestad & Flanders, 2002; Kjelsås & Augestad, 2004). However, it should be underlined that this measure of eating disorders may be too liberal, since using the total EDI means also including the subscales that measure more general psychiatric problems (Pigott et al., 1991). Particularly in research such as the present study, where the aim was to examine eating disorders in OCD patients, using the total EDI could result in falsely high prevalence numbers.

In addition to examining the prevalence, we also carried out a correlational analysis to assess whether eating disorders influence the treatment outcome for OCD. Moreover, we conducted a paired samples t-test of the EDI scores before and after treatment to assess the changes in eating disorder symptoms. Paired sample t-tests were also performed to see if there were changes in symptoms of OCD (as measured by Y-BOCS) and depression (as measured by BDI). Additionally, effect sizes for these changes were calculated by using *Cohen's d* with pooled standard deviations.

## Results

### Prevalence

The OCD patients in the present study scored higher on the different EDI scales than physically active students (Table 2). However, as presented in the table the effect sizes ranged from low to moderate.

Table 2

*Mean, Standard Deviation, and Effect Size for the EDI Scores for OCD Patients and Physically Active Students*

	OCD (n= 93)		Physically active students (n= 1482)		<i>d</i>	
	Women (n= 67)	Men (n= 26)	Women (n= 905)	Men (n= 577)	Women	Men
	M (SD)	M (SD)	M (SD)	M (SD)		
DT	7.12 (7.13)	3.62 (3.65)	3.80 (5.01)	1.12 (4.92)	.54	.58
BN	2.32 (3.91)	1.00 (1.94)	1.24 (2.43)	0.74 (2.89)	.33	.11
BD	11.72 (9.43)	4.76 (5.11)	7.86 (7.41)	2.40 (3.43)	.46	.54
EDI	52.66 (34.26)	37.60 (21.37)	25.84 (19.50)	18.01 (13.48)	.96	.58

*Note.* DT = EDI – Drive for Thinness; BN = EDI – Bulimia Nervosa; BD = EDI – Body Dissatisfaction; EDI = Total EDI index. The scores for physically active students are collected from the Kjelsås and Augestad (2004) study. The effect size (*d*) was calculated by using *Cohen's d* with pooled standard deviations.



Looking closer at the sample of OCD patients in the present study, the results showed that 16.3% of the sample had a score above cut-off on the subscale *Drive for Thinness* (Figure 1). Furthermore, women had a higher mean score than men on this subscale, and there was a significant difference between the genders ( $p = .003$ ), with a moderate effect size ( $d = .62$ ). Besides, none of the men, but 24.5% of the women, had scores above cut-off.

Figure 1. Prevalence Calculated from the Subscale *Drive for Thinness* on the EDI

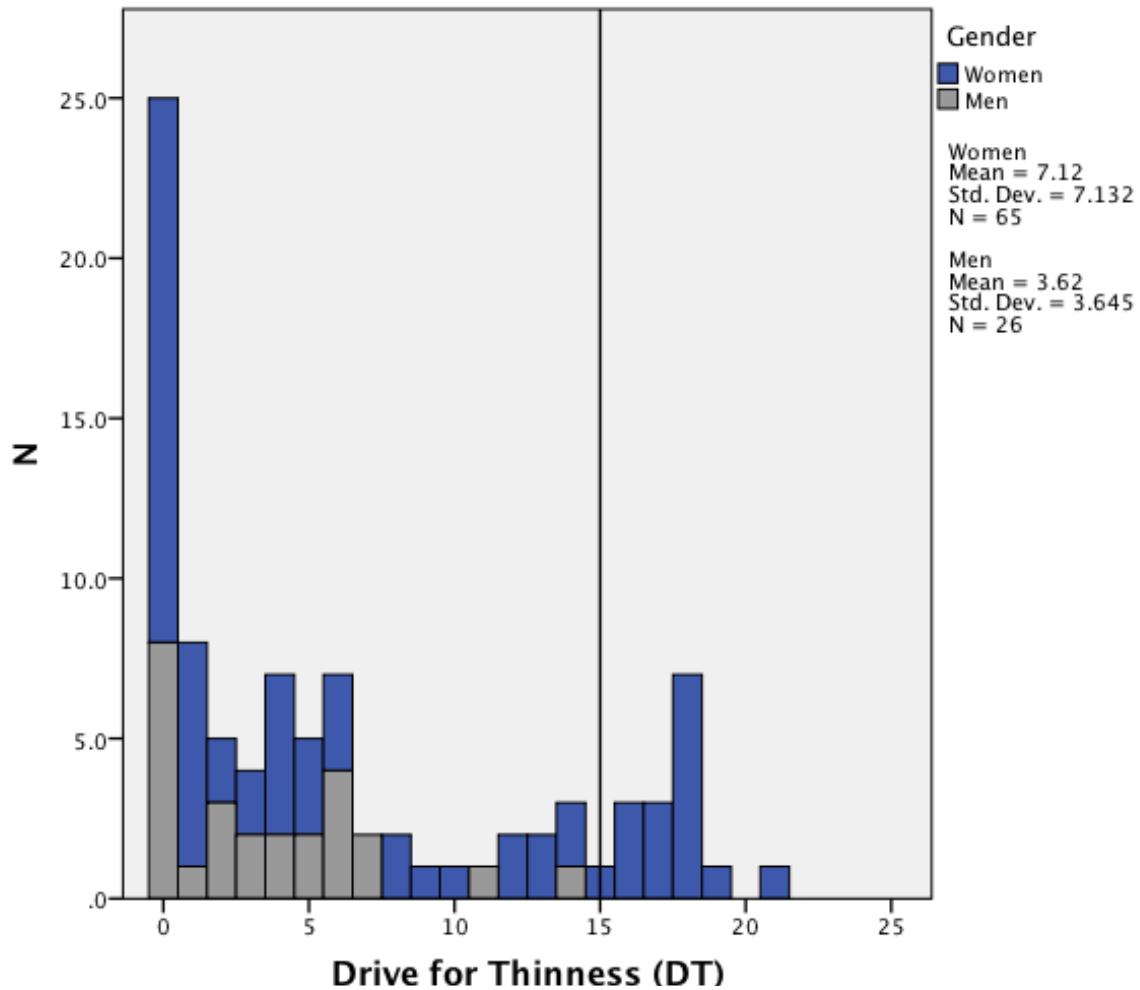


Figure 1. The vertical line represents the cut-off score on the EDI subscale *Drive for Thinness*, at 15 points or higher ( $\geq 15$ ).

On the subscale *Bulimia Nervosa*, 14.3% of the sample had a score above cut-off. In addition, the graph (Figure 2) shows that women had a higher mean score than men on this subscale. There was a significant difference between the genders ( $p = .04$ ), and the effect size was relatively low ( $d = .43$ ). Furthermore, 11.5% of the men and 16.7% of the women had scores above the cut-off.

Figure 2. Prevalence Calculated from the Subscale *Bulimia Nervosa* on the EDI

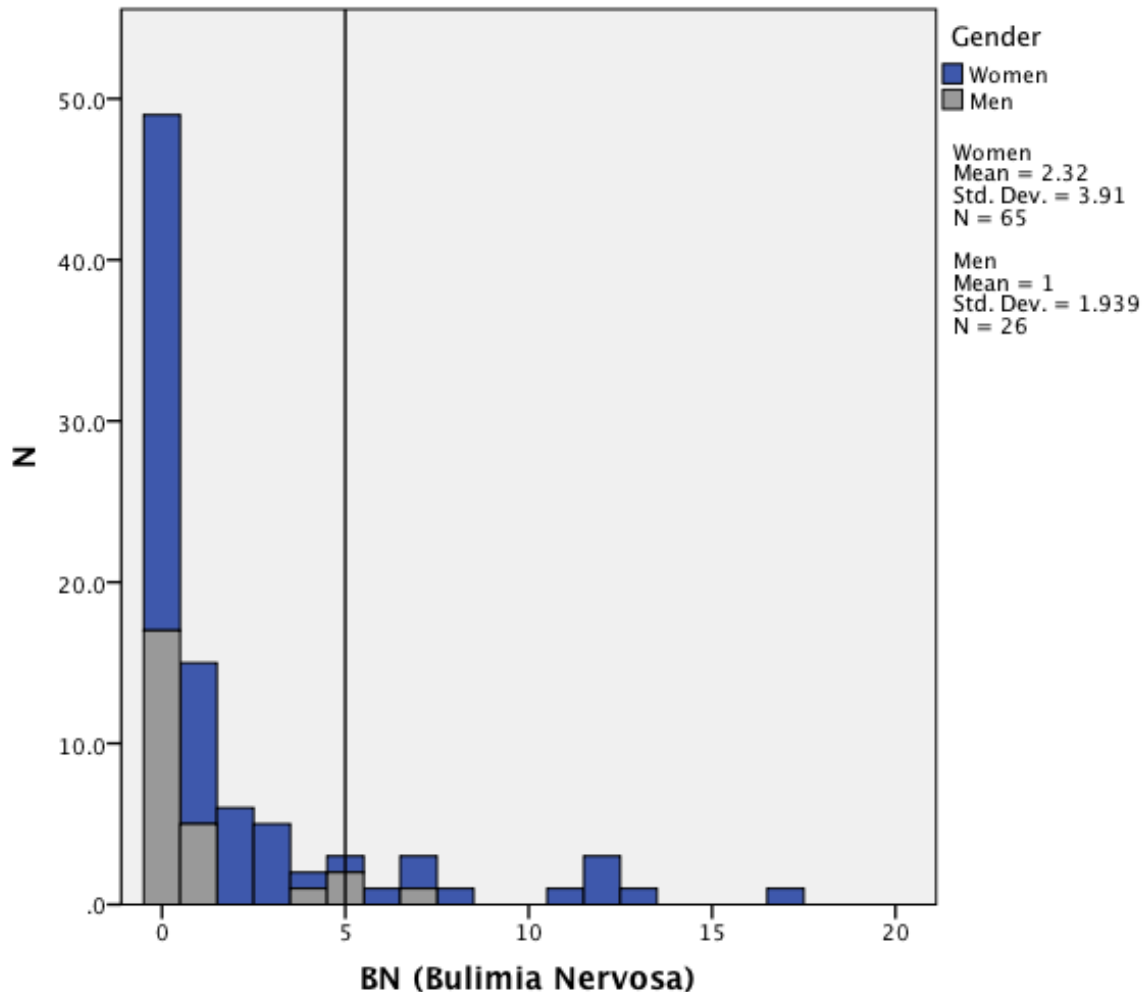


Figure 2. The vertical line represents the cut-off score on the EDI subscale *Bulimia Nervosa*, at 5 points or higher ( $\geq 5$ ).

On the subscale *Body Dissatisfaction*, 30.9% received a score above cut-off (Figure 3). The women in the sample had a higher mean score than the men in the sample, and there was a significant difference ( $p = .00$ ) between the genders also on this subscale, with a large effect size ( $d = .92$ ). Moreover, there were relatively more women above cut-off than men, with the percentages being 40% and 12%, respectively.

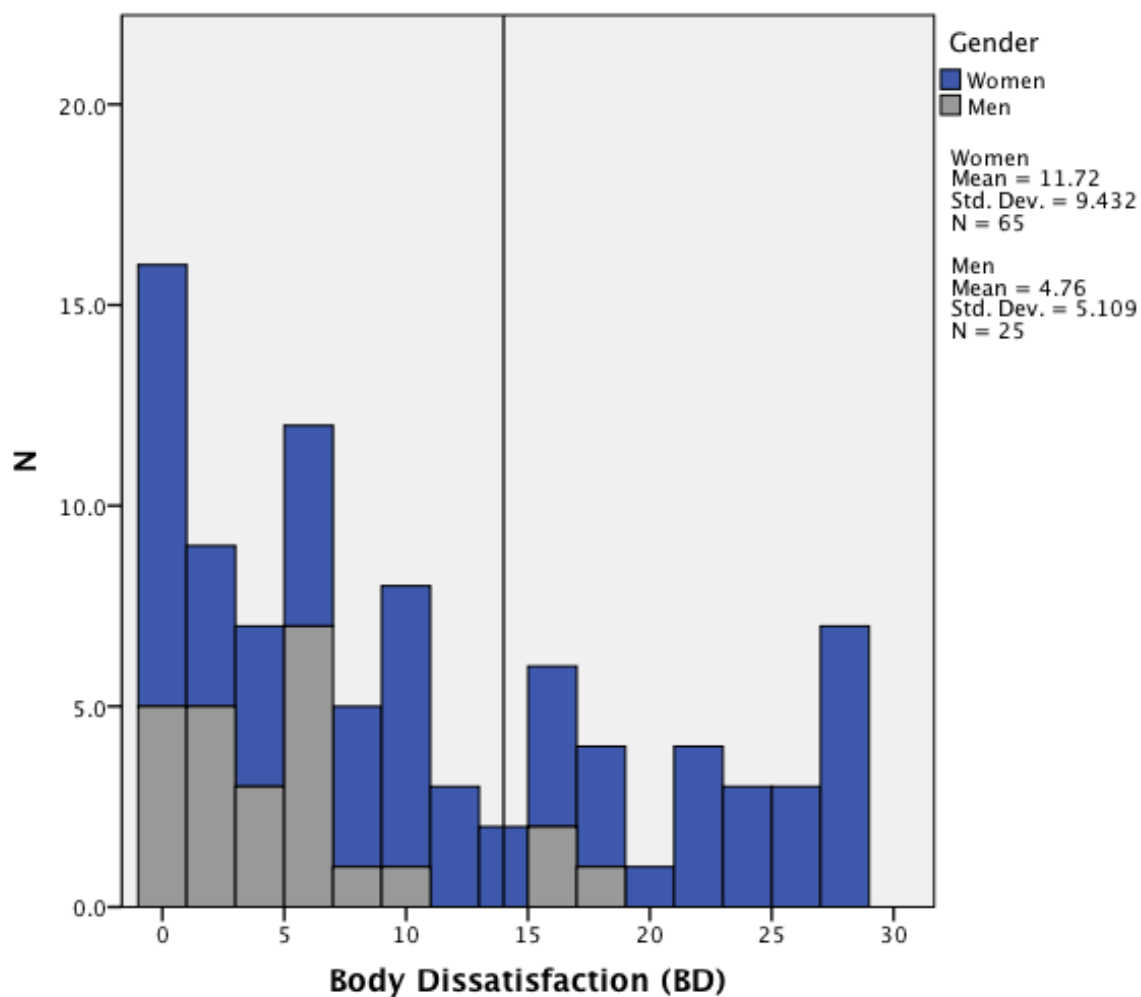
Figure 3. Prevalence Calculated from the Subscale *Body Dissatisfaction* on the EDI

Figure 3. The vertical line represents the cut-off score for the EDI subscale Body Dissatisfaction, at 14 points or higher ( $\geq 14$ ).

On the total EDI index, 53.1% of the sample had scores above the cut-off. It is important to underline again that using the total EDI score as an indicator of eating disorders may give falsely high scores, since all of the subscales are included. As illustrated in Figure 4, women had a higher mean level of EDI scores than men. The difference between the genders was significant ( $p = .02$ ), and the effect size was moderate ( $d = .53$ ). Further, relatively more women than men had scores above cut-off, with the percentages being 57.2% and 36%, respectively. In comparison to our sample of patients with OCD, 18.45% of the physically active women (from the Kjelsås and Augestad (2004) study) scored above cut-off on the total EDI. The corresponding percentage for men in this comparison group was 6.07%.

Figure 4. Prevalence Calculated from the Total Score on the Eating Disorder Inventory

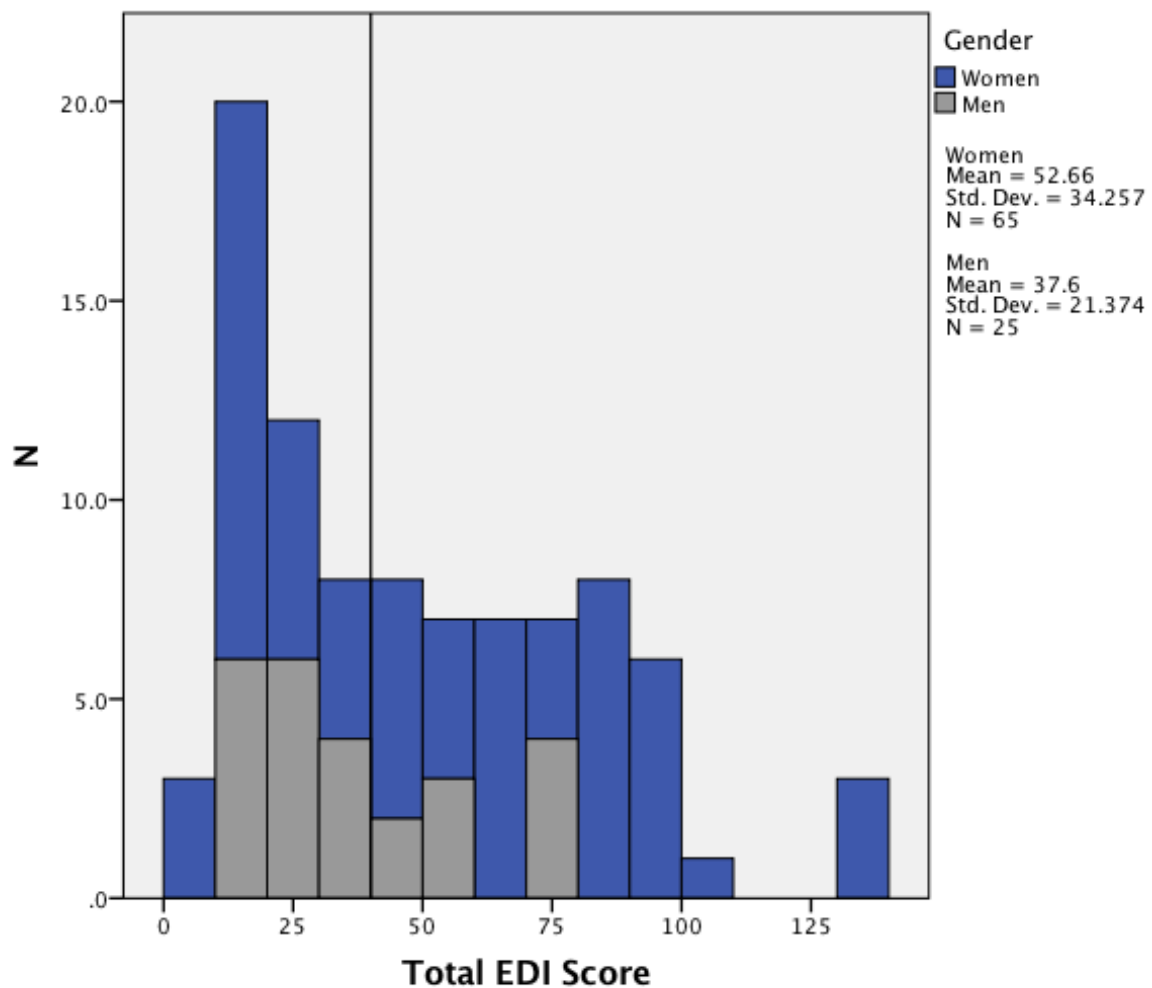


Figure 4. The vertical line represents the cut-off score for the Total EDI, at 40 points or higher ( $\geq 40$ ).

### Eating Disorders Effect on Treatment Outcome

As Table 3 shows, there were no significant correlations between symptoms of eating disorders prior to treatment and symptoms of OCD post treatment. Neither did depression scores before treatment influence OCD scores after treatment. However, the results indicated significant correlations between eating disorder symptoms before treatment and depression symptoms both prior to and after treatment.

Regarding how the different EDI scores related to each other, there were significant correlations between all the subscales and between the subscales and the total EDI score, as would be expected.

Table 3

*Correlational Analysis for Pre-Treatment and Post-Treatment Tests for Symptoms of Eating Disorders, Depression, and Obsessive-Compulsive Disorder*

	BN pre	BD pre	EDI pre total	BDI pre	Y-BOCS pre	Y-BOCS post	BDI post
DT pre	.69**	.71**	.79**	.43**	.16	-.01	.30**
BN pre		.60**	.69**	.40**	.10	-.14	.32**
BD pre			.83**	.52**	.14	.03	.38**
EDI pre total				.71**	.35**	.08	.53**
BDI pre					.44**	.14	.66**
Y-BOCS pre						.27*	.22*
Y-BOCS post							.31**

*Note.* DT= EDI – Drive for Thinness; BN= EDI – Bulimia Nervosa; BD= EDI – Body Dissatisfaction; EDI= Eating Disorder Inventory; BDI= Beck Depression Inventory; Y-BOCS: Yale-Brown Obsessive-Compulsive Scale.

\*  $p < .01$ .

\*\*  $p < .05$ .

### **Changes in Symptoms of Obsessive-Compulsive Disorder, Depression and Eating Disorders**

Paired samples t-tests and effect sizes comparing Y-BOCS, BDI, and EDI scores before and after treatment are presented in Table 4. The data provide evidence that there was a significant change in OCD symptoms, and the effect size was high. Furthermore, the depression symptoms were also significantly changed, although here the effect size was moderate. In addition, there was a significant change on the total EDI score, with a small effect size. However, the mean scores on the three subscales, *Drive for Thinness*, *Bulimia Nervosa*, and *Body Dissatisfaction* did not change significantly from pre to post treatment, and the effect sizes were low. Therefore, it is evident that there were some, or all, of the other subscales (EDI 4-8: *Ineffectiveness*; *Perfectionism*; *Interpersonal distrust*; *Interoceptive awareness*; and *Maturity fears*) that changed.

Table 4

*Paired Samples T-Test and Effect Sizes for Pre-Treatment and Post-Treatment Symptoms of Obsessive-Compulsive Disorder, Depression, and Eating Disorder*

Measure	N	Pre-treatment mean (SD)	Post-treatment mean (SD)	<i>t</i>	<i>p</i>	<i>d</i>
Y-BOCS	88	24.34 (4.29)	13.98 (6.28)	14.73	.00	1.93
BDI	82	16.91 (10.50)	10.88 (10.11)	6.40	.00	0.59
DT	75	6.13 (6.41)	5.57 (6.23)	1.46	.15	0.09
BN	75	1.84 (3.27)	1.60 (2.96)	1.03	.31	0.08
BD	74	9.99 (9.18)	9.62 (8.95)	.82	.42	0.04
EDI total	73	47.88 (30.73)	39.81 (38.31)	4.07	.00	0.23

*Note.* Y-BOCS= Yale-Brown Obsessive Compulsive Scale; BDI= Beck Depression Inventory; EDI total= total score for the Eating Disorder Inventory; DT= the EDI subscale *Drive for Thinness*; BN= the EDI subscale *Bulimia Nervosa*; BD= the EDI subscale *Body Dissatisfaction*.

### Discussion

The first aim of this study was to look at the prevalence of eating disorders among patients with OCD. The results indicated that there was a higher prevalence of eating disorders among patients with OCD than in the normal population. The second aim was to investigate if comorbid eating disorders had an effect on the ERP-treatment for OCD. The results brought forward evidence that eating disorder symptoms prior to treatment and OCD symptoms after treatment were uncorrelated. The third aim of this study was to examine whether the ERP-treatment for OCD influenced eating disorder symptoms. The study showed that the first three EDI subscales (*Drive for Thinness*, *Bulimia Nervosa*, and *Body Dissatisfaction*) did not change significantly between pre and post treatment. The total EDI scores changed significantly; however, it was the remaining subscales (EDI 4-8) that could explain this change, which again indicated a change in general psychopathology. In other words, it seemed as if patients with OCD, who also had tendencies of eating disorders, were able to utilize the ERP-treatment for OCD; their symptoms of OCD were significantly reduced. On the other hand, ERP-treatment for OCD did not seem to influence symptoms of eating disorders.

## Prevalence

Looking closer at the prevalence data it is evident that all of the EDI scales used to measure eating disorders were elevated in patients with OCD. The subscale *Drive for Thinness* indicated that 16.3% of the OCD patients had symptoms of eating disorders. Using the subscale *Bulimia Nervosa* suggested that 14.3% had symptoms of eating disorders, while the subscale *Body Dissatisfaction* indicated that 30.9% of the OCD patients had eating disorder symptoms. The total EDI indicated a very high prevalence of eating disorders in OCD patients, at 53.1%. It is plausible to assume that using the total EDI as a measure of eating disorders, thus including the five latter subscales, gave a falsely high prevalence here. Other researchers support this assumption by pointing out that these subscales measure more general psychopathology (Pigott et al., 1991), and that the first three subscales are those that directly reflect attitudes and behaviours towards body shape and eating (Garner et al., 1983). Nonetheless, taken into consideration the findings from the subscales, the present study indicated that patients with OCD had an unusual high prevalence of eating disorders. This tendency is further supported by the comparison between the sample of OCD patients in the present study, and the physically active students from the Kjelsås and Augestads (2004) study.

Furthermore, as Pigott et al. (1991) point out in a similar prevalence study, it is important to note that these high scores can also be a reflection of pervasive obsessional characteristics that affect all attitudes and behaviour in OCD patients, and therefore also appetite and appearance. Another possibility, mentioned by the same researchers, is that the findings can indicate shared characteristics of abnormal eating behaviour and body image distortion for patients with OCD and patients with eating disorders.

The term comorbidity can be used to describe two independent disorders that co-occur, two disorders with a common underlying aetiology, or two disorders with a causal relation between them. Comorbidity can also be a result, or an artefact, of the shared diagnostic criteria in the diagnostic system (Kendall & Clarkin, 1992). As discussed, OCD and eating disorders have some common psychological, neurobiological, and genetic elements. There has been a discussion on whether eating disorders are a part of the continuum of “obsessive compulsive spectrum disorders” (e.g. Bienvenu et al., 2000; Lochner & Stein, 2006), and this can to some extent be seen as a discussion about how to define comorbidity. It is important to take note of this discussion, however it is beyond the scope of the current study to engage in the debate.

It is worth mentioning that there was a significant gender difference in the prevalence of eating disorders in patients with OCD. Women had higher mean scores than men on the total EDI and on the three subscales. Accordingly, higher percentages of women had scores above the cut-off point on all the scales we used. This gender difference is not surprising taken into consideration that such a gender difference is also found in the normal population (American Psychiatric Association, 2000). However, the gender differences found in the present study stand partly in contrast to the findings of Pigott et al. (1991), who did not indicate the same tendency. Their study showed that, when looking at a sample of OCD patients, there were only significant gender differences on two of the subscales: Women scored significantly higher than men on *Body Dissatisfaction*, while men scored significantly higher than women on *Interpersonal Distrust*.

### **The Effect of Eating Disorders on Treatment of Obsessive-Compulsive Disorder**

As mentioned, comorbid eating disorders did not seem to influence the treatment outcome for patients with OCD treated with ERP. The patients achieved reductions in symptoms of both OCD and depression. Furthermore, comorbid depression before treatment did neither seem to influence the treatment outcome for OCD. Taken into consideration what was mentioned in the introduction, separating between severity levels of depression could possibly have led to different results (Abramowitz, 2004). One can also, highly speculatively, ask whether separating between severity levels of eating disorders could result in similar tendencies as when separating between severity levels of depression. In addition, it would be interesting to see whether eating disorders and/or depression could influence the relapse rate of OCD.

One reason for wondering whether, specifically, severe levels of eating disorders could influence the treatment of OCD, is the assumption that malnourishment can reduce the patient's ability to benefit from psychotherapy (Bulik et al., 2007). However, the EDI does not assess whether the patients are malnourished, it measures psychological and behavioural traits common in eating disorders (Garner, 1983). That being said, if the patients had suffered from a severe eating disorder and were malnourished, they would most likely have a primary diagnosis of eating disorders rather than OCD, and would have received treatment accordingly.

It appeared that symptoms of eating disorders were more related to depression than to obsessive-compulsive disorder. Pre-treatment symptoms of eating disorders seem to be correlated to depression both before and after treatment. People who are depressed tend to



remember more negative than positive material (Matt, Vázquez, & Campbell, 1992). In accordance with this notion, it is possible that depressed patients often report their symptoms strongly on different psychiatric inventories. This might be one explanation to why eating disorders seem more closely related to depression than to OCD.

### **The Effect of Treatment for Obsessive-Compulsive Disorder on Eating Disorders**

Numerous controlled studies have found that manual based treatment for one specific disorder can influence symptoms of another disorder (for an overview, see Wilson, 1997), however such a tendency is not found in the present study. The EDI subscale scores used in this study were relatively stable between the beginning of treatment and after treatment. However, the total EDI scores changed significantly between pre-treatment and post-treatment, with the post-treatment scores being the lowest. This could suggest that ERP-treatment for OCD also reduces symptoms of eating disorders. Nonetheless, a more plausible explanation for this reduction is that the scores on the other EDI subscales (*Ineffectiveness*, *Perfectionism*, *Interpersonal Distrust*, *Interoceptive Awareness*, *Maturity Fears*) are reduced. Since these subscales are included in the total EDI scores, this change may reflect the reduction of psychopathology related to OCD or other psychiatric disorders.

As mentioned, Oljatunji et al. (2010) found that treatment of eating disorders can influence comorbid OCD. They further observed that changes in symptoms of OCD only partly explained changes in eating disorders, while changes in eating disorders fully explained changes in symptoms of OCD. The researchers suggest that the reason for this may be that the patients underwent therapy for eating disorders, rather than for OCD. In the present study, however, there were no significant changes in symptoms specific for eating disorders after treatment for OCD. There are some important differences between the study of Oljatunji et al. (2010) and the current study. Firstly, the studies focus on opposite diagnoses. Secondly, the first-mentioned study only used the total EDI-III (a different version of EDI, but which also includes general psychological subscales) as a measure of eating disorder symptoms, while the present study also looked at the first three EDI subscales, which are specific for eating disorders.

### **Methodological Considerations**

As with most research this study has some methodological weaknesses, and could be improved in different ways. One point of improvement is the number of participants, which is relatively low. If the study had included more patients, the statistical power of the study could

have been stronger. Another limitation of this study is that it does not include follow-up data, and thus cannot examine if OCD patients with comorbid eating disorders have a higher relapse-rate than OCD patients without comorbid eating disorders. Further, the study does not have a control group to which we could compare the prevalence scores; such a comparison group would have made it easier to see the EDI scores for the normal population. To compensate for this we reviewed the EDI scores from another study (Kjelsås & Augestad, 2004).

Another weakness of this study is that some patients received ERP-treatment in groups while others received the ERP-treatment individually. Even though the two treatments contained the same fundamental elements, there is some uncertainty to whether the findings in the present study are specific for one or the other. Moreover, a review article indicates that, despite the efficacy of cognitive behavioural therapy for both groups and individuals, slightly poorer results for treatment in groups are reported (Jónsson & Hougaard, 2009). This tendency is important to take note of, seeing as a majority of the patients in this study underwent ERP-based group therapy. Considering this, the present study did some comparisons between the groups, and found that individual treatment was moderately more effective than group treatment. Furthermore, the patients that participated in the individual treatments had somewhat lower indications of eating disorder symptoms. That being said, the main aims of this study were to look at general tendencies: Prevalence of eating disorders in patients with OCD and how eating disorders affect treatment for OCD.

Another possible pitfall in this study, which generally applies to studies of epidemiology in clinical populations, is sampling bias (Altman & Shankman, 2009). This bias, also called Berkson's bias, occurs because patients with multiple disorders tend to have more symptoms, and thus are more likely to be referred for treatment (Berkson, 1946). Thus, the findings of comorbidity may be overestimated as a result of sampling bias.

Furthermore, data from self-report forms such as the EDI, can be biased in different ways, by for instance social desirability, extreme or moderate response styles, and response bias. However, the EDI has proved to be valid in several ways (Garner et al., 1983). Further adding to the strength of the present study is that the other measures used, BDI (Beck et al., 1979) and Y-BOCS (Goodman et al., 1989a, 1989b), also are standardized, and found to be reliable and valid. Self-report forms also hold the strengths of being economical, easy to administer, and time efficient.

Since the EDI is not a diagnostic instrument, another point of improvement could be to use other, or additional measures, of eating disorders. Moreover, a limitation of this study is

that it does not specifically separate between the different types of eating disorders when examining the prevalence of eating disorders among patients with OCD. Besides, future research should also examine whether there are distinctions in how the different types of eating disorders affects the treatment of OCD.

An additional reason for including other measures of eating disorders is that EDI is a self-report measure, and, as mentioned, certain biases may influence the results. By including a measure administered through interviews one could possibly get other results. However, a strong relationship has been found between studies using interviews and studies using self-report forms (Mann et al., 1983 as cited in Kjelsås & Augestad, 2004). Additionally, the EDI is a widely used instrument; it is especially valuable when administered at different points in time, and it provides information about clinical status and response to treatment (Garner, 1990). Furthermore, the EDI has been used for similar research purposes earlier, and there are therefore established cut-off scores (e.g. Meltzer et al., 2001; Torstveit et al., 2008). In addition to assessment with Y-BOCS, EDI, and BDI, all of the patients were assessed with a diagnostic interview, SCID, which adds to the strength of this study.

### **Conclusion and Clinical Implications**

The present study confirms that there is a relatively high occurrence of comorbid OCD and eating disorders, and therefore we need to pay attention to this patient group. The significance of this is further emphasized by the other results in the present study showing that ERP-treatment for OCD did not lead to improvement of eating disorder symptoms. Moreover, other researchers also underline this importance with their research showing a higher prevalence of suicide attempts in this patient group (Sallet et al., 2010).

This study showed that the effect of treatment for OCD on patients with comorbid eating disorders was similar to the effect of treatment for OCD on patients without comorbid eating disorders. Symptoms of OCD were significantly reduced, and symptoms of eating disorders did not seem to affect the treatment response. However, seeing as the symptoms of eating disorders persisted, the discussion on how to treat comorbid psychiatric disorders becomes important. As mentioned introductorily, a recent study was conducted on a multimodal treatment program for patients with comorbid eating disorders and OCD (Simpson et al., 2013). This study found that the multimodal treatment program was efficient, and that patients had significant reductions in both symptoms of eating disorders and OCD. Comparing the results of the multimodal treatment in the Simpson et al. (2013) study to the results in the present study, it is evident that both types of treatments lead to significant

reductions in OCD, with high effect sizes. However, it should be noted that the effect size was a bit higher in the present study than in the multimodal treatment program. On the other hand, while the present study found no changes in symptoms of eating disorders, the results of the multimodal treatment showed that such symptoms were significantly reduced, and the effect size of this was relatively high.

As these researchers point out, since the study had a naturalistic design, they cannot determine which elements of the treatment led to reduction of eating disorders and OCD (Simpson et al., 2013). They further emphasize that, although other researchers have found that treatment for eating disorders can reduce symptoms of OCD (Olatunji et al., 2010), it is not evident that such treatment can do the same when the OCD is as severe as in their own sample.

It is important to consider the different meanings of comorbidity when trying to understand the importance of comorbidity to treatment planning (Kendall & Clarkin, 1992). According to Kendall and Clarkin (1992) this is important because one will intervene in different ways if the two comorbid disorders have a common underlying etiology or a causal relation between them, than if the disorders are independent and only co-occur. The patients in the present study had a primary diagnosis of OCD. For individuals with comorbid eating disorders and OCD it is often recommended that the most prominent disorder should be treated first, especially if the patients' compulsion is related to eating and thus maintains the eating disorder (Fairburn, 2008). An article, providing case illustrations, suggests treating patients' anxiety first would be easier, since they may lack motivation for treating their eating disorder (Becker, Zayfert, & Pratt, 2010). Although, as underlined by these authors, medical problems related to eating disorders may need immediate intervention, and should, if so, be treated first. A clinical implication of the present study is that patients are able to utilize treatment for OCD, regardless of having comorbid eating disorders or not. However, another implication is to pay attention to possible difficulties related to eating disorders, and, if required, to give treatment specifically directed towards these issues.

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## Appendix

Table

*Studies on the prevalence of eating disorders in obsessive-compulsive disorder*

Study	N	Country	Measurement	ED (%)	AN (%)	BN (%)
Prabhu et al., 2012	253	India	OCD assessment form; M.I.N.I Plus	0		
Grabe et al., 2000	61	Germany	EDI, SADS-LA-IV	Sig. higher scores than 288 normal controls		
Tamburrino et al., 1993	31	United States	Y-BOCS; Survey, incl. history of ED	39 <sup>a</sup>		
Pigott et al., 1991	59	United States	EDI	Sig. higher scores than 60 normal controls		
Sallet et al., 2010	815 <sup>c</sup>	Brazil	SCID-1; Y-BOCS, DY-BOCS	2.0		2.1
Pinto et al., 2006	293	United States	SCID I; The Butler Hospital OCD Database; Y-BOCS	0.0/ 3.1 <sup>a</sup>		1.0/ 3.1 <sup>a</sup>
LaSalle et al., 2004	334	United States	SCID	9.3 <sup>a</sup>		9.6 <sup>a</sup>
Rubenstein et al., 1992	62	United States	SCID	9.7 <sup>a</sup>		4.8 <sup>a</sup>
du Toit et al., 2001	85	South Africa	SCID; Y-BOCS; SCID-OCSD	2.4/ 5.9 <sup>a</sup>		3.5/ 4.7 <sup>a</sup>
De Mathis et al., 2008	330	Brazil	Y-BOC; SCID	3.3		2.7
Torresan et al., 2013	858	Brazil	DY-BOCS; SCID-1	3.0 <sup>b</sup>		3.1
Kasvikis et al., 1986	280	England	Review of case record	6 <sup>acd</sup>		
Fahy et al., 1993	105	England	Review of case records	11.0 <sup>ac</sup>		

*Note.* ED = eating disorders; AN = anorexia nervosa; BN = bulimia nervosa; M.I.N.I Plus = MINI International neuropsychiatric Interview Plus, version 5.0; EDI= Eating Disorder Inventory; SADS-LA-IV= Schedule for Affective Disorders and Schizophrenia – Lifetime version modified for the study of anxiety disorders; Y-BOCS = Yale-Brown Obsessive Compulsive Scale; DY-BOCS = Dimensional Yale-Brown Obsessive-Compulsive scale; SCID = Structured Clinical Interview for the DSM-IV Axis 1 disorders; SCID-OCSD= SCID for obsessive-compulsive spectrum disorders.

<sup>a</sup> Lifetime prevalence.

<sup>b</sup> In this study 4.6% of the women and 0.8% of the men had anorexia, while 4.6% of the women and 1.1% of the men had bulimia.

<sup>c</sup> These study only assessed the patients for anorexia.

<sup>d</sup> The prevalence for women was 11%, while none of the men in the sample had anorexia.

<sup>e</sup> This study also measured binge-eating disorder, and found a prevalence of 7.2%.