The Prevalence of Comorbid Obsessive-Compulsive Disorder in Individuals with Eating Disorders: An Epidemiological Meta-Analysis

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Abstract

The present study aimed to provide a meta-analytic estimate of the prevalence of obsessive-compulsive disorder (OCD) amongst those with a current primary eating disorder (ED) diagnosis, and to isolate predictors of comorbid OCD. An online search of *PubMed* and *PsycINFO* was conducted with a Boolean search phrase incorporating keywords related to *OCD*, *EDs*, *comorbidity*, *prevalence*, and *epidemiology*, complemented by reference review and contact with experts in the field. Articles were included if they (a) reported an observational study examining current ED diagnoses, (b) used a semi-structured or structured diagnostic interview for OCD and ED diagnosis, (c) applied *DSM* or *ICD* criteria, (d) included adolescent or adult samples (age > 12), (e) included patient or community samples, and (f) reported lifetime or current OCD comorbidity. From the 846 articles identified, 35 lifetime and 42 current estimates of comorbid OCD were calculated. Analyses revealed an aggregate lifetime OCD prevalence of 13.9% $CI_{95\%}$ [10.4 to 18.1] and current OCD prevalence of 8.7% $CI_{95\%}$ [5.8 to 11.8] across EDs. Moderator analyses revealed the prevalence of OCD to be greatest in anorexia nervosa binge-eating purging type, in patient versus community samples, among those with an earlier age of ED onset, and those with lower mean BMIs. Awareness of the populations at greatest risk of comorbid OCD in EDs will ensure appropriate diagnosis, while facilitating development of treatments targeting their shared etiology.

Keywords: obsessive-compulsive disorder, comorbidity, eating disorder, anorexia, bulimia, binge eating

General Summary

This research project investigated the prevalence of obsessive-compulsive disorder (OCD) in those diagnosed with an eating disorder (ED). The main objectives of this research included conducting a review of the literature examining OCD in EDs while providing a true estimate of OCD comorbidity using data from previously conducted studies. Furthermore, risk factors were identified to determine when those with an ED are the most vulnerable for experiencing OCD. Using a meta-analytic approach, the past findings of multiple studies that focused on the same question were combined to calculate accurate estimates for the frequency of OCD in EDs. A search of research databases found 846 potentially relevant articles, making it possible to calculate 35 lifetime and 42 current estimates of OCD in EDs. Following combination of the findings, lifetime OCD prevalence was approximately 13.9% and current OCD prevalence was 8.7% across EDs. The highest rates of OCD were found among individuals with anorexia nervosa binge-eating purging type, in patient samples, among those diagnosed with an ED earlier in their life, and those with a lower body weight. Greater awareness of which EDs are more vulnerable for experiencing OCD is important to be able to diagnose and treat patients effectively when they have more than one mental health diagnosis.

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The Prevalence of Comorbid Obsessive-Compulsive Disorder in Individuals with Eating Disorders: An Epidemiological Meta-Analysis

Eating disorders (EDs) and obsessive-compulsive disorder (OCD) have been found to be highly comorbid, with similar underlying symptoms, traits, and characteristics implying a potential shared vulnerability. Understanding comorbid disorders is important because living with comorbid psychiatric or physical health conditions has a detrimental impact on the prognosis of all presenting conditions, exacerbating symptoms and diminishing treatment efficacy (Sartorious, 2013; Swinbourne & Touyz, 2007). For instance, the co-occurrence of an anxiety disorder (inclusive of OCD in past versions of the *DSM)* in those receiving inpatient care for anorexia nervosa (AN) is associated with longer hospital stays (Lievers et al., 2008). Further, the likelihood of attempting suicide is already heightened in those with eating disorders (Ahn et al., 2019; Udo et al., 2019) and is exacerbated further amongst those also coping with OCD (Torres et al., 2006). As a result, this leads to an elevated risk of ED relapse amongst those with greater severity of OCD symptoms (Carter et al., 2004).

While past literature has illustrated that co-morbid OCD commonly occurs in EDs, estimates to date are inconsistent and there is a need for proper estimation of the prevalence of their co-occurrence. This is evidenced throughout the literature with low prevalence estimates of comorbid OCD in ED samples of 1.9% (Striegel-Moore et al., 2001) to moderate rates such as 29.5% (Milos et al., 2001) and higher estimates extending beyond 42.9% (Hsu et al., 1992). Quantification of how common comorbid OCD truly is across ED subtypes is vital to understand who is most at risk for OCD as it has been shown to predict poorer ED treatment response and worse prognosis (Altman & Shankman, 2009). Therefore, this research is imperative to inform the potential

development of new interventions and the refinement of current treatments for those with comorbid OCD in EDs.

The advancement of assessment, diagnosis, and treatment of those with comorbid OCD with primary ED diagnoses relies upon an improved understanding of a biopsychosocial model for EDs with the presence of comorbid OCD. Increased empirical support for shared genetic, biological, and psychosocial etiological factors is imperative to understand the underlying mechanisms driving symptom exacerbation, disorder maintenance, and experiencing further burden with OCD. The following literature will paint a picture of obsessive-compulsive disorder, OCD symptom classification, and review OCD prevalence across a variety of contexts. Next, the refinement of ED diagnostic criteria will be summarized per ED followed by examination of the prevalence of EDs. Lastly, utilizing a biopsychosocial framework the intricacies of shared etiological and transdiagnostic factors between OCD and EDs will be examined.

Defining and Assessing Obsessive-Compulsive Disorder

Obsessive-compulsive disorder is a chronic condition characterized by the manifestation of obsessions, such as intrusive thoughts or images (e.g., fears one has become contaminated by germs), coupled with compulsive behavioural responses intended to reduce the distress caused by those obsessions (e.g., repetitive handwashing; Abramovitch et al., 2021; APA, 2013). The *DSM-5* obsessive-compulsive disorder (OCD) classification defines an obsession as the presence of persistent thoughts or urges causing clinically significant levels of distress followed by an attempt to suppress or counterbalance the urge by engaging in a compulsion (e.g., having taboo sexual thoughts causing extreme discomfort followed by behaviours to achieve order and symmetry; Abramovitch et al., 2021a). In contrast, a compulsion is illustrated by engaging in repetitive behaviours often guided by a set of informal ritualistic rules to minimize exacerbated anxiety from

the intense obsessions (APA, 2013). Obsessions or compulsions persist for a prolonged time and cause substantial impairment in one's daily functioning. For example, an individual may experience an intense urge to harm someone they care about followed by engaging in compulsive behaviours such as repeating words silently for a set number of times to reduce the considerable discomfort (Abramovitch et al., 2021b).

The debilitating nature of this condition often bears detrimental consequences for the psychosocial and economic well-being of those afflicted, including reduced quality of life, lower instances of marriage, loss of employment, and carryover effects onto family members (Calvocoressi et al., 1995; Hollander et al., 2016; Koran et al., 1996; Leon et al., 1995; Magliano et al., 1996; Pozza et al., 2018; Rasmussen & Eisen, 1992; Samuels & Nestadt, 1997). These influences are intensified when OCD is comorbid with other psychiatric conditions, with comorbidity linked to OCD symptom exacerbation, notable decline in daily functioning, and poorer prognosis (Pinto et al., 2006; Swinbourne & Touyz, 2007; Torres et al., 2013).

The *Diagnostic and Statistical Manual of Mental Disorders* (*DSM)* is widely used by clinicians to guide assessment and diagnosis of a variety of psychiatric conditions by providing descriptions outlining diagnostic features that permit diagnosis of a particular condition (e.g., characteristic patterns of behaviours or specific types of thoughts). As a result, over or underestimation of the prevalence of anxiety-related disorders and differential categorization of ED subtypes is bound to occur given changes in the diagnostic criteria with each new version of the *DSM*. Over the past few decades, each edition of the *DSM* has been further refined, thus prevalence may be influenced by the diagnostic criteria that is currently being employed at the time of the publication, the presence or assessment of comorbid disorders, and whether dual diagnoses were acceptable at the time of the assessment (e.g., the *DSM-III-R* did not allow for

diagnosis of simultaneous anxiety-related disorder diagnoses, therefore, clinicians would have been faced with choosing a primary anxiety diagnosis over another).

Employment of the *DSM-5* diagnostic criteria also enables clinicians to note whether an individual can determine whether their obsessions are illogical or logical by specifying whether the individual expresses: (1) good insight where they can recognize on their own that the intrusive thoughts are not very likely to be accurate, (2) poor insight meaning they believe that their thoughts may be feasible and plausible, or (3) a complete absence of insight as evidenced by full belief that the enduring obsessions are accurate (APA, 2013). For example, an individual who has washed their hands three times may genuinely believe that their hands are still contaminated by germs and lack the awareness to challenge this intrusive thought.

Obsessive-compulsive symptoms often parallel symptoms observed in other psychiatric disorders, including anxiety (e.g., high levels of worry followed by adamant reassurance seeking; Kobori & Salkovskis, 2012), eating (e.g., hedonic eating for food-driven reward to regulate emotions; Monteleone et al., 2017), or substance use disorders (e.g., experience of high anxiety sensitivity followed by substance use to reduce the intensity; Cuzen et al., 2014). Therefore, it is critical to determine that symptom expression is not due to another condition or a pharmacological or addictive substance side effect (APA, 2013).

OCD Symptom Classification and Assessment. Numerous assessment measures have been developed to capture the heterogeneity and themes across OCD symptoms. McKay and colleagues (2004) reviewed the many plausible symptom categorizations derived from the following validated psychometric measures of the presence or severity of OCD symptoms: the Maudsley Obsessional Compulsive Inventory (MOCI; Hodgson & Rachman, 1977), Padua Inventory (PI; Sanavio, 1988),

and the Yale–Brown Obsessive Compulsive Scale symptom checklist (YBOCS-SC; Goodman et al., 1989).

The MOCI is one of the earliest validated measures of OCD symptom expression which utilizes three distinct symptom clusters: washing, checking, and doubting/conscientiousness. Next, the PI was constructed to examine more "repulsive" or taboo urges, which ultimately support the possibility of five symptom clusters, including washing, checking, rumination, impulses, and precision (McKay et al., 2014; Sookman et al., 2005). As the identified symptom domains from factor analyses of the MOCI and PI were only inclusive of the behaviours noted in the instruments' items, future investigations began to move towards evaluating the Y-BOCS checklist for assessment of OCD. The Y-BOCS provides a more comprehensive assessment of a broader range of obsessions and compulsions obtained through a semi-structured interview with a clinician than the previously explored self-report measures (McKay et al., 2014).

The Y-BOCS-SC supports clinician assessment of 60 OCD symptoms resulting in the classification of symptoms into 15 unique categories. The presence of obsessions was now able to be grouped into one of eight types of obsessions: contamination, symmetry, hoarding, aggressive, sexual, religious, somatic, or miscellaneous. Subsequently, compulsive behaviours were categorized into seven classes: washing, checking, counting, ordering, hoarding, repeating, and miscellaneous (Goodman et al., 1989). There is no consensus to date on the best way to subclassify OCD symptoms but Abramowitz et al. (2003; 2010) recommended categorizing OCD into four symptom dimensions that more accurately capture the range of mental compulsions.

The *Dimensional Obsessive-Compulsive Scale* (DOCS; Abramowitz et al., 2010) assesses OCD symptoms based on four domains: (a) worry about contamination (e.g., feeling one has been in contact with germs followed by repetitive washing to get rid of the germs; Abramovitch et al.,

2021a), (b) responsibility for harm (e.g., the belief that one's actions were responsible for endangering someone else followed by compulsive checking to prevent the harm believed to occur), (c) intrusive taboo thoughts (e.g., thinking about sexual behaviours with an unacceptable person followed by avoidance of the individual to prevent triggering more intrusions; Abramovitch et al., 2021b; Sookman et al., 2005), and (d) the need for symmetry or completeness (e.g., thinking the books on your shelf are not in order followed by re-arranging the books until they are displayed perfectly or feel "just right"; Abramovitch et al., 2021a; 2021b).

OCD Prevalence. The prevalence of psychiatric conditions such as OCD fluctuate depending on the measurement window and the measures used. OCD prevalence is represented by the proportion of individuals in a population who meet all diagnostic criteria outlined above for a specified period of time. Prevalence is commonly divided into two rates: (1) point prevalence, which represents having met diagnostic criteria at a single point in time; and (2) period prevalence, which is defined by a specific period of interest (e.g., having experienced OCD in the last month or 12 months; Bhopal, 2002; Carroll, 2013). In clinical epidemiological research, however, a rate for lifetime prevalence is more widely reported as an extension of period prevalence as it refers to having met diagnostic criteria for a disorder such as OCD at any point in one's lifetime.

Bhopal (2002) discusses the core difference between the prevalence windows with respect to the breadth of time that is captured and the associated disadvantages of the timeframe not encompassed. Bhopal (2002) and Carroll (2013) define point prevalence as a rate that symbolizes the frequency of cases of a disease or disorder at a specific point in time, which is advantageous as it provides a snapshot of a specific moment in time but fails to be useful in determining the long-term impact of a disorders course or prognosis. In contrast, period prevalence rates aim to

address the concern with point estimates by accounting for a larger duration of time that one may meet diagnostic criteria for a disease or disorder (Bhopal, 2002).

The most all-encompassing estimate in clinical epidemiology is the lifetime prevalence rate; however, with the use of a longer interval between diagnosis and recall the greater the risk for errors in recall. Retrospective reporting of past experiences of having met diagnostic criteria for a disorder brings a high likelihood for underestimation (Streiner et al., 2016). Streiner and colleagues (2016) argue that forgetting and error in recall likely distort any available lifetime estimates reported in literature as they rely on recall of the past which is subject to biases (e.g., the tendency to recall the past in a manner that is in favour of oneself). Furthermore, lifetime estimates fail to account for prevalence changes in response to increased availability of or decreased access to efficacious psychiatric services (Moffitt et al., 2020). Streiner and colleagues (2016) encourage future research to place a greater focus on current measurements (e.g., point prevalence or 1 month period prevalence) to decrease the impact of errors in recall and inflated estimates. An astonishing example of the impact of errors in recollecting the past is illustrated by Moffitt et al. (2020), who compared prospective estimates of *DSM* disorders in a birth cohort from New Zealand to retrospective reports over the same 15-year period between the ages of 18 to 32 years. Moffit and colleagues (2020) found nearly double the prevalence for rates for every disorder in prospective versus retrospective reports. The findings of this study highlight how retrospective reports can drastically underestimate lifetime prevalence and the authors hypothesize this may be due to increased memory for chronic disorders as opposed to forgetting past acute symptoms that were experienced for a shorter duration.

Although lifetime prevalence may be clinically useful to aid in determining the commonality of coping with a disorder throughout one's lifetime, current prevalence brings

potential for greater accuracy and can account for factors that may contribute to increased or decreased prevalence. For example, Taylor and Asmundson (2020) discuss how the current pandemic is likely to bring an increase upwards of 10% in psychiatric disorders inclusive of anxiety and related disorders following the global COVID-19 pandemic like other natural disasters. As a result, by using point prevalence we can determine at a specific moment in time whether OCD prevalence has increased with consideration of the current societal climate. Similarly, period prevalence may be markedly higher in 2020 or beyond for a duration of time whereas, the lifetime rate would simply remain inflated as it encompasses one's entire life regardless of historical factors.

OCD prevalence can vary depending on the specified period measured, as well as the gender and age of the samples. A recent meta-analysis by Fawcett and colleagues (2020) illustrates how the prevalence of OCD globally varies depending on the specified period. Their aggregate prevalence estimates for the general population revealed OCD was most prevalent during the lifetime window (1.3%), followed by the current window (1.1%), and period timeframe (0.8%). Whereas the literature shows that in pediatric samples OCD is more common in boys than girls (Mathes et al., 2019), this shifts in adolescence, and in adult samples women are at 1.6 times greater risk for being diagnosed with OCD than men in their lifetime (Fawcett et al., 2020). Gender also affects OCD symptom expression, with males more commonly reporting taboo thoughts, while females more commonly experience contamination or aggressive obsessions (Mathes et al., 2019). Finally, the potential for experiencing increased vulnerability for OCD was also found among younger adults than those in older adulthood (Fawcett et al., 2020).

Among clinical samples, the rate of OCD comorbidity significantly increases contingent upon the presence of co-occurring conditions. In a meta-analysis conducted by Ferentinos et al.

(2020) on comorbid OCD among those with bipolar spectrum disorders, lifetime and current prevalence was approximated at 10.9% and 11.2%, respectively. OCD in the general population as assessed in control samples was estimated as 2.5% for lifetime and 1.6% for current prevalence which also aligns with the global prevalence aggregated by Fawcett et al. (2020). As a result, those with bipolar spectrum disorders were 4.4 times more likely to experience comorbid OCD than the general population, further depicting the substantial increase in OCD prevalence when comorbid with another psychiatric condition (Ferentinos et al., 2020). The prevalence of OCD across ED subtypes has also been examined extensively, often exhibiting higher rates of comorbidity in AN samples than in BN samples (Altman & Shankman, 2009; Sallet et al., 2010; Simpson et al., 2013; Swinbourne & Touyz, 2007).

The Evolution of Eating Disorder Diagnosis

Newer editions of the *DSM* made substantial changes to ED criteria, and the prevalence of anorexia nervosa has been shown to change considerably depending on whether *DSM-III-R* diagnostic criteria or *DSM-IV* criteria were applied (Sunday et al., 2001). The introduction of the *DSM-III-R* enabled clinicians to derive simultaneous anxiety or mood-related disorder diagnoses, which was not possible in the previous edition of the *DSM* (Swinbourne & Touyz, 2007). The inception of the *DSM-III-R* provided a more thorough description of comorbid psychiatric disorders informing clinicians about the possibility of observing increased prevalence of comorbidity. This was especially the case among those working with ED populations (e.g., increased incidences of OCD among those with anorexia nervosa and bulimia nervosa; Braun et al., 1994).

The publication of the *DSM-IV* brought further revisions to existing diagnostic criteria; however, this time core behavioral and psychological manifestations were grounded by empirical

evidence. Furthermore, *DSM-IV* brought forth sizeable changes in how eating disorders were classified with the inclusion of supplementary ED subtypes (e.g., anorexia nervosa [AN] or bulimia nervosa [BN], restricting [AN-R, BNNP] or binge-eating/purging type [AN-BP, BNP]; Sunday et al., 2001). An overview of acronym definitions for ED diagnoses and subtypes is outlined in Table 1. Previously, individuals with AN who presented with binge eating and purging behaviours would be provided with a dual diagnosis of anorexia nervosa and bulimia nervosa, but with the inception of the *DSM-IV* criteria, a combined diagnosis was no longer permitted (Sunday et al., 2001). Instead, if the individual was underweight, the diagnosis was AN (i.e., either AN-R or AN-BP).

The *DSM-IV* classification of psychiatric disorders included an ED subsection; however, the implementation of the *DSM-5* also introduced a more inclusive name known as "Feeding and Eating Disorders." The *DSM-5* feeding and eating disorder criteria changes integrated more representative and observable eating behaviours for each eating disorder. Furthermore, the updates provided a more comprehensive classification system to support an accurate depiction of diagnosis and to encourage new research investigating clinicians' utilization of specific empirically supported interventions for particular patterns of disordered eating and cognitions. This update also brought alterations across criteria used for all eating disorder diagnoses to reduce the frequency that eating disorder not otherwise specified (EDNOS; e.g., diagnosis assigned to those who present with significant disordered eating but do not meet full criteria for other ED diagnoses) was diagnosed. EDNOS was substituted with the integration of avoidant restrictive food intake disorder (ARFID), unspecified feeding or eating disorder (UFED), and other specified feeding or eating disorder (OSFED) to bring greater clarity to subthreshold disordered eating behaviour

(APA, 2013; Lindvall et al., 2016). The remainder of this section will describe each of the ED diagnoses in greater detail.

Table 1

Overview of ED Subtypes and Acronyms.

Note. Descriptions informed by the *DSM-5* (APA, 2013).

Eating Disorder Subtype	Acronym	Defining Characteristics
Anorexia Nervosa	AN	Extreme food restriction leading to a significantly low body weight while accompanied by an intense fear of weight gain or body image distortion. May also engage in excessive exercise.
Anorexia and Bulimia Nervosa	ANBN	Dual diagnosis of anorexia nervosa and bulimia nervosa (classification only used in studies employing the *DSM-III-R*).
Anorexia Nervosa Binging/Purging Type	ANBP	Meets diagnostic criteria for AN and also reports regular binge eating and/or purging behaviours (e.g., self-induced vomiting and/or misuse of diuretics).
Anorexia Nervosa Restricting Type	ANR	Meets diagnostic criteria for AN but does not report regular binge eating and/or purging behaviours (e.g., vomiting, misuse of laxatives or diuretics).
Bulimia Nervosa	BN	Recurrent binge eating (i.e., eating an abnormally large amount of food, given the context, accompanied by a sense of loss of control) and compensatory behaviours to offset the calories consumed during a binge. Overconcern about weight and shape while weight is typically within the normal range.
Bulimia Nervosa Non-Purging Type	BNNP	Recurrent episodes of binge eating followed by non-purging compensatory behaviours to offset the calories consumed (e.g., fasting or excessive exercise).
Bulimia Nervosa Purging Type	BNP	Recurrent episodes of binge eating followed by purging behaviours to compensate for the ingested food to prevent impact on weight or shape (e.g., self-induced vomiting, misuse of laxatives or diuretics).
Binge Eating Disorder	BED	Recurrent binge eating (i.e., eating an abnormally large amount of food, given the context, accompanied by a sense of loss of control) and in the absence of compensatory behaviours such as purging, fasting or excessive exercise. Weight is typically above the healthy weight range.
Eating Disorder Not Otherwise Specified	EDNOS	Individuals with clinically significant eating disorder symptoms who fail to meet strict DSM criteria for AN, BN or BED (e.g., recurrent purging behaviour in the absence of binge eating; subthreshold AN where despite significant weight loss, weight remains within the normal range). In the DSM-5, EDNOS is referred to as Other Specified Feeding and Eating Disorder (OSFED) and Unspecified Feeding and Eating Disorder (UFED).

Anorexia Nervosa. There are common themes across feeding and eating disorders such as adherence to unhealthy eating patterns and various preoccupations concerning one's body or psychological response to food consumption. Despite the significant overlap across symptom expression, there are noteworthy differences that are solely characteristic of only certain eating disorders. Anorexia nervosa (AN) is characterized by extreme food consumption restriction, fear of gaining weight, and significantly low body weight. Individuals with AN tend to be preoccupied with their weight or shape, experience significant distress or impairment in their lives, and often fail to acknowledge the severity of their behaviours or the detrimental consequences on physical and mental health (APA, 2013). AN presenting in the last three months can presently be further dichotomized as either AN-restricting type (AN-R) as represented by excessive restriction of nutritional intake in the absence of binge-eating or behaviours to get rid of the food previously consumed. In contrast, AN-binge-eating/purging type (AN-BP) is exemplified when individuals also engage in binge-eating and/or purging behaviour to counteract the calories that have been consumed (APA, 2013).

Bulimia Nervosa. Bulimia nervosa (BN) is characterized by frequent binge eating episodes involving the consumption of an objectively large amount of food given the context and feeling a loss of control over one's consumption (APA, 2013). Individuals with BN also regularly engage in behaviours to compensate for binge eating. Further, they evaluate their self-worth in accordance with their shape or weight. A BN diagnosis requires that binge eating episodes and compensatory behaviours occur at least once a week for a minimum of 3 months. BN also has two subtypes that can be specified: (1) BN purging type (BNP), where compensation occurs through self-induced vomiting or misuse of laxatives/diuretics; or, (2) BN non-purging type (BNNP), where the compensatory behaviours include fasting or excessive exercise but do not involve purging (APA,

2013). Individuals with BN are often within a normal weight range in comparison to those with AN who are underweight.

Binge Eating Disorder. Engagement in frequent binge eating episodes is also the hallmark of another eating disorder referred to as binge eating disorder (BED). The binge-eating episodes occur in an isolated period of time with food intake that is out-of-context for the elapsed duration of time and accompanied by loss of control over eating (Brownley et al., 2016). To meet diagnostic criteria for BED, the binge eating episodes must also include at least three of the following: quick-paced food consumption, eating beyond a point of comfort, hedonic eating, consumption in the absence of others, and feeling embarrassed or guilty for eating a sizable quantity of food in one sitting (APA, 2013). The remaining criteria include clinically significant levels of distress, binge eating episodes at least once a week over three months, and the absence of purging or compensatory behaviours (e.g., as observed in BN). The severity of BED is defined on a continuum based on the frequency of binge eating episodes per week ranging from mild (1-3) to extreme (14 or more) binge eating episodes (APA, 2013).

***Specified and Unspecified Eating Disorders*.** One of the core hopes of restructuring the *DSM-5* feeding and eating disorder section was to reduce the use of EDNOS diagnosis. To achieve this goal, BED was modified to be an ED diagnosis and EDNOS was replaced with more descriptive possibilities to differentiate between individuals not meeting full ED diagnostic thresholds. The *DSM-5* introduced ARFID as a new categorization to represent individuals who avoid or restrict food intake leading to significant weight loss, inadequate nutrition, dependence on food supplements, and impaired daily functioning. ARFID can only be diagnosed when the lack of eating is not due to cultural practices or pre-existing health conditions and exerts no influence on weight or shape perception (APA, 2013). ARFID is utilized when a clinician cannot or does

not identify why the individual fails to meet full threshold criteria for other EDs. When individuals present with feeding or eating disturbances at a subthreshold level, they now meet classification with an UFED. Lastly, OSFED is assigned when there is an expression of characteristic disordered eating patterns and marked distress with specification of how the individual fails to meet full threshold criteria for an ED (APA, 2013). For example, in atypical anorexia nervosa (AAN), all AN diagnostic criterion are met except that, despite significant weight loss, weight is still within the normal weight range (Sawyer et al., 2016).

Prevalence of EDs. The prevalence of EDs, like OCD, can be influenced by various factors including the prevalence measurement window and genders examined. A meta-analysis conducted by Galmiche and colleagues (2019) examining the prevalence of EDs between 2000-2018 revealed a lifetime aggregate estimate inclusive of all EDs of 8.4% for women and 2.2% for men. Further estimation of the prevalence of current EDs was calculated, resulting in a prevalence of 5.7% for women and 2.2% for men (Galmiche et al., 2019). Regardless of the prevalence, men were more likely to report overeating whereas women exhibited greater loss of control once eating had begun alongside preoccupation with body size, fasting, and compensatory behaviours like purging (Striegel-Moore et al., 2009). Moreover, it is important to keep in mind that the prevalence of EDs among males is often underestimated (Gorrell & Murray, 2019). When examining ED prevalence among a transgender sample, individuals assigned female at birth were at greater risk of an ED than those assigned as male (Deimer et al., 2018). This further highlights the importance of considering both sex and gender to truly identify when an individual may be vulnerable for experiencing an ED with comorbid OCD.

Shared Etiology and Personality Traits

Overlap between OCD and EDs is evidenced by the obsessive and compulsive tendencies commonly displayed by individuals with EDs (e.g., preoccupation with body weight and shape, body checking, food rituals or compulsive weighing). However, an additional diagnosis of OCD is only given if the individual also exhibits obsessions and compulsions unrelated to eating or weight (APA, 2013). Despite differences in how obsessions and compulsions are experienced across the disorders, the function of the compulsive behaviours in both EDs and OCD is to reduce levels of apprehension, anxiety, and overall negative affect associated with obsessive thoughts (Altman & Shankman, 2009; Swinbourne & Touyz, 2007). The obsessive-compulsive symptom dimensions of contamination obsessions and cleaning compulsions have previously been identified as the most commonly exhibited OCD symptoms among those with comorbid OCD and EDs (Hasler et al., 2005). When examining a network model of obsessive-compulsive symptoms and ED symptoms, fear of weight gain and caloric restriction exhibited the most robust connections for ED symptoms and intrusions by obsessions was identified as the most crucial bridge symptom for OCD (Meier et al., 2019). Bridge symptoms refer to psychosocial factors or behaviours that link a group of characteristic symptoms associated with EDs with a group of symptoms associated with OCD. In the case of comorbid OCD and EDs, intrusions were identified by Meier et al. (2019) as the most prominent symptom linking the experience of both disorders. Next, observed behaviours and personality traits in OCD and EDs will be explored to further illustrate how the disorders are intertwined across of a breadth of psychosocial factors.

Ego-syntonic and Ego-Dystonic Behaviours. The obsessive and compulsive behaviours experienced by those with OCD or EDs can be further categorized as either syntonic or dystonic suggesting a common underlying vulnerability for both disorders. When a behaviour is classed as

ego-syntonic, the thoughts align with one's personal values and do not deviate away from an individual's typical demeanour or personality (e.g., AN patients tend to be unaware of the harm the disordered eating is having on their overall wellbeing, therefore, they do not see being underweight or restrictive eating as something of concern; Joelson, 2016). In contrast, when the behaviour is characterized as ego-dystonic the behaviours or thoughts do not align with an individual's self-schema leading to feelings of disgust and anxiety (e.g., an OCD patient may be self-aware that checking that the door is locked 3 times in a row is not required, therefore, leading to feeling of loss of control or distress as they find the behaviours distressing; Joelson, 2016).

As a result, the obsessional and compulsive nature of EDs typically falls under the ego-syntonic categorization with the exception of binge eating which symbolizes an ego-dystonic behaviour and for OCD within the ego-dystonic domain. For example, AN can be contrasted with OCD based on the ego syntonic nature of the obsessive or compulsive behaviour experienced with no response to antidepressants whereas in BN, the impulsive behaviours (i.e., binge eating and purging) are responsive to a combination of CBT and a variety of antidepressants (Pearlstein, 2002). Binge eating, which is a hallmark of BED, mirrors ego-dystonic OCD compulsions in that the urge overpowers one's ability to refrain for engaging in the behaviour to reduce distress (e.g., an individual has an intense urge to consume a mass quantity of food to feel better after an anxiety provoking situation but is unable to resist acting on the urge; Pearlstein, 2002). Shared psychological features across conditions, central to both OCD and EDs are traits such as perfectionism, neuroticism, conscientiousness, harm avoidance, perceived loss of control, impulsivity, and intolerance of uncertainty (Altman & Shankman, 2009; Dahlenberg et al., 2019; Hoffman et al., 2012; Levinson et al., 2019b; Sallet et al., 2010). Next, the role of common personality factors engrained in OCD and EDs will be reviewed.

Perfectionism. Perfectionism is one of the most frequently discussed personality traits associated with both EDs and OCD and may therefore represent a common underlying vulnerability mechanism. Perfectionism can be defined as upholding high expectations of oneself despite unrealistic attainment (Shafran et al., 2002). Upon review of prevalent personality traits in EDs, high expression of perfectionism has been positively correlated with ED psychopathology across EDs (e.g., AN, BN, BED, and OSFED) with strong resistance to ED interventions. Perfectionism can also be multifaceted, which is evident by the different conceptualizations and assessment measures of perfectionistic traits. For instance, the Frost Multidimensional Perfectionism Scale (FMPS; Frost et al., 1990) is a self-report measure that examines the following six dimensions of perfectionism: concern over making mistakes, high personal standards, feeling doubtful over one's performance or actions, perception of high parental expectations, parental criticism, and the need for organization (Farstad et al., 2016; Frost et al., 1990; Lewis et al., 2013). AN and BN samples are both associated with more significant worry over mistakes and doubt about their behavior compared to other EDs (Farstad et al., 2016).

In another model of perfectionism, The Comprehensive Model of Perfectionistic Behaviour (Hewitt et al., 2017), perfectionism is sub-classified into three domains often measured by the Multidimensional Perfectionism Scale to assess for the presence of self-oriented, other-oriented, and socially prescribed perfectionism (MPS; Hewitt & Flett, 1991; Hewitt et al., 2017). Hewitt and colleagues (2017) have found that perfectionism amongst those with EDs is significantly more likely to be self-oriented or socially prescribed, than other-oriented. Using Hewitt and Flett's model of perfectionism, self-oriented perfectionism is particularly linked to those with AN, compared to other EDs (Benson, 2003).

A crossover between OC and ED symptoms has been observed in those with OCD who engage in ritualistic eating patterns. Ritualistic eating behaviour has been associated with more severe OCD and greater impairment (Jassi et al., 2016). As a result, perfectionistic personality traits present as a risk factor for the development of EDs, especially in AN and OCD. For example, Machado et al. (2014) compared a group of women with AN to healthy controls finding significantly elevated rates of perfectionism in AN accompanied with negative attitudes about weight or shape of others and themselves prior to the onset of AN. When greater concern over mistakes was experienced by those with AN, greater severity of both AN and comorbid OCD has also been observed (Levinson et al., 2019). EEG studies support the notion that a part of this impairment is attributable to what Robinson and Abramovitch (2020) coined as "obsessive slowness" where excessive monitoring or preoccupation with checking for perfectionism hinders cognitive functioning. Even in those without OCD, excessive checking over one's work out of fear of mistakes leads to a slower rate of productivity as preoccupation may takeover until things feel "just right." These findings exemplify the potential for perfectionism as a precursor and risk-factor for the development of AN, but also EDs and OCD more broadly.

Neuroticism, Conscientiousness, and Harm Avoidance. Pollack and Forbush (2013) demonstrated from their dimensional modelling using a hierarchical multiple regression approach that neuroticism and perfectionism mediate the relationship between obsessive-compulsive symptoms and ED symptoms. The persistence component of perfectionism tends to be observed more frequently in AN and BN than BED (Atiye et al., 2015). Furthermore, AN is also associated with the highest rates of harm avoidance which involves an intense response to aversive stimuli (Atiye et al., 2015) followed by those with BED (Peterson et al., 2010) whereas, BN tends to be characterized more by novelty seeking and negative emotionality (Peterson et al., 2010).

Another personality trait that is commonly observed across OCD and EDs is conscientiousness which involves completion of tasks in a meticulous manner. Kotov et al. (2010) conducted a meta-analysis examining the relationship between personality traits and common psychiatric disorders and discovered that conscientiousness provided the second strongest correlation with psychiatric disorders (including OCD). However, it is important to keep in mind the impact of conscientiousness on OCD severity remains ambiguous with past findings demonstrating heightened beliefs of responsibility (Manos et al., 2010) and others who have discovered it exerts no influence on OCD manifestation at all (Wetterneck et al., 2011). With regards to EDs, significantly greater scores for conscientiousness using the *NEO Five Factor Inventory* for personality traits has been observed in those with ANR than ANBP (Bollen & Wojciechowski, 2004). As a result, it is likely the case that those who are high in neuroticism or conscientiousness as observed in those with OCD and AN may also be more prone to experience higher rates of harm avoidance.

Perceived Level of Control. Central to both conditions is the desire for greater control (i.e., obsessions) or feeling a complete loss of control over behaviour (e.g., compulsive behaviours). Restriction of food consumption as seen across EDs can be viewed as an act to garner greater control and binge eating in BN or BED exemplifies how the loss of control may be expressed in EDs. In response to contextual factors in the environment, a similar competition exists between the desire to have more control in the future than the actual level of control one currently has over their behaviour is believed to stimulate compulsive responses in OCD (Froreich et al., 2016). Higher ED and OC symptom expression is associated with a lower sense of control, feelings of inadequacy, and greater fear of losing control (Froreich et al., 2016). Froreich and colleagues (2016) found that 20-30% of the variance in ED and OCD symptomology was explained by fear

of losing control of oneself and that this predicted their co-occurrence more than any other perceptions of one's competence (e.g., feeling inadequate). As a result, this presents more evidence supporting the notion that EDs and OCD may share underlying concerns with a desire for greater control.

Impulsivity. Paralleling the need for control is the strong correlation between impulsivity in both OCD and EDs. Higher levels of impulsivity have been observed in clinical samples of both OCD and EDs than in the general population with the potential for the lack of inhibitory control to present as a deficit in controlling one's behaviours (Boisseau et al., 2012). Impulsivity is characterized by five core tenants: negative urgency, positive urgency, lack of awareness of behaviour, sensation seeking, and difficulty with task completion (Farstad et al., 2016). Negative urgency, defined as acting rashly when experiencing negative emotions, is the only facet of impulsivity that plays a role across all EDs and presents as a risk factor for EDs and comorbid OCD (Farstad et al., 2016). Impulsivity, in general, is positively associated with compulsivity, which refers to the exertion of excessive control over one's behaviour. Impulsive risky decision making has been observed more robustly in those with EDs than those with OCD who tend to make less risky choices (Boisseau et al., 2013). Individuals who score higher on impulsivity and compulsivity indices demonstrate more severe ED psychopathology, highlighting the similarity to obsessive-compulsive symptomology (Engel et al., 2005). Therefore, impulsivity is another possible underlying mechanism that may be perpetuating symptom maintenance in both OCD and EDs.

Intolerance of Uncertainty. Lastly, intolerance of uncertainty is another factor implicated across both conditions. Intolerance of uncertainty is motivated by a drive to find certainty and difficulty coping with unpredictable or ambiguous situations. Individuals who possess a high

intolerance of uncertainty will have a low tolerance of situations when the ambiguity cannot be resolved and will in turn feel significant levels of distress (Jacoby et al., 2013). Intolerance of uncertainty in OCD has primarily been observed in those who experience obsessions stemming from immense doubt about their actions and compulsions involving checking (Jacoby et al., 2013; Sarawgi et al., 2013). In OCD, intolerance of uncertainty serves as a precipitating factor that stimulates feelings of anxiousness and compulsions (such as checking behaviour) to reduce the distress caused by the current situation's level of ambiguity (Gentes & Ruscio, 2011; Reilly et al., 2021).

In EDs, a comparable pattern has been observed with greater intolerance of uncertainty in AN as opposed to BN. This uncertainty is predominantly linked to concern about one's body size and extreme motivation to strive for the thin ideal in the beginning, however, when the ED persists the obsessions transition to fixation on controlling one’s weight or fear of weight gain. For EDs, restriction of food consumption serves as a compulsive behaviour to mitigate the feelings of increasing discomfort when one is dissatisfied with their body (Kesby et al., 2017; Konstantellou et al., 2019). Kesby and colleagues (2017) have also proposed that binging, purging, and repetitive evaluation of one's body may also represent different disordered eating behaviours trying to attenuate the heightened distress from the intolerance of uncertainty. This proposed theory parallels what is observed in the bi-directional relationship between obsessions and compulsions as observed in OCD. Therefore, intolerance of uncertainty has been postulated as another underlying mechanism perpetuating symptom maintenance in both OCD and EDs.

Elicitation of Obsessive-Compulsive Symptomology via Semi-Starvation

As compensatory behaviours or restriction of food intake are commonly observed across EDs, it is also important to explore the impact that changes to the gastrointestinal system and semi-

starvation have on the risk of experiencing comorbid OCD. The gut microbiome and endocrine system have also been identified across disorders as key biological contributors for perpetuating symptom expression in EDs and OCD. Extreme caloric restriction and intermittent cyclic eating patterns observed (such as binge eating after experiencing psychological pain followed by food restriction until a stressor presents again) in EDs greatly influence alteration of the composition of gut microbes and cells that form the gut microbiome (Butler et al., 2021). The overall health of the gastrointestinal tract is dependent on the type of nutrients consumed and the frequency of dietary changes exerting an influence on overall metabolic functioning (Butler et al., 2021; Skowron et al., 2020). The interplay between eating patterns, diet, and the gut microbiome directly affects the weight and shape of individuals with EDs by altering body composition in response to eating patterns (Butler et al., 2021).

A recent study investigating the role of the gut microbiome in AN inpatients found that alpha diversity (such as the variety and concentration of different bacteria in the biome) was correlated with more severe ED psychopathology, depression, and preoccupation with weight or shape (Kleiman et al., 2015). Furthermore, richness of healthy bacteria in the gut microbiome continually reduced as those with AN reached a healthy weight suggesting that alpha diversity in those with AN contributes to symptom exacerbation and may even be a predisposing factor (Kleiman et al., 2015). Disruption of the gut microbial balance in AN patients has been linked with negative affect, altered eating patterns, and found to exert an influence on weight gain via changes in body composition as well as neuroendocrine functioning (e.g., impaired regulation of hormones such as serotonin as modulated by the gut-brain axis; Skowron et al., 2020; Turna et al., 2016).

Similarly, the health of the gastrointestinal system has been implicated in the pathogenesis of OCD. There is growing evidence supporting the notion that serotonin reuptake inhibitors (SRIs)

reduced obsessive-compulsive behaviours in mice studies and in a similar fashion using a pre-treatment with probiotics diminished the severity of obsessive-compulsive symptoms (Turna et al., 2016). In animal behaviour research with mice, consumption of probiotic supplements for a minimum of 30 days was correlated with an overall reduction in obsessive-compulsive tendencies (Kantak et al., 2014). Therefore, disruption of the gastrointestinal system is implicated in the development of both EDs and comorbid OCD stemming from restrictive caloric intake and nutritional deficiencies caused by the eating behaviours linked to EDs. Therefore, it may be that common biological factors explain the high comorbidity between these conditions as disrupted by the disordered eating patterns.

Disruption of the gut microbiome has been identified as a biological mechanism that influences exacerbation of both ED and comorbid OCD symptom severity, however, it is also feasible for obsessive-compulsive symptoms to be elicited even in individuals without an ED if a state of semi-starvation has been achieved (Keys et al., 1950; Phillipou et al., 2018; Swinbourne & Touyz, 2007). One of the most influential studies examining the impact of fasting behaviours leading to semi-starvation in healthy individuals without EDs was conducted by Keys et al. (1950) commonly known as the Minnesota Starvation Study. This experiment involved 3 months of consumption of 3200 kcal of food per day followed by a 6-month period of semi-starvation with consumption of only 1800 kcal (Keys et al., 1950; Kalm et al., 2005). Participants were expected to participate in housekeeping duties, take classes, and walk approximately 35 kilometres a day to burn double the calories consumed putting them into a caloric deficit. Keys and colleagues found as the experiment progressed, the participants were more irritable, had little patience, and began to seek isolation with less motivation to participate in any daily activities. The males also experienced more sensitivity to cold, became more tired, experienced hair loss, and experienced

quick muscle fatigue leading to reduced coordination skills. Cognitively, individuals became increasingly consumed with thoughts pertaining to food and/or eating with undeniable fixation on when they would eat next or how to prolong the rations they had been provided.

One of the largest changes was in psychosocial behaviours including personality as semi-starvation continued with marked improvement throughout the refeeding phase (Keys et al., 1950). As nourishment of the men began to improve, many of the obsessions about food and compulsions to mitigate the intrusive thoughts about eating diminished. As Phillipou and colleagues (2018) explain, as extreme food restriction in healthy individuals or those with AN occurs, inadequate levels of serotonin are maintained as 95% of serotonin production is stimulated by the gastrointestinal tract in the gut microbiome. Even after a 24-hour fasting experiment with young adults with no ED, participants expressed strong cravings for any type of food but particularly sweet nutrient dense food (Cameron et al., 2014). Therefore, it is crucial to for those with EDs to no longer be malnourished in order to tease apart whether the cognitive alterations (such as obsessive and compulsive behaviours) have been maintained due to starvation effects or the presence of comorbid OCD. If the obsessive-compulsive tendencies are primarily driven by semi-starvation, then once a nourished state has been reached symptom improvement should be observed and if behaviours persist, underlying OCD may be present.

Genetic and Neurological Underpinnings of OCD and EDs

Relatively high rates of comorbidity between EDs and OCD may also be expected due to the shared genetic features across these conditions. EDs share substantial genetic overlap with OCD with past research having found a high genetic correlation, particularly with anorexia nervosa (AN; Watson et al., 2019). For example, in a recent evidence-based synthesis Yilmaz et al. (2020) reported high genetic relations between AN and OCD with elevated risk associated with enhanced

neurobiological gene expression. The overlap further extends to familial associations as observed in twin studies examining comorbid OCD in AN (Cederlöf et al., 2015) and shared anomalies in the prefrontal cortex lending to similar underlying mechanisms contributing to the expression of both disorders (Song et al., 2021).

The Role of Neurological Mechanisms. Beyond the significant overlap in obsessive symptomatology between OCD and EDs, parallels in neurobiological factors also exist (Altman & Shankman, 2009; Swinbourne & Touyz, 2007). Individuals who binge and/or purge show more activation while completing the go/no-go task in the bilateral precentral gyri, anterior cingulate cortex, and the middle and superior temporal gyri (Lock et al., 2011). These areas of the brain are associated with control of motor movement, emotion regulation, and speech production as well as comprehension. Contrastingly, individuals who engage in primarily restricting eating behaviours show greater activation in the hypothalamus and right dorsolateral prefrontal cortex while completing the go/no-go task (Lock et al., 2011). These regions of the brain are central to homeostatic regulation, executive control, and memory. Investigation of neural correlates when participants with OCD complete a variety of cognitive tasks (e.g., go/no-go, stop-signal, Flanker tasks, etc.) revealed increased activation in the orbitofrontal cortex, anterior cingulate cortex, and caudate nucleus (Maia et al., 2008).

The activated brain regions in EDs and OCD vary in accordance with the cognitive task being completed, however, stimulation of the anterior cingulate cortex (ACC) is most frequently observed. However, the majority of the activated areas are implicated in inhibitory control processes. In an MRI study of patients with AN, reduced size of the right dorsal ACC was associated with impaired perceptual and conceptualization abilities with normalization of the ACC volume after weight had been restored (McCormick et al., 2008). The ACC has also been identified

as a neurological region with potential to influence emotional affect and is influenced by the extent of control stemming from the limbic-frontal circuitry to mitigate against negative affect (Banks et al., 2007; Stevens et al., 2011). Garcia-Soriano and colleagues (2014) provide support for the role that emotion regulation plays in the maintenance and exacerbation of EDs and OCD, with emotional disturbance predicted by intolerance of uncertainty as well as control importance in OCD (e.g., the urge to gain control over one's behaviours or thoughts) versus thought importance in EDs (e.g., the value attributed or level of significance an individual gives a thought such as perceiving oneself as unhealthily large). Emotional disturbance in both EDs and OCD was best predicted by thought action fusion providing another shared mechanism between the two disorders. Therefore, it is probable that neural connections for inhibitory control may serve as a predictor of ED subtypes when accounting for differences in neurological response in conjunction with expressions of impulsivity as differential activation of regions in the brain have been observed between ANBP and ANR.

Influence of Neurochemicals and Genetics on Comorbidity. From a neurobiological perspective, connections between 5-HT serotonin receptors has consistently been found between EDs and OCD. When high levels of 5-HT serotonin markers are present, restricting and avoidant behaviours are observed but when low levels of 5-HT serotonin markers are discovered, disinhibitory behaviours such as binging/purging are elevated across EDs and OCD (Jarry & Vaccarino, 1996). The genetic overlap between AN and OCD has also been identified in the serotonin and glutamate pathways, which contribute to the overall entanglement between both psychiatric conditions (Mas et al., 2013). Murphy and colleagues (2013) delved into genetic heterogeneity where they found immense overlap amongst genetic mutations in anxiety-related disorders. Empirical support for a number of potential genetic risk factors for the development of

OCD have been identified such as a polymorphism in the serotonin transporter gene, with longer alleles in those with OCD (SLC6A4), genetic mutation to the brain-derived neurotrophic factor gene (BDNF), polymorphism in neuronal glutamate transporters (EEACI, EAAT3) which influence excitatory pathways and neurons related to anxiety-related behaviours (e.g., tics), chromosomal genetic mutations (e.g., I425V increasing risk for OCD by six times), and inheritability through genetic transmission in families (Murphy et al., 2013; Pearlstein, 2002).

In AN and BN, dysfunction of the serotonin pathways also occurs due to a polymorphism of the serotonin transporter (5-HTTLPR). A significant association between the S allele and S carrier genes has been found in AN but neither appear to be correlated with BN (Lee & Lin, 2010). Furthermore, the role of glutamate pathways in the brain also applies to the development of EDs with a significant reduction in overall glutamate levels found in AN (Godlewska et al., 2017). It has been suggested that lower levels of glutamate in EDs may be expected when restriction is involved as the body may utilize amino acids as an alternative source of energy. As a result, it is increasingly evident that the shared etiology between EDs and OCD goes beyond the symptomology of the disorders and also stems from underlying neurobiological factors.

Comorbid OCD in Individuals with Eating Disorders

Given the immense commonalities and shared risk factors between the disorders, it is no surprise there are higher rates of OCD comorbidity in those with EDs than the general populace. The prevalence of comorbid OCD in those with primary ED diagnoses is currently uncertain given inconsistent results. Estimates to date range from as a low as 0% in an adolescent sample of those with AN (Herpertz-Dahlmann et al., 1996) to as high as 79.1% in AN sample comprised of participants with both ANBP and ANR (Halmi et al., 2003). Moreover, comorbid OCD in AN also varies even within a moderate prevalence range with past estimates of 13.6% (Caspi et al., 2016)

to Tseng and colleagues' (2016) rate of 29.6% in their AN sample. A similar pattern is observed in the clinical epidemiological literature exploring OCD comorbidity in BN samples with extreme heterogeneity in prevalence observed with lower rates at 3.8% (Schmidt et al., 2008) to more modest rates of 29.6% (Tseng et al., 2016). The only semi-consistent findings tends to be derived from those examining OCD prevalence in BED with estimates falling at approximately 1% (Wilfley et al., 2000) with gradual elevation to 3.4% (Vardar & Erzengin, 2011).

Variation in these estimates may be influenced by methodological differences across studies, including diagnostic criteria or assessment methods, year of study, mean age of the sample, or the sampling procedure for the population (e.g., community, outpatient, or inpatient sample). For example, in the context of AN, Halmi et al.'s (2003) extremely high prevalence rate was based on an AN sample including family members but Hepertz-Dahlmann et al.'s (1996) report of no comorbidity was obtained from a non-familial sample. Another plausible contributing factor is whether the co-occurrence of OCD in those afflicted by EDs may vary with earlier age of onset and longer durations of living with an ED (Milos et al., 2001; Thornton & Russell, 1997). Similarly, differences in developmental factors such as age may play a role in the variability of reported comorbidity as OCD is more likely to be diagnosed in younger than older samples (Fawcett et al., 2020).

The recruitment of different types of ED samples such as inpatient, outpatient, or community samples may affect reported OCD prevalence. Higher rates of comorbid OCD tend to be observed amongst inpatient ED samples whereas, in community samples prevalence is often lower (e.g., no report of OCD in AN community sample; Hudson et al., 2007). In contrast, research to date has found that OCD comorbidity is greatest in AN patient samples and more prominent in those with AN binge-eating/purging type (ANBP) than those with AN restricting type (ANR; Altman &

Shankman, 2009). Margolis and colleagues (1994) found on average those with ANBP experience 3.8 co-occurring diagnoses in comparison to those with ANR whom met criteria for only 2.3 co-occurring diagnoses.

In ideal circumstances, individuals would seek professional help to assist in coping through the difficult times and it has been found that the prevalence of help-seeking behaviours increase with the presence of comorbid anxiety disorders (Mackenzie et al., 2012). This further illustrates the importance of having adequate care and supports available to those who are living with comorbid psychiatric conditions as they face immense vulnerability to seek care. Experience of comorbid psychiatric conditions including anxiety-related disorders also had an impact on hospitalization leading to prolonged length of stay (e.g., co-occurrence of *DSM-IV* anxiety disorders; Furlanetto et al., 2003). Giving consideration specifically to ED inpatient treatment, co-occurrence of anxiety disorders led to AN inpatients remaining under hospital care longer than those who did not experience a comorbid condition (i.e., *DSM-IV* anxiety disorders categorization included OCD; Lievers et al., 2008).

Psychiatric comorbidity is common in EDs and prevalence of comorbid OCD may also be affected by the type of sample that is studied. Community samples often report lower prevalence rates of OCD comorbidity and in some instances no OCD comorbidity at all for AN samples (e.g., Hudson et al., 2007). Experience of multiple comorbid psychiatric conditions was identified in 22.4% of individuals with a mood or anxiety disorder in a Canadian sample (Meng & D'Arcy, 2012) and 35.4% (Mohammadi et al., 2007) to 62% of individuals with OCD in community samples (Torres et al., 2006). This concern is not solely for OCD populations but also extends to the prevalence of psychiatric comorbidity in EDs. It is increasingly clear that there is immense

variation in comorbidity estimates reported to date where individuals with EDs are at particularly greater risk for comorbid OCD.

The Present Study

Given the heterogeneity in estimates of the prevalence of OCD comorbidity in EDs to date, the goal of the present study was to provide a meta-analytic estimate of lifetime and current OCD comorbidity in EDs using a fully Bayesian multi-level modelling approach. A secondary goal was to isolate predictors of their co-occurrence that could point to shared underlying mechanisms and to identify ED populations most at risk of experiencing comorbid OCD. Although the focus was on samples with a current ED diagnosis, matched control samples or control groups (without a current ED) identified via our literature search were also included if they assessed and reported the prevalence of OCD using the same methodology for diagnosis as employed for the ED sample. This allowed examination of the prevalence of OCD in eating disorders relative to the general populace.

Examining potential moderators of ED and OCD comorbidity is crucial for researchers and clinicians to be aware of whom amongst ED populations may be more vulnerable to experiencing comorbid OCD. The proposed model will evaluate evidence for or against such claims by including – (a) year of publication, (b) geographic region, (c) sample type, (d) diagnostic criteria, (e) diagnostic interview, (f) mean age, (g) age of ED onset, (h) duration of ED, (i) ED subtype, (j) mean body mass index (BMI), and (k) study quality – as potential moderators of the prevalence of OCD in ED in the analyses. The present meta-analysis will examine the moderation of reported comorbid OCD prevalence in EDs across different samples (inpatient, outpatient, community), age ranges, and over time according to different diagnostic interviews and criteria.

Hypotheses

A variety of different exploratory moderators will be evaluated to identify sources of heterogeneity within the reported comorbidity estimates, including the year of publication, country, population, diagnostic criteria, diagnostic interview, mean age, age of ED onset, duration of ED, ED subtype, mean BMI, and study quality. In light of the aforementioned empirical support in the earlier sections exploring the relationship between symptom severity and psychiatric comorbidity, it is hypothesized that the prevalence of comorbid OCD will be greater amongst inpatient or outpatient ED samples compared to community ED samples. Individuals with EDs and OCD are likely to experience greater impairment in many facets of life and to find themselves seeking psychiatric care through inpatient or outpatient programs.

Method

Literature Search

The Preferred Reporting Items for Systematic Review and Meta-Analyses (PRISMA) guidelines were followed. An online search of *PubMed* and *PsycINFO* was conducted using the Boolean search phrase: *("eating disorders" OR "Anorexia Nervosa" OR "Bulimia" OR "Bulimia Nervosa" OR "Binge-Eating Disorder" OR "Binge Eating Disorder" OR "Other Specified Feeding or Eating Disorder" OR "OSFED" OR "Unspecified Feeding or Eating Disorder" OR "UFED" OR "Avoidant Restrictive Food Intake Disorder" OR "Avoidant/Restrictive Food Intake Disorder" OR "ARFID" OR "Eating Disorder Not Otherwise Specified" OR "EDNOS") AND ("obsessive-compulsive disorder" OR "obsessive compulsive disorder" OR "OCD") AND ("epidemiology" OR "prevalence" OR "comorbidity" OR "co-morbidity" OR "comorbidities" OR "comorbid")*. The search was conducted until May 27th, 2019 without date or language

restrictions and was supplemented by articles referenced in the obtained sources and from contact with experts for missing or unpublished data.

Inclusion and Exclusion Criteria

Each article was screened by title and abstract or full-text review to determine study eligibility for inclusion in the meta-analysis. Observational studies (cross-sectional and prospective longitudinal designs) were included if they (a) utilized a semi-structured or structured interview for OCD and ED diagnosis, (b) employed *DSM-III-R* or newer criteria (APA, 1987; APA, 1994; APA, 2013), (c) included an inpatient, outpatient, or community sample, and (d) included adolescents or adults (age > 12). Articles were excluded if they (a) used self-report measures, (b) retrospectively reviewed patient charts, (c) presented a case report, or (d) reported OCD prevalence in special ED populations (e.g., genetic samples with first-degree relatives; see Figure 1).

Genetic studies assessing the prevalence of OCD in those with EDs were only included if comorbidity was assessed in the probands themselves, therefore, when first-degree relatives were evaluated instead, these samples were excluded. Exclusion of first-degree relative ED samples occurred as those with a family history of EDs or OCD are at an elevated risk for experiencing the disorders, thus not being representative of a typical ED sample. For example, Lilenfeld et al. (1998) conducted a family-based epidemiological study where they interviewed probands with EDs, a female control group, and a sample of first-degree relatives of the probands. For our purposes, only the data pertaining to the ED probands were coded and the data for first-degree relatives were not extracted on the grounds of representing a specialized sample known to be at increased risk for EDs and OCD.

When presented with longitudinal studies, the time point that met all inclusion criteria and presented the greatest amount of data was included and the others were excluded for utilization of the same sample. For example, Godart et al. (2003) was included instead of Godart et al. (2006), as the earlier publication provided more informative value with estimates for the sample with a current ED diagnosis whereas, Godart et al. (2006) presented comorbidity estimates based on having met ED diagnostic criteria at one point in their lifetime (thus, many of the sample had recovered for this follow-up assessment no longer meeting current criteria for an ED as required for inclusion). Concerns regarding specific studies were resolved through correspondence with my research committee and conversation with the publications' authors, where feasible.

Data Extraction

The following data from each article were extracted: author name, year of publication, sample size, gender (male, female, mixed), gender composition, ED measure (e.g., Eating Disorder Examination [EDE; Fairburn & Cooper, 1993], Structured Clinical Interview for *DSM-IV* Axis I Disorders [SCID-I; First et al., 2002], Mini-International Neuropsychiatric Interview [MINI; Sheehan et al., 1998]), ED diagnostic criteria (*DSM-III-R*, *DSM-IV*, *DSM-5*, *ICD-10*), OCD measure (e.g., SCID, MINI, Anxiety and Related Disorders Interview Schedule [ADIS; Grisham et al., 2004]), OCD diagnostic criteria (*DSM-III-R*, *DSM-IV*, *DSM-5*, *ICD-10*), age range of sample, mean age, population (inpatient, outpatient, community, mixed, controls), country, mean age of ED onset, mean duration of illness, mean age of OCD onset, mean BMI, and OCD prevalence separated by ED subtype.

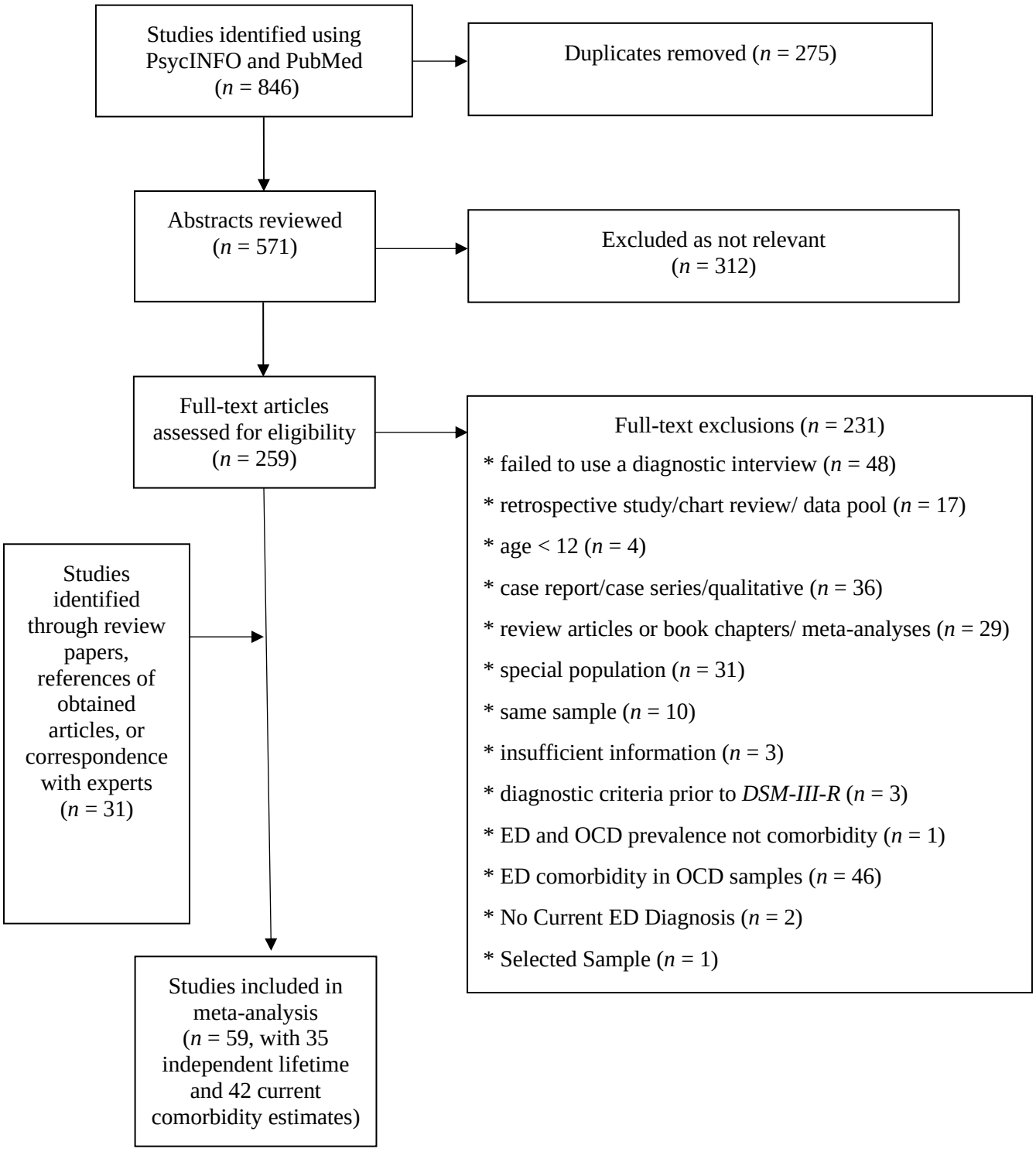

Figure 1. Meta-analysis inclusion flowchart.

The majority of studies clearly reported lifetime prevalence, whereas point and period prevalence estimates are rarely differentiated in clinical epidemiological research. For ease of interpretation, lifetime prevalence was defined as having met diagnostic criteria for OCD at one point throughout their lifetime and current prevalence denoted having met diagnostic criteria in alignment with the assessment window for the structured or semi-structured diagnostic interviews employed (e.g., the SCID defines the current timeframe as a period of one month). If the prevalence windows were not reported in-text or could not be confirmed by the corresponding authors, it was inferred that the co-occurrence of OCD was assessed for the current period. Prevalence of comorbid OCD was also coded and further categorized across EDs as well as by diagnoses (e.g., AN) and subtype (e.g., ANBP) to evaluate whether certain conditions exhibit higher rates of OCD prevalence. Due to changes between the *DSM-IV* and *DSM-5* classification of BED, review of ED subtype classification was reviewed for consistency across estimates and revealed no BED in EDNOS samples. The percent of the healthy control sample diagnosed with OCD, when reported, was also coded.

Quality Ratings of Study Methodology

Study quality was examined using a 10-point assessment, generated in accordance with previous measures for clinical epidemiological research (Fawcett et al., 2020; Hoy et al., 2012). Items were adapted from both measures and two additional items were created to improve applicability to the current clinical epidemiological literature (e.g., reporting of prevalence window, and prevalence by ED subtype). Table 2 provides the items used to assess the quality of each included study along with the scoring guide. Higher scores signified higher quality. Study quality was rated independently by D.H.D. and co-supervisor E.J.F. with 98.8% agreement and with all disagreements resolved through discussion.

Table 2

Measure of Quality Ratings

Item	Scoring Guide	Source
1. Was the population clearly defined with demographic characteristics of the study population? (e.g., age, sex, ethnicity, martial status, education, sample type [community, outpatient, inpatient]).	Not reported in the article/only one or two of the above = **0** Three or more of the above listed = **1**	Adapted from Fawcett et al. (2020)
2. Was the study's sample a close representation of the target population?	Not a close representation of the target population (all one gender or all one ethnicity OR strict exclusion criteria*) = **0** Close representation to the target population (e.g., mix of genders or mix of ethnicities) = **1**	Adapted from Hoy et al. (2012)
3. Was some form of random selection used to select the sample?	Consecutive/convenience sample or not reported = **0** Random sample or two-stage screening method (e.g., a large population is screened and those deemed high risk are interviewed) = **1**	Adapted from Hoy et al. (2012) and Fawcett et al. (2020)
4. Was the likelihood of non-response bias minimal?	The response/participation rate after participants were deemed to meet inclusion/exclusion criteria for the study was < 75%, not reported, or an analysis was performed comparing responders and non-responders showing a significant difference in relevant demographic characteristics = **0** The response/participation rate after participants were deemed to meet inclusion/exclusion criteria for the study was 75% or higher or an analysis was performed comparing responders and non-responders which did not show a significant difference in relevant demographic characteristics = **1**	Adapted from Hoy et al. (2012)
5. Who administered the diagnostic interview?	Trained Lay Person/Not Reported = **0** Trained Clinician/Researcher/Allied Mental Health Worker or Trainee = **1**	Fawcett et al. (2020)
6. Was an acceptable case definition of OCD used in the study?	No/Unclear = **0** Yes = **1**	Hoy et al. (2012)
7. Were data collection methods standardized?	No/Unclear = **0** Yes = **1**	Fawcett et al. (2020)
8. Was the prevalence window for OCD clearly defined?	Inference based on diagnostic measure used and method = **0** Clearly identified in-text (e.g., "lifetime"; specifying "current" (e.g., 1 month)) = **1**	Item created by D.H.D. & E.J.F.
9. What was the length of the prevalence period?	Report of only lifetime OCD prevalence = **0** Report of only current OCD prevalence = **1** Reports both lifetime and current OCD prevalence = **1**	Adapted from Hoy et al. (2012)
10. Was the prevalence of OCD broken down according to ED?	No report of OCD prevalence by ED even if ED subtype data available = **0** Inclusion of OCD prevalence by ED reported/ only 1 ED examined as per study objectives = **1**	Item created by D.H.D. & E.J.F.

*Strict exclusion criteria: studies employing inclusion criteria that was stricter than *DSM* criteria (e.g., extreme weight requirements, prolonged duration of symptoms, or higher frequency of behaviours such as an increased number of binges than normally expected) or studies which excluded almost all other possible health conditions (e.g., illustrates a specialized ED sample as EDs are often comorbid with other health complications).

Statistical Approach

A fully Bayesian multi-level modelling approach was utilized to obtain aggregate estimates of lifetime and current OCD prevalence. The resulting logistic regression models were fit using *brms 2.13.5* (Burkner, 2018) in *RStudio 1.3.1073* (R Studio Team, 2020) with *R 4.0.2* (R Core Team, 2020); because this approach used logistic regression, effect size calculations were not needed prior to fitting the model, as prevalence was estimated in logit-space during the fitting process itself. All models were fit, and convergence was assessed using R-hat <1.01 (Gelman et al., 2014; Gelman & Hill, 2007) as well as visual inspection. Prevalence estimates were then back-transformed into percentages prior to reporting. Past research employing a similar methodological approach as utilized in the present meta-analysis include Fawcett et al. (2020), Fawcett et al. (2019), and Fawcett et al. (2016). Separate models were fit to the lifetime and current data. Each model was run with samples using 4 chains each with 10,000 iterations and a warm-up of 5000 iterations thinned by 1 leading to a total of post warm-up samples equating to 20,000. Random intercepts and slopes were included in each model, as appropriate, to account for heterogeneity across samples and dependency between estimates. Analyses of publication bias were not undertaken as such analyses are not generally considered relevant to epidemiological models (e.g., Borenstein, 2019, p. 173).

In Bayesian modelling, estimates are based on the posterior distribution calculated for each parameter. These posterior distributions reflect previous beliefs concerning plausible values for those parameters after (i.e., posterior to) seeing the data and are derived from the combination of prior beliefs and the data itself. These prior beliefs are quantified as parametric distributions and referred to in Bayesian modelling as priors. The priors utilized for our primary analyses are summarized below. The prior expectations relating to the intercept of the lifetime models assumed

that the average prevalence in a typical sample should range between <1% and 68%. The prior was calibrated for the random effects to permit the "true" prevalence within any given sample to vary anywhere from <1% to 94%. For current models, these ranges were tightened such that the average prevalence in a typical sample should range between <1% and 44% with the "true" prevalence within any given sample varying anywhere from <1 % to 85%. In either case, the prior for slopes within the moderator models was represented by a normal distribution centred at 0 with a standard deviation of 1.

For the analyses of the ED subtypes, the model intercept was removed and priors for each subtype were implemented directly. Here, the prior utilized was described for the intercept in the preceding paragraph, with the exception that the control samples adopted a prior inspired by the posterior from Fawcett et al. (2020). A slightly informed prior was applied to the control groups in all models giving consideration to what expected OCD prevalence would be in an average global female sample as the majority of samples in the present meta-analysis were exclusively female. Fawcett and colleagues (2019) found the prevalence of lifetime OCD to be 1.5% $CI_{95\%}$ [1.0 to 2.1] and current OCD to be 1.1% $CI_{95\%}$ [0.6 to 2.0] for the female population. Therefore, the prior employed on the lifetime models ranging between 6% and 22.3% reflected the average prevalence in a typical female sample. This equates to a normal distribution with a mean of -4.18 and standard deviation of 1.

For current models, the prior employed was slightly tightened compared to lifetime as current OCD prevalence tends to be lower thus the probable prevalence in an average sample for current OCD would be expected to range from 2.3% and 9.5%; this would be represented by a normal distribution with a mean of -4.5 and standard deviation of 1 (Fawcett et al., 2019a). Specifically, for that condition this prior was calibrated to expect that the prevalence in a typical

control sample should range between <1% and 10% or <1% and 7% for lifetime and current estimates, respectively.

The slopes and standard deviations for random effects were fit with a prior consistent with the normal distribution with a mean of 0 and standard deviation of 1, while correlations were fit using a prior based on the LKJ distribution (Lewandowski et al., 2009) with eta set to 4 (which would reduce the likelihood of extreme values). These priors are reflective of the prevalence expected to be seen in an average, predominantly female sample with the belief that the "true" prevalence in any sample may range from <0.1 % to 88.1% for comorbid lifetime OCD and that current OCD ranging from <0.1% to 95.3%.

Heterogeneity was quantified in the initial models using prediction intervals, which estimate the range of plausible "true" prevalence estimates expected in a new sample of similar methods and demographics to those included in the relevant analysis (see Table 3; Inthout et al., 2016). I^2 values were not calculated because they are not strictly meaningful and often misused (e.g., Borenstein, 2017, 2019). I^2 represents the ratio of variability between studies to the total variability (between + within studies) in the data; therefore, it does not quantify between-study variability directly and is also influenced by within-study variability.

Calculation of Odds Ratios, Risk Ratios, and Prediction Intervals

For each moderator model, odds ratios (ORs) were reported as a measure of effect size, reflecting whether individuals in one condition (e.g., studies using the SCID) were at greater odds of experiencing OCD than individuals in the reference condition (e.g., studies using the MINI). Additionally, risk ratios (RRs) were calculated for categorical predictors indicating more directly the greater risk associated with one condition (e.g., those with AN) over the other (e.g., those in the general public). ORs and RRs were calculated directly based on the back-transformed posterior

prevalence estimates. Prediction intervals were also calculated for the lifetime and current aggregate estimates to aid in evaluating variation in OCD prevalence and to further quantify where future observations might fall, assuming similar methods and demographics (Inthout et al., 2016). Greater details about the modelling approach have been outlined in the respective sections corresponding to the principal findings from each model.

Moderator Analyses

Due to variability in reporting of the potential a priori moderators of interest, each moderator was analyzed separately to allow for the maximum number of studies in each sub-sample to be incorporated, thereby maximizing statistical power. For categorical moderators, a minimum of 3 estimates per category were required for inclusion when fitting the model or were otherwise excluded due to the uncertainty inherent in drawing comparisons with so few estimates. For continuous moderators (e.g., age of onset), estimates were centred and scaled prior to fitting the model, with ORs reported for only the slope unless otherwise stated in Table 4.

The following eleven variables were examined as potential moderators: (a) year of publication, (b) geographic region, (c) sample type, (d) diagnostic criteria, (e) diagnostic interview, (f) mean age, (g) age of ED onset, (h) duration of ED, (i) ED subtype, (j) mean BMI, and (k) study quality. Each moderator was modelled individually as a result of the small number of studies and variation in reporting across the variables of interest.

Results

Description of Studies

The search initially identified 846 studies, of which 59 studies were coded (see Figure 1). This process produced 35 estimates of lifetime and 42 estimates of current OCD prevalence among those with a current primary ED diagnosis. Most studies were conducted in North America

(Lifetime: 40%, Current: 33.3%) and Europe (Lifetime: 40%, Current: 59.5%), with a minority in Oceania (Lifetime: 8.6%, Current: 2.4%) and Asia (Lifetime: 11.4%, Current: 4.8%). Figure 2 illustrates where all of the samples were derived from based on the shaded-in countries. Samples were predominantly female (95.9%), with a mean age of 25.6 years, average ED onset at 18 years, mean ED duration of 6.1 years, and mean age of OCD onset at the age of 13. Five included studies were exclusively adolescent ED samples ranging in age from 12 to 18 (e.g., Agras et al., 2014; Fornasari et al., 2014; Rojo-Moreno et al., 2015; Salbach-Andrae et al., 2008; Striegel-Moore et al., 2001), with 4 additional studies having sample mean ages within the adolescent range (e.g., Blachno et al., 2014; Rastam et al., 1992; Schmidt et al., 2008; Stein et al., 2012; see Table 3). All remaining ED samples recruited participants with primary ED diagnoses within the age range of 12 and 60 years.

Figure 2. Global representation of included ED samples. Shaded regions on the map indicate samples included in our lifetime and current models.

Eating disorder diagnosis was derived using a variety of semi-structured or structured clinician interviews in accordance with *DSM-III-R* criteria (27.3%), *DSM-IV* criteria (64.9%), *DSM-5* (5.2%), or *ICD-10* (2.6%) depending on the date of publication. Representation of ED diagnoses across the entire sample was highest for BN (22.7%) followed by AN (15.9%), ANR (14.7%), ANBP (12.3%), BNP (6.7%), BNNP (6.7%), EDNOS (6.7%), BED (6.1%), and lowest for any ED (4.9%). Samples categorized as "any ED" were samples comprised of a variety of ED diagnoses where prevalence of comorbid OCD was reported for the entire ED sample, therefore, these estimates provide an empirical estimate of comorbid OCD in a general ED sample but not by diagnoses. OCD diagnosis was also confirmed through the use of validated semi-structured or structured interviews based on *DSM-III-R* criteria (36.4%), *DSM-IV* criteria (62.3%), or *ICD-10* (1.3%). Quality ratings ranged from 4 to 9 with a mean quality score of 6.2 (*SD* = 1.3, see Table 3). The largest differences between moderate and high scores for quality ratings were predominantly attributable to scoring low on items 2, 3, 4, and 8 (see Table 2).

Table 3

Characteristics of OCD & ED Comorbidity Studies

Publication	Country (Region)	OCD Criteria	OCD Measure	OCD Measurement Window	Sample Type	Mean Age	Mean ED Age of Onset	Mean ED Duration	Mean BMI	Quality Rating	% Female	Prev. Any ED, % (n)	Prev. Control, % (n)
Halmi et al. (1991)	USA (N. America)	*DSM-III-R*	DIS	LT	O	29.0	…	9.6	…	8	100.0	24.4 (45)	6.4 (62)
Halmi et al. (1991)	USA (N. America)	*DSM-III-R*	DIS	CUR	O	29.0	…	9.6	…	8	100.0	13.4 (45)	…
Levinson et al. (2019)[f]	USA (N. America)	*DSM-IV*	MINI	CUR	IO	25.4	…	…	20.3	5	97.2	21.4 (42)	…
Suda et al. (2014)	England (Europe)	*DSM-IV*	SCID	CUR	O	26.8	…	10.0	15.3	4	100.0	20.0 (20)	…
Swinbourne et al. (2012)	Australia (Oceania)	*DSM-IV*	ADIS	CUR	IO	25.2	17.3	7.8	18.8	6	100.0	3.0 (100)	…
Kountza et al. (2018)	France (Europe)	*DSM-IV*	MINI	CUR	IO	29.0	…	…	15.4	5	…	20.0 (30)	…
Kountza et al. (2018)	Greece (Europe)	*DSM-IV*	MINI	CUR	IO	29.0	…	…	15.4	5	…	16.7 (30)	…
Grilo et al. (2009)	USA (N. America)	*DSM-IV*	SCID	LT	C	44.9	25.9	…	37.1	7	76.7	2.7 (404)	…
Grilo et al. (2009)	USA (N. America)	*DSM-IV*	SCID	CUR	C	44.9	25.6	…	…	7	76.7	2.2 (404)	…
Anderluh et al. (2009)	England (Europe)	*ICD-10*	EATATE	LT	IO	27.3	16.7	11.2	18.1	5	100.0	31.6 (88)	…
Salbach-Andrae et al. (2008)	Germany (Europe)	*DSM-IV*	CIDI	CUR	IO	15.1	13.7	1.3	15.3	6	100.0	16.8 (101)	…
Speranza et al. (2001)	France (Europe)	*DSM-IV*	MINI	CUR	IO	19.5	18.1	3.6	18.2	7	100.0	15.7 (89)	0.0 (89)
Speranza et al. (2001)	France (Europe)	*DSM-IV*	MINI	LT	IO	19.5	18.1	3.6	18.2	7	100.0	19.1 (89)	1.1 (89)
Rubenstein et al. (1993)[f]	USA (N. America)	*DSM-III-R*	SCID	LT	O	32.0	…	…	20.5	6	100.0	32.0 (25)	…
Rubenstein et al. (1993)[f]	USA (N. America)	*DSM-III-R*	SCID	CUR	O	32.0	…	…	20.5	6	100.0	32.0 (25)	…
Matsunaga et al. (1999a)	Japan (Asia)	*DSM-IV*	SCID	CUR	O	23.7	19.7	3.8	…	6	100.0	39.6 (53)	…
Blachno et al. (2014)[f]	Poland (Europe)	*DSM-IV*	SADS	CUR	I	14.8	14.9	1.4	14.6	6	100.0	0 (137)	…
Schmidt et al. (2008)	England (Europe)	*DSM-IV*	EATATE	CUR	O	17.6	…	2.6	21.1	7	97.6	8.2 (73)	…
Jordan et al. (2008)[e]	New Zealand (Oceania)	*DSM-III-R*	SCID	LT	O	23.7	17.8	5.2	…	6	100.0	8.4 (188)	…
Iwasaki et al. (2000)	Japan (Asia)	*DSM-IV*	SCID	LT	O	22.3	18.5	3.7	16.5	6	100.0	18.8 (171)	…
Godart et al. (2003)	France (Europe)	*DSM-IV*	MINI	LT	IO	21.0	…	3.7	17.4	7	100.0	18.8 (271)	3.7 (271)
Godart et al. (2003)	France (Europe)	*DSM-IV*	MINI	CUR	IO	21.0	…	3.7	17.4	7	100.0	14.4 (271)	1.8 (271)
Jimenez-Murcia et al. (2007)	Spain (Europe)	*DSM-IV*	SCID	CUR	IO	23.7	17.7	9.2	…	4	100.0	3.3 (60)	…
Milos et al. (2001)	Germany (Europe)	*DSM-IV*	SCID	CUR	IOC	27.4	19.8	8.5	19.3	7	100.0	29.5 (237)	…
Rojo-Moreno et al. (2015)	Spain (Europe)	*DSM-IV*	SADS	CUR	C	…	…	…	…	7	47.8	8.6 (35)	1.6 (927)

Albert et al. (2001) †	Italy (Europe)	*DSM-IV*	SCID	LT	O	27.6	20.4	…	25.5	6	100.0	10.5 (38)	…
Albert et al. (2001) †	Italy (Europe)	*DSM-IV*	SCID	CUR	O	27.6	20.4	…	25.5	6	100.0	10.5 (38)	…
Bellodi et al. (2001)	Italy (Europe)	*DSM-IV*	DIS	CUR	O	22.2	17.5	4.7	…	6	100.0	13.2 (136)	1.4 (72)
Boujut et al. (2012) ‡	France (Europe)	*DSM-IV*	MINI	CUR	OC	28.4	…	…	…	4	100.0	18.3 (169)	…
Brouwer et al. (2009)	France (Europe)	*DSM-IV*	MINI	CUR	O	28.3	…	9.7	19.3	6	100.0	24.4 (29)	…
Thornton & Russell (1997)	Australia (Oceania)	*DSM-III-R*	CIDI	LT	I	…	16.9	…	…	4	100.0	20.5 (68)	…
Powers et al. (1988)§,a, f	USA (N. America)	*DSM-III-R*	SCID	CUR	C	28.8	…	…	22.9	6	100.0	3.3 (30)	…
Keck et al. (1990)§,a	USA (N. America)	*DSM-III-R*	SCID	LT	O	28.0	20.0	…	…	6	100.0	13.4 (67)	…
Keck et al. (1990)§,a	USA (N. America)	*DSM-III-R*	SCID	CUR	O	28.0	20.0	…	…	6	100.0	10.5 (67)	…
Bulik et al. (1996)	New Zealand (Oceania)	*DSM-III-R*	SCID	LT	OC	…	20.0	…	22.9	6	100.0	3.5 (114)	…
Lennkh et al. (1998)	Germany (Europe)	*DSM-IV*	SCID	LT	I	24.0	…	…	18.4	5	100.0	18.2 (66)	…
Lennkh et al. (1998)	Germany (Europe)	*DSM-IV*	SCID	CUR	I	24.0	…	…	18.4	5	100.0	10.6 (66)	…
Lilenfeld et al. (1998)	USA (N. America)	*DSM-III-R*	SADS	LT	IOC	25.0	16.7	…	…	6	100.0	35.6 (73)	5.0 (44)
Matsunaga et al. (1999b)	Japan (Asia)	*DSM-III-R*	SCID	CUR	IO	25.6	20.0	5.5	17.5	6	100.0	33.3 (78)	…
Fornari et al. (1992)	USA (N. America)	*DSM-III-R*	SADS	LT	O	…	…	…	…	7	95.2	42.7 (63)	…
Brewerton et al. (1995)§	USA (N. America)	*DSM-III-R*	SCID	LT	O	28.4	…	…	…	4	100.0	3.4 (59)	…
Thiel et al. (1995)	Germany (Europe)	*DSM-III-R*	Clinician Interview	CUR	I	25.0	…	…	…	4	100.0	36.6 (93)	…
Rastam (1992)	Sweden (Europe)	*DSM-III-R*	Clinician Interview	CUR	C	16.0	14.0	1.6	18.3	8	96.1	0.0 (51)	0.0 (51)
Rastam (1992)	Sweden (Europe)	*DSM-III-R*	Clinician Interview	LT	C	16.0	14.0	1.6	18.3	8	96.1	9.8 (51)	0.0 (51)
Godart et al. (2000)	France (Europe)	*DSM-III-R*	CIDI	CUR	IO	…	17.8	4.9	…	8	96.8	7.9 (63)	…
Godart et al. (2000)	France (Europe)	*DSM-III-R*	CIDI	LT	IO	…	17.8	4.9	…	8	96.8	9.5 (63)	…
Rabe-Jablonska (2003)	Poland (Europe)	*DSM-IV*	MINI	CUR	O	38.2	…	…	…	5	100.0	0.0 (21)	…
Rabe-Jablonska (2003)	Poland (Europe)	*DSM-IV*	Clinician Interview	LT	O	38.2	…	…	…	5	100.0	4.8 (21)	…
Herzog et al. (1992)	USA (N. America)	*DSM-III-R*	SADS	CUR	O	23.4	18.3	6.6	…	8	100.0	2.6 (229)	…
Hsu et al. (1992)	England (Europe)	*DSM-III-R*	SCID	CUR	O	…	17.3	…	…	6	100.0	0.0 (7)	…
Hsu et al. (1992)	England (Europe)	*DSM-III-R*	SCID	LT	O	39.5	…	…	…	6	100.0	42.9 (7)	…
Braun et al. (1994) ¶	USA (N. America)	*DSM-III-R*	SCID	LT	I	24.6	17.5	7.0	…	5	100.0	18.1 (105)	…
Bossert-Zaudig et al. (1993)	Germany (Europe)	*DSM-III-R*	SCID	LT	I	23.1	17.4	7.1	…	5	100.0	4.2 (24)	…
Vardar & Erzengin (2011)	Turkey (Europe)	*DSM-IV*	SCID	CUR	C	17.0	…	…	21.4	9	86.8	1.4 (68)	0 0 (68)
Striegel-Moore et al. (2001)	USA (N. America)	*DSM-IV*	SCID	LT	C	30.5	20.2	…	32.1	9	100.0	3.8 (212)	…

Striegel-Moore et al. (2001)	USA (N. America)	*DSM-IV*	SCID	CUR	C	30.5	20.2	…	32.1	9	100.0	1.9 (212)	…
Wilfley et al. (2000)[a]	USA (N. America)	*DSM-III-R*	SCID	LT	OC	…	…	…	37.1	9	83.0	1.0 (162)	…
Wilfley et al. (2000)[a]	USA (N. America)	*DSM-III-R*	SCID	CUR	OC	…	…	…	37.1	9	83.0	1.0 (162)	…
Schwalberg et al. (1992)	USA (N. America)	*DSM-III-R*	ADIS	LT	IO	26.3	19.7	…	…	6	100.0	15.0 (20)	…
Steiger et al. (2019)[b, f]	Canada (N. America)	*DSM-IV*	SCID	LT	IO	24.2	16.8	7.7	15.2	6	100.0	15.4 (91)	0.0 (45)
Steiger et al. (2019)[b, f]	Canada (N. America)	*DSM-IV*	SCID	CUR	IO	24.2	16.8	7.7	15.2	6	100.0	13.2 (91)	0.0 (45)
Thaler et al. (2013)[c, f]	Canada (N. America)	*DSM-IV*	SCID	LT	IO	25.8	15.2	9.7	22.6	…	100.0	12.0 (276)	…
Thaler et al. (2013)[c, f]	Canada (N. America)	*DSM-IV*	SCID	CUR	IO	25.8	15.2	9.7	22.6	…	100.0	6.5 (276)	…
Caspi et al. (2017)	Israel (Asia)	*DSM-IV*	SCID	LT	I	…	…	…	18.0	6	100.0	12.5 (64)	…
Fichter et al. (2008)	Germany (Europe)	*DSM-IV*	SCID	CUR	O	…	…	…	…	6	100.0	7.7 (65)	…
Fornasari et al. (2014)[e]	Italy (Europe)	*DSM-IV*	SADS	LT	O	15.5	14.0	…	17.0	6	100.0	13.3 (15)	…
Fornasari et al. (2014)[e]	Italy (Europe)	*DSM-IV*	SADS	CUR	O	15.5	14.0	…	17.0	6	100.0	6.7 (15)	…
Levitan et al. (2006)	Canada (N. America)	*DSM-IV*	SCID	CUR	O	26.3	…	…	…	6	100.0	20.0 (165)	…
Machado et al. (2014)	Portugal (Europe)	*DSM-IV*	SCID	LT	IO	20.0	15.2	…	15.1	5	100.0	12.8 (86)	…
Mangweth-Matzek et al. (2010)	Austria (Europe)	*DSM-IV*	SCID	LT	IOC	26.5	…	…	20.2	5	0.0	28.1 (32)	0.0 (43)
Milos et al. (2013)	Switzerland (Europe)	*DSM-IV*	SCID	LT	IOC	28.6	…	9.3	…	5	100.0	31.8 (192)	…
Schneier et al. (2016)	USA (N. America)	*DSM-IV*	SCID	CUR	I	26.9	…	…	…	8	97.0	13.3 (30)	…
Stein et al. (2012)[d]	Israel (Asia)	*DSM-IV*	SCID	LT	I	16.1	…	…	17.1	6	100.0	17.7 (96)	…
Tseng et al. (2016)[e]	Taiwan (Asia)	*DSM-IV*	MINI	LT	O	27.4	19.7	…	21.6	7	88.2	28.8 (288)	0.0 (81)
Agras et al. (2014)[e]	USA (N. America)	*DSM-IV*	SADS	CUR	O	15.3	…	1.1	…	6	89.2	11.4 (158)	…
Lavender et al. (2013)[a,e]	USA (N. America)	*DSM-IV*	SCID	LT	O	25.3	…	…	17.2	8	100.0	15.5 (116)	…
Weider et al. (2015)	Norway (Europe)	*DSM-IV*	MINI	CUR	IO	28.0	…	11.7	18.9	8	85.9	6.2 (81)	…

[†]Sample type (inpatient vs. outpatient) not specified, assumed outpatient, no response from author seeking clarification.
[‡]Approximated prevalence based on in-text data, no response from author seeking clarification.
[§]Required on average at least 3 binge eating episodes per week for the past 6 months (as compared with 2 episodes required by *DSM-III-R)*.
[¶]ED age of onset and duration provided as ranges, thus coded at the midpoint (e.g., 6.8-7.2 coded as 7.0).
[a]Required specific weight criteria for study eligibility.
[b]H. Steiger, PhD (unpublished data, 2015)
[c]H. Steiger, PhD (unpublished data, 2012), quality rating of the publication not assessed as majority of the data was received directly from the author not the publication.
[d]Combined BN and EDNOS together for BN as reported by authors.
[e]Exclusion of Criterion D for AN diagnosis.
[f]Estimated mean BMI based on in-text data reported on majority of the sample.
Note. Weighted averages calculated for mean age, mean ED age of onset, ED duration, and BMI when reported in-text per ED subtype.
Abbreviations: ADIS = Anxiety Disorder Interview Schedule; CIDI = Composite International Diagnostic Interview; C = Community; CUR = current prevalence window; DIS = Diagnostic Interview Schedule; *DSM-III-R* = Diagnostic and Statistical Manual of Mental Disorders 3rd Edition-Revised; *DSM-IV* = Diagnostic and Statistical Manual of Mental Disorders 4th Edition, EDE = Eating Disorder Examination; I = Inpatient; ICD-10 = International Classification of Diseases, Tenth Revision; IO = Inpatient and Outpatient; IOC = Inpatient, Outpatient, and Community; K-SADS = Kiddie

Schedule for Affective Disorders and Schizophrenia, LT = lifetime prevalence window, MINI = Mini-International Neuropsychiatric Interview; O = Outpatient; OC = Outpatient and Community; SCID = Structured Clinical Interview for *DSM*, SADS = Schedule for Affective Disorders and Schizophrenia.

Aggregate Prevalence Estimate of Lifetime and Current OCD Comorbidity

Lifetime prevalence refers to the percentage of participants with EDs who met diagnostic criteria for OCD at some point throughout the course of their life. As depicted in Figure 3, the aggregate lifetime prevalence of OCD in a typical sample was estimated to be 13.9% $CI_{95\%}$ [10.4 to 18.1] and prediction intervals revealed clear heterogeneity, with the estimated "true" prevalence across studies ranging anywhere from 2.5% to 39.9%. Next, a model was fit for the current prevalence window which signifies having met diagnostic criteria for OCD for a specified period at the time of assessment (e.g., within the past month). As illustrated in Figure 4, current prevalence in an average sample was estimated to be 8.7% $CI_{95\%}$ [5.8 to 11.8] with prediction intervals also supporting broad heterogeneity, with the estimated "true" prevalence across studies ranging anywhere from 1% to 37.6%. Lifetime prevalence of comorbid OCD in EDs was higher than for current prevalence; however, this was anticipated as the lifetime window encompasses both the current and past prevalence.

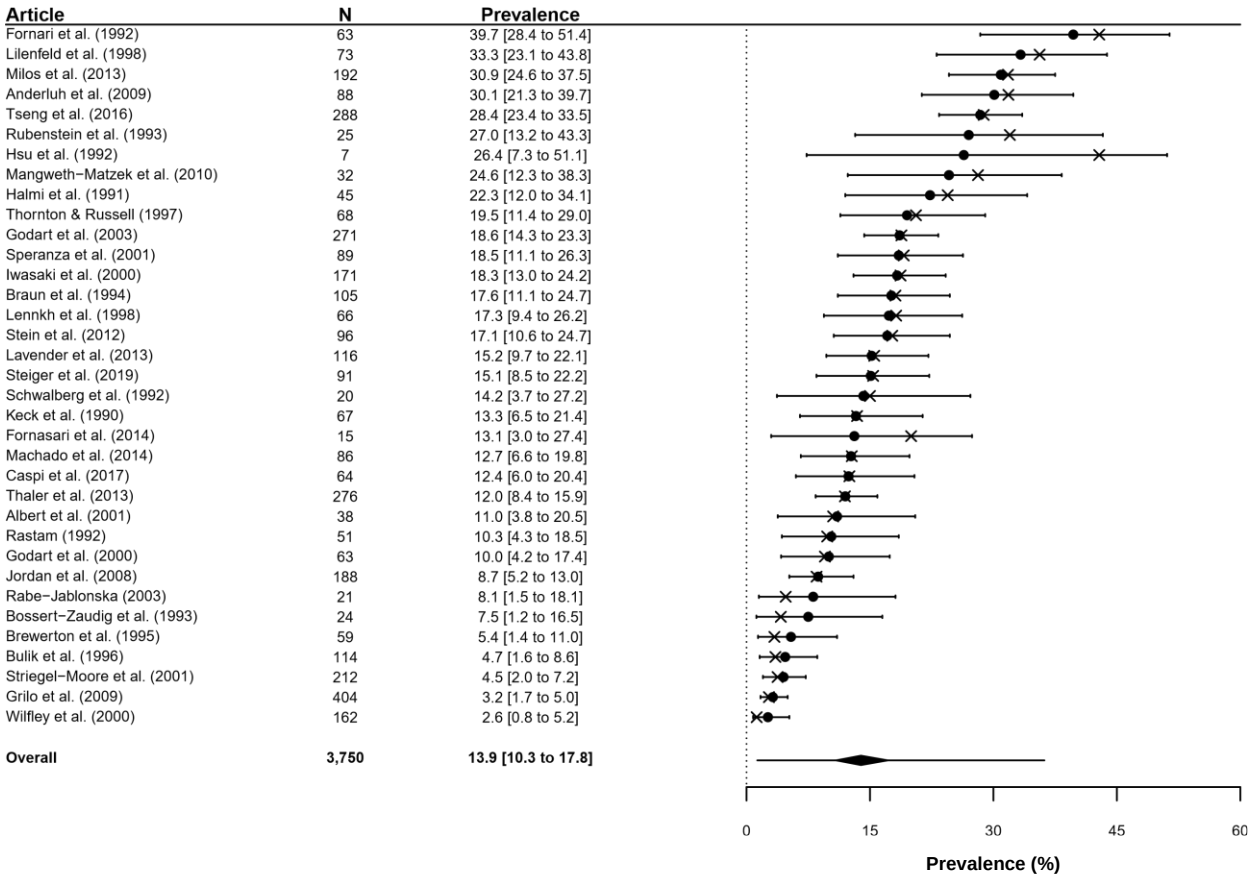

Figure 3. Forest plot representing OCD prevalence (%) for the lifetime measurement window. Points and error bars reflect Bayesian model estimates with the corresponding 95% confidence intervals. The prevalence reported in each publication is represented by an "x" and the aggregate estimate is illustrated by the final entry label "overall." The 95% prediction interval is denoted by the thin black line radiating from the aggregate point estimate.

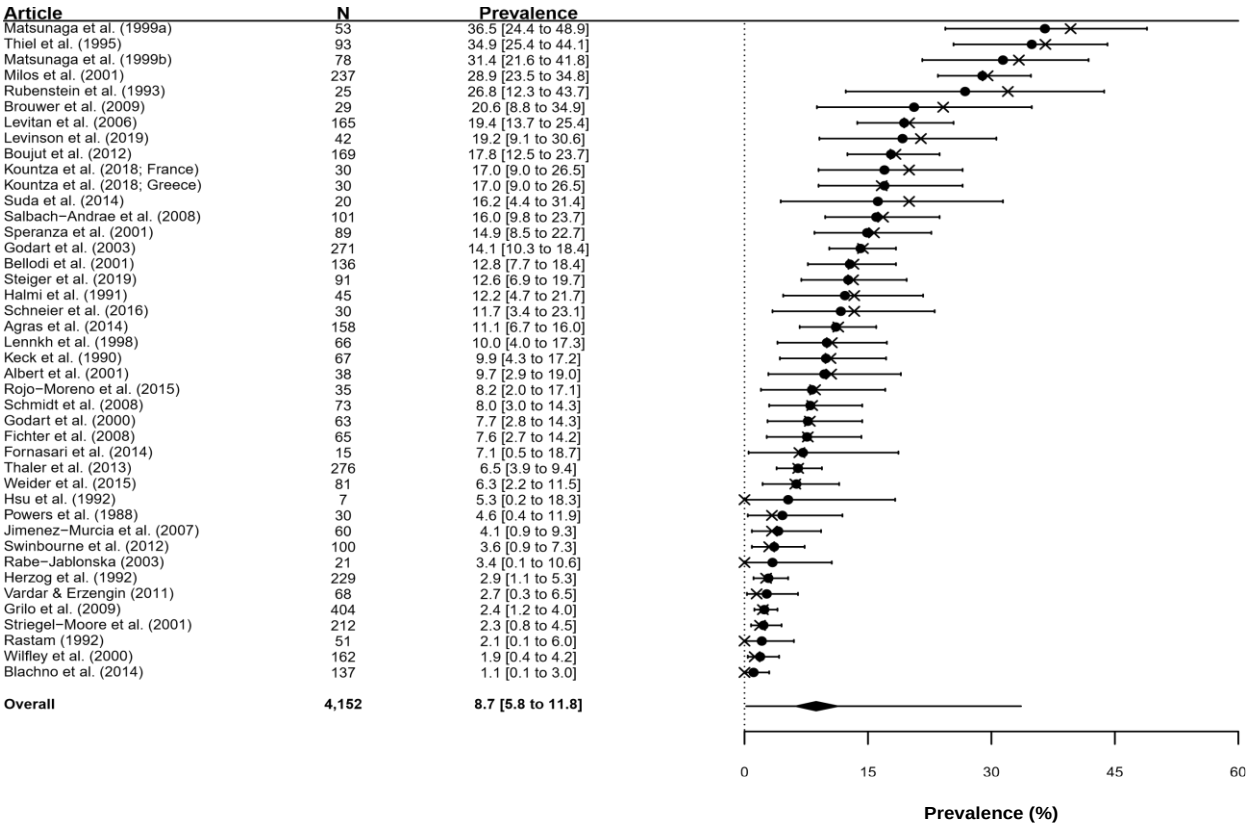

Figure 4. Forest plot representing OCD prevalence (%) for the current measurement window. Points and error bars reflect Bayesian model estimates with corresponding 95% confidence intervals. The prevalence reported in each publication is represented with an "x" and the aggregate estimate is illustrated by the final entry label "overall." The 95% prediction interval is denoted by the thin black line radiating from the aggregate point estimate.

Credible Moderator Analyses

Moderator analyses revealed credible differences or trends related to ED subtype, sample type, age of ED onset, BMI, and quality ratings. No convincing support was found for the remaining moderators, which will be discussed in the following sub-section reviewing all non-credible moderators. Results for *all* moderators are summarized in Table 4.

Table 4

Summary of OCD Comorbidity Moderator Analyses

	Lifetime					Current				
	No. of Estimates	Prev., % (95% CI)	Diff., % (95% CI)	OR (95% CI)	RR (95% CI)	No. of Estimates	Prev., % (95% CI)	Diff., % (95% CI)	OR (95% CI)	RR (95% CI)
Year	35	…	…	1.0 (0.7 to 1.4)	…	42	…	…	1.0 (0.7 to 1.5)	…
Geographic Region										
N. America	14	12.5 (8.2 to 18.6)	…	…	…	14	7.5 (4.3 to 12.5)	…	…	…
Europe	14	15.7 (10.0 to 23.4)	3.1 (-5.0 to 11.8)	0.8 (0.4 to 1.5)	1.3 (0.7 to 2.2)	25	8.8 (5.6 to 13.0)	1.3 (-4.4 to 6.3)	1.2 (0.6 to 2.4)	1.2 (0.6 to 2.2)
Oceania	3	9.5 (3.9 to 21.2)	2.9 (-8.8 to 10.8)	1.4 (0.5 to 3.7)	1.5 (0.5 to 3.3)	…	…	…	…	…
Asia	4	17.7 (8.5 to 32.5)	5.1 (-5.5 to 19.9)	0.7 (0.3 to 1.7)	1.5 (0.6 to 2.8)	…	…	…	…	…
Age	29	…	…	0.8 (0.6 to 1.2)	…	35	…	…	1.0 (0.7 to 1.6)	…
ED Age of Onset	21	…	…	0.7 (0.5 to 1.0)	…	20	…	…	1.2 (0.7 to 2.4)	…
ED Duration (Years)	13	…	…	1.3 (0.9 to 1.8)	…	21	…	…	1.1 (0.7 to 2.0)	…
BMI	21	…	…	0.5 (0.4 to 0.6)	…	26	…	…	0.6 (0.4 to 0.9)	…
AN Only BMI	18	…	…	1.0 (0.8 to 1.4)	…	17	…	…	1.0 (0.6 to 1.7)	…
BN Only BMI	13	…	…	0.8 (0.5 to 1.6)	…	11	…	…	0.4 (0.1 to 1.1)	…
OCD Diagnostic Criteria										
DSM-III-R	17	13.4 (8.7 to 19.5)	…	…	…	11	7.0 (3.5 to 13.0)	…	…	…
DSM-IV	17	13.6 (9.1 to 19.7)	0.2 (-7.3 to 7.7)	1.0 (0.5 to 1.8)	1.0 (0.6 to 1.8)	31	9.4 (6.3 to 13.5)	2.3 (-4.2 to 7.5)	0.7 (0.3 to 1.6)	1.3 (0.7 to 2.8)
OCD Diagnostic Measure										
SCID	22	11.8 (8.5 to 16.4)	…	…	…	20	8.6 (5.3 to 13.4)	…	…	…
MINI	3	19.5 (8.8 to 36.7)	7.6 (-3.7 to 24.7)	1.8 (0.7 to 4.5)	1.6 (0.7 to 3.3)	9	12.2 (6.0 to 22.8)	3.5 (-3.8 to 14.3)	1.5 (0.6 to 3.6)	1.4 (0.6 to 3.0)
SADS	3	25.4 (10.9 to 45.8)	13.4 (-1.2 to 34.0)	2.5 (0.9 to 6.5)	2.1 (0.9 to 4.1)	5	4.4 (1.7 to 11.0)	4.1 (-2.7 to 9.4)	0.5 (0.2 to 1.4)	1.9 (0.7 to 5.3)
Population										
Controls	8	1.6 (0.5 to 3.5)	…	…	…	7	1.1 (0.4 to 2.0)	…	…	…

EDs	72	14.2 (10.6 to 18.5)	12.6 (8.5 to 17.0)	10.3 (4.2 to 35.2)	8.9 (3.8 to 30.1)	89	8.8 (6.2 to 12.1)	7.7 (5.0 to 11.0)	8.7 (4.3 to 26.2)	8.0 (4.0 to 23.7)
Primary Diagnosis										
Controls	8	1.6 (0.5 to 3.4)	…	…	…	7	1.1 (0.4 to 2.0)	…	…	…
AN	31	20.3 (15.9 to 25.0)	18.6 (14.0 to 23.5)	16.0 (6.8 to 52.9)	12.9 (5.7 to 41.9)	39	13.0 (9.2 to 17.0)	11.9 (7.9 to 16.0)	13.6 (6.5 to 43.2)	11.9 (5.9 to 37.2)
ANBN	3	23.1 (5.3 to 54.4)	21.4 (3.6 to 52.8)	19.0 (3.0 to 112.2)	14.5 (2.8 to 61.1)	…	…	…	…	…
Any ED	3	30.1 (15.0 to 44.7)	28.5 (13.2 to 43.1)	26.8 (8.4 to 101.1)	18.9 (6.9 to 64.2)	3	10.0 (2.0 to 29.2)	8.9 (0.0 to 28.1)	10.4 (1.7 to 58.2)	9.4 (1.7 to 43.6)
BN	28	12.1 (8.2 to 17.2)	10.4 (6.1 to 15.6)	8.6 (3.4 to 30.0)	7.7 (3.2 to 25.8)	31	7.0 (3.9 to 10.9)	5.9 (2.7 to 9.8)	6.9 (2.9 to 22.8)	6.4 (2.8 to 21.0)
BED	4	5.9 (1.8 to 18.5)	4.2 (-0.2 to 16.8)	4.0 (0.9 to 21.9)	3.8 (0.9 to 18.7)	6	2.0 (1.0 to 4.0)	1.0 (-0.4 to 3.1)	1.9 (0.7 to 6.9)	1.9 (0.7 to 6.7)
EDNOS	3	16.7 (4.3 to 33.8)	15.0 (2.6 to 32.2)	12.4 (2.4 to 56.4)	10.4 (2.3 to 41.2)	8	4.1 (1.2 to 9.6)	3.0 (0.0 to 8.6)	3.9 (1.0 to 16.1)	3.8 (1.0 to 14.8)
ED Subtype										
Controls	8	1.6 (0.5 to 3.4)	…	…	…	7	1.1 (0.4 to 2.0)	…	…	…
AN	9	19.5 (10.4 to 32.0)	17.9 (8.5 to 30.4)	14.9 (5.1 to 56.7)	12.1 (4.6 to 42.3)	17	9.7 (5.2 to 15.3)	8.6 (4.0 to 14.2)	9.8 (4.0 to 31.3)	8.9 (3.8 to 27.8)
ANBP	10	22.2 (13.4 to 31.7)	20.5 (11.5 to 30.2)	17.3 (6.5 to 62.1)	13.6 (5.5 to 46.0)	10	19.2 (13.9 to 24.7)	18.0 (12.8 to 23.6)	21.4 (10.5 to 65.4)	17.4 (9.0 to 51.9)
ANR	12	18.8 (13.8 to 23.8)	17.1 (11.9 to 22.3)	14.0 (6.1 to 47.0)	11.5 (5.3 to 37.9)	12	14.2 (8.0 to 20.7)	13.1 (6.9 to 19.6)	14.9 (6.3 to 47.7)	12.9 (5.7 to 39.7)
ANBN	3	23.3 (5.4 to 54.3)	21.6 (3.6 to 52.6)	18.9 (2.9 to 115.9)	14.4 (2.7 to 62.9)	…	…	…	…	…
Any ED	3	30.2 (14.7 to 44.8)	28.5 (12.9 to 43.0)	26.0 (8.1 to 98.8)	18.3 (6.6 to 62.8)	3	10.0 (2.0 to 29.0)	8.9 (0.8 to 28.0)	10.4 (1.7 to 53.9)	9.4 (1.7 to 40.7)
BED	4	5.8 (1.8 to 18.6)	4.2 (-0.3 to 16.9)	3.9 (0.9 to 21.2)	3.7 (0.9 to 18.1)	6	2.0 (1.0 to 3.8)	0.9 (-0.4 to 2.9)	1.9 (0.7 to 6.6)	1.9 (0.7 to 6.4)
BN	18	11.8 (7.1 to 18.4)	10.1 (5.0 to 16.9)	8.2 (3.1 to 29.5)	7.3 (2.9 to 25.5)	19	6.4 (3.0 to 11.7)	5.3 (1.7 to 10.6)	6.2 (2.3 to 21.5)	5.9 (2.2 to 19.7)
BNNP	5	15.4 (7.6 to 26.5)	13.7 (5.7 to 24.9)	11.2 (3.7 to 42.3)	9.6 (3.4 to 34.1)	6	9.9 (2.8 to 19.3)	8.8 (1.7 to 18.3)	10.0 (2.4 to 37.7)	9.0 (2.3 to 32.0)
BNP	5	12.0 (6.7 to 19.4)	10.3 (4.7 to 17.8)	8.3 (3.0 to 30.1)	7.4 (2.9 to 25.7)	6	6.9 (3.7 to 12.1)	5.8 (2.5 to 11.0)	6.8 (2.8 to 22.4)	6.4 (2.7 to 20.5)
EDNOS	3	16.8 (4.8 to 34.2)	15.1 (2.9 to 32.6)	12.3 (2.5 to 55.7)	10.3 (2.4 to 40.9)	8	4.1 (1.3 to 9.8)	3.0 (0.0 to 8.7)	4.0 (1.0 to 15.7)	3.8 (1.0 to 14.5)
Sample Type (Collapsed)										
Patient	26	16.5 (12.7 to 19.9)	…	…	…	33	10.6 (7.5 to 14.2)	…	…	…
Community	3	5.0 (2.5 to 10.0)	11.1 (5.1 to 15.6)	3.7 (1.6 to 7.8)	3.5 (1.5 to 6.7)	6	2.7 (1.2 to 6.2)	7.7 (3.4 to 11.7)	4.2 (1.7 to 10.3)	3.9 (1.6 to 9.2)
Sample Type										
Inpatient	6	14.2 (8.2 to 23.1)	…	…	…	5	6.7 (2.7 to 14.5)	…	…	…
Inpatient and Outpatient	8	15.3 (9.6 to 22.9)	1.0 (-9.3 to 10.7)	0.9 (0.4 to 2.0)	1.1 (0.6 to 2.1)	12	11.1 (6.3 to 17.9)	4.2 (-4.4 to 12.2)	0.6 (0.2 to 1.6)	1.6 (0.6 to 4.5)
Outpatient	12	16.6 (10.9 to 23.6)	2.4 (-8.1 to 11.6)	0.8 (0.4 to 1.8)	1.2 (0.6 to 2.2)	16	10.7 (6.4 to 16.4)	3.9 (-4.7 to 10.9)	0.6 (0.2 to 1.6)	1.6 (0.6 to 4.2)
Outpatient and Community	3	5.3 (2.3 to 11.4)	8.7 (0.5 to 18.1)	3.0 (1.1 to 8.2)	2.7 (1.1 to 6.8)	…	…	…	…	…

Community	3	7.7 (4.2 to 14.2)	6.4 (-2.0 to 15.6)	2.0 (0.8 to 4.5)	1.8 (0.8 to 3.8)	6	3.8 (1.9 to 7.8)	2.8 (-2.3 to 10.3)	1.8 (0.6 to 5.1)	1.7 (0.6 to 4.6)
Inpatient and Outpatient and Community	3	26.5 (13.7 to 43.5)	12.0 (-3.3 to 30.2)	0.5 (0.2 to 1.3)	1.9 (0.8 to 3.9)	…	…	…	…	…
Sample Type (Linear)	32	…	…	1.8 (1.1 to 3.0)	…	41	…	…	1.9 (1.0 to 3.3)	…
Study Quality	34	…	…	0.8 (0.6 to 1.1)	…	41	…	…	0.6 (0.4 to 0.8)	…

Note. Risk ratios have been inverted to reflect increased risk for the higher prevalence category. All continuous moderator estimates were centred and scaled prior to fitting the models, with ORs only reported for the slope.
Abbreviations: ADIS = Anxiety Disorder Interview Schedule; CIDI = Composite International Diagnostic Interview; DIS = Diagnostic Interview Schedule; *DSM-III-R* = Diagnostic and Statistical Manual of Mental Disorders 3rd Edition-Revised; *DSM-IV* = Diagnostic and Statistical Manual of Mental Disorders 4th Edition, EDE = Eating Disorder Examination; ICD-10 = International Classification of Diseases, Tenth Revision; K-SADS = Kiddie Schedule for Affective Disorders and Schizophrenia, MINI = Mini-International Neuropsychiatric Interview, SCID = Structured Clinical Interview for *DSM*, SADS = Schedule for Affective Disorders and Schizophrenia.

ED Subtype. First, we modelled prevalence separately for each ED subtype and the control samples to provide a baseline estimate. This was accomplished using three models either (a) collapsing all EDs into a single category (Any ED, Control); (b) collapsing EDs into primary diagnoses (AN, ANBN for *DSM-III-R* studies only, BN, BED, EDNOS, Any ED, Control); or (c) including all ED subtypes (AN, ANBP, ANR, ANBN for *DSM-III-R* studies only, BN, BNP, BNNP, BED, EDNOS, Any ED, Control). An overview of acronym definitions and differentiation between ED subtypes is outlined in Table 1. Estimates from individuals with combined anorexia and bulimia (ANBN) were included in the initial model but were excluded from the current ED diagnosis and subtype models as there were only two publications for the current prevalence window (i.e., Halmi et al., 1991; Herzog et al., 1992). Publications reporting OCD prevalence in a sample comprised of those with various primary EDs (denoted by any ED, ANBN, or EDNOS samples) were included in the models, however, the articles reporting these mixed categorizations led to estimates derived from a variety of sample compositions (i.e., Hsu et al., 1992 reported a prevalence estimate for a sample comprised of 2 ANR, 2 ANBP, 1 BN, and 2 EDNOS participants). These models are summarized in Table 4 as (a) population, (b) primary diagnosis, and (c) ED subtype, respectively and depicted in Figure 5.

Overall, our initial model revealed those diagnosed with an ED to be 8.9 times, $CI_{95\%}$ [3.8 to 30.1], more likely to meet OCD criteria in their lifetime and 8.0 times, $CI_{95\%}$ [4.0 to 23.7], more likely to meet current OCD criteria at the time of measurement, relative to those without an ED (see Figure 5). Furthermore, the model estimated lifetime prevalence for controls at 1.6% $CI_{95\%}$ [0.5 to 3.4], and the current estimate at 1.1% $CI_{95\%}$ [0.4 to 2.0]. This finding aligns with Fawcett et al.'s (2020) meta-analytic estimate of 1.3% for lifetime OCD prevalence for the general

population, suggesting that the controls included for comparison were representative of the general populace.

Next, a model was re-fit to examine whether OCD prevalence was moderated by ED diagnosis revealing that risk was particularly heightened for those diagnosed with AN, *RR* = 12.9 $CI_{95\%}$ [5.7 to 41.9] (Lifetime OCD) and *RR* = 11.9 $CI_{95\%}$ [5.8 to 37.2] (Current OCD), or BN, *RR* = 7.7 $CI_{95\%}$ [3.2 to 25.8] (Lifetime OCD) and *RR* = 6.4 $CI_{95\%}$ [2.8 to 21.0] (Current OCD), whereas those with BED demonstrated a non-credible trend favouring greater risk than controls, *RR* = 3.8 $CI_{95\%}$ [0.9 to 18.7] (Lifetime OCD) and *RR* = 1.9 $CI_{95\%}$ [0.7 to 6.7] (Current OCD) but lesser risk than all other EDs. Although BED was the ED at the least risk, this latter finding should be interpreted with caution due to the scarcity of BED estimates and the uncertainty inherent in the resulting comparisons. Further, those with AN were at particular risk, and were in fact 1.7 times, $CI_{95\%}$ [1.1 to 2.6], and 1.8 times, $CI_{95\%}$ [1.1 to 3.4], more likely to meet OCD criteria in their lifetime and at the time of measurement than those with BN, respectively.

Next, I analyzed OCD prevalence broken down by the specific ED subtypes which supported similar conclusions as illustrated in Figure 5. Most notably, ANBP was identified as the subtype exhibiting the numerically highest lifetime (22.2%) and current (19.2%) OCD comorbidity.[1] Despite shared purging symptom expression, individuals with ANBP were at 1.8 times $CI_{95\%}$ [0.9 to 3.6] greater risk for OCD in their lifetime and 2.8 times $CI_{95\%}$ [1.4 to 5.4] more likely to be diagnosed with current OCD than those with BNP. Given that BN also has two subtypes, we examined whether BNP was at greater risk for comorbid OCD than BNNP. Although

[1] It is worth recognizing that both ANBN and Any ED exhibit higher prevalence estimates than ANBP for the current model. However, both include few samples for the lifetime (3 estimates each) and current measurement windows (2 and 3 estimates, respectively), resulting in broad confidence intervals. Further, ANBN is no longer considered a valid diagnosis and Any ED is a mixture of ED diagnoses.

there was a slight trend favouring greater OCD diagnosis in BNNP than BNP, *RR* = 1.3 $CI_{95\%}$ [0.5 to 2.7] (Lifetime) and *RR* = 1.4 $CI_{95\%}$ [0.4 to 3.5] (Current), this difference was not credible.

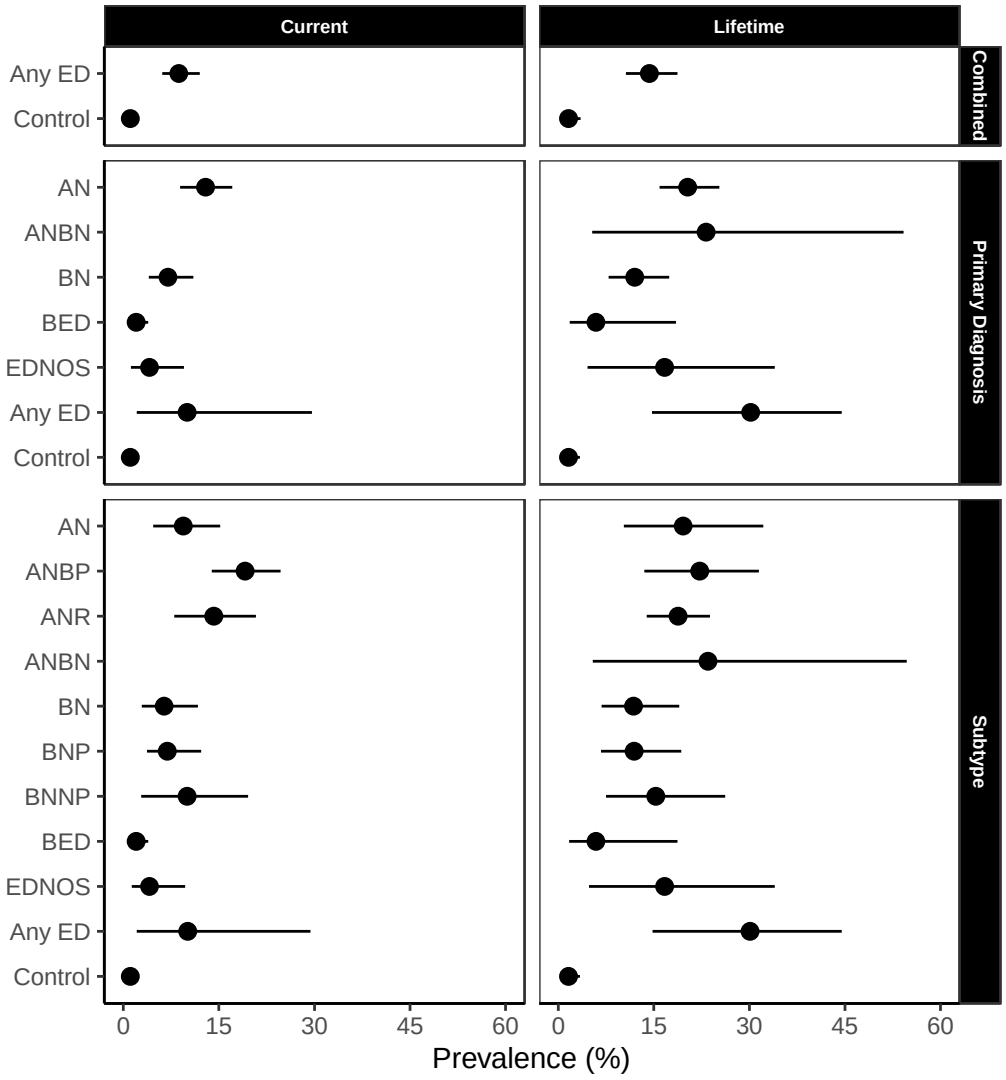

Figure 5. Forest plots illustrating OCD prevalence (%) by EDs for current and lifetime prevalence windows presented separately for the (a) population, (b) primary diagnosis, and (c) ED subtype models as described in-text. Points and errors bars reflect model estimates with corresponding 95% confidence intervals. The classification "ANBN" was excluded from the current models as there were too few estimates. Refer to Table 1 for the abbreviations with corresponding definitions.

Sample Type. The setting from which ED participants were recruited was analyzed next, by coding samples as either a community (recruited using advertisements shared in the community; no direct referrals), outpatient (receiving outpatient treatment with no requirement to remain on site for the duration of care), inpatient (residential care), or mixed sample (e.g., inpatient and outpatient, outpatient and community, or inpatient/outpatient/community). Outpatient/community and mixed inpatient/outpatient/community samples were excluded from all of the sample type models for the current measurement window (e.g., Boujut et al., 2012; Milos et al., 2001; Wilfley et al., 2000) as there were too few estimates per sample type (< 3).

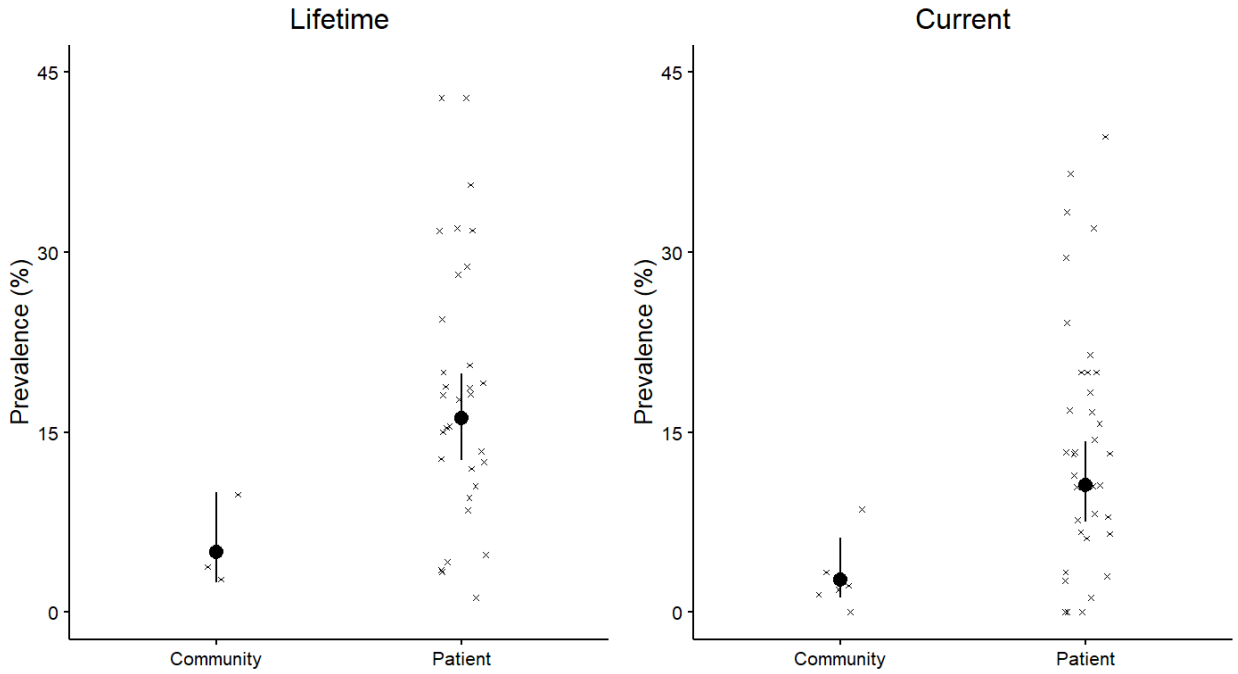

Figure 6. Plot illustrating OCD prevalence (%) by sample type (collapsed as community or patients) for lifetime and current prevalence. Points and error bars reflect the estimates from the Bayesian model with the corresponding 95% confidence intervals. The prevalence reported in each publication is represented by an "x" and were jittered on the x-axis for visibility.

The initial model collapsed all patient categories into a single group for comparison against the community samples. Report of combined outpatient/community samples in either the lifetime (e.g., Bulik et al., 1996; Lavender et al., 2013; Wilfley et al., 2000) or current models (Boujut et al., 2012; Wilfley et al., 2000) were excluded from this analysis, because they could not be dichotomously categorized as either a patient or community sample. For the same reason, mixed inpatient/outpatient/community samples were excluded for the lifetime model (e.g., Lilenfeld et al., 1998; Mangweth-Matzek et al., 2010; Milos et al., 2013) and current model (e.g., Milos et al., 2001). As summarized in Table 4 and depicted in Figure 6, this analysis found that those receiving treatment were 3.5 times, $CI_{95\%}$ [1.5 to 6.7], and 3.9 times, [1.6 to 9.2], more likely to experience OCD in the lifetime and current measurement windows, respectively. This suggests that individuals seeking outpatient or inpatient treatment may exhibit greater psychopathology than those with EDs in the community. Next, the model was re-fit to examine the prevalence of OCD for each population. As exemplified in Figure 7 this provided further support for the earlier analysis: OCD was least prevalent in community samples, with prevalence increasing amongst outpatient and inpatient samples.

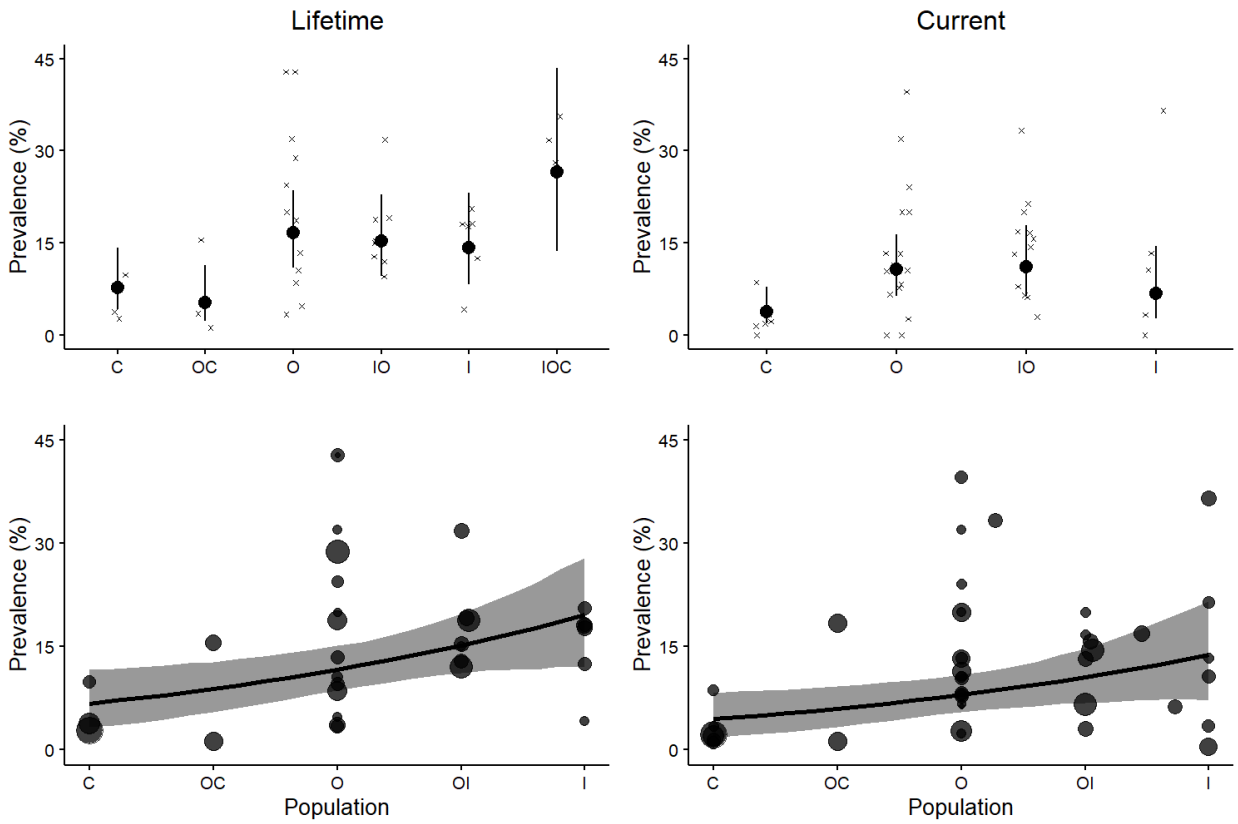

Figure 7. Plot illustrating OCD prevalence (%) by sample type for lifetime and current prevalence. Points and error bars reflect the estimates from the Bayesian model with the corresponding 95% confidence intervals. The prevalence reported in each publication is represented by an "x" and were jittered on the x-axis for visibility. The regression line reflects the prevalence predicted by the corresponding moderator model and the 95% confidence intervals are illustrated by the grey overlay. I = inpatient, IO = inpatient and outpatient, O = outpatient, OC = outpatient and community, C = community, IOC = inpatient/outpatient/community.

To better evaluate this potential linear trend, sample type was re-coded into an ordinal variable thought to reflect increasing intensity of psychiatric care. Specifically, community samples were recoded as -1, outpatients as 0, and inpatients as 1, with samples resting between two categories coded proportionate to the relative mixture of those categories (e.g., Salbach-

Andrae et al., 2008 reported 73% inpatients and 27% outpatients, therefore, would be coded as 0.73). Studies where the composition of the combined sample was unknown were coded as +/- 0.50, placing the sample equally between the two categories. This coding scheme no longer required any particular number of estimates for any given category, permitting inclusion of the combined outpatient-community samples in both models and inpatient samples in the current model. As a result, only one study remained excluded, because it combined inpatients, outpatients, and community samples (Milos et al., 2001).

Lifetime and current OCD prevalence increased linearly as a function of the increasing intensity of psychiatric care: Lowest prevalence was observed in community samples with a gradual increase extending to inpatients whilst variability was broadest among outpatients. As a result, the linear trend was prominent for lifetime and current OCD, providing support for the plausibility that comorbid OCD prevalence increases as ED psychopathology becomes more severe (see Figure 7).

ED Age of Onset. Findings are mixed with respect to the order of ED and OCD occurrence, with some samples showing nearly all cases (~94%) experienced anxiety disorder onset (inclusive of OCD at time of study) preceding their ED (Bulik et al., 1997) and other samples showing nearly equal representation of anxiety disorder onset before versus in the same-year or after ED onset (Carrot et al., 2017; Godart et al., 2003). Due to insufficient reporting of the age of onset for OCD we were unable to determine sequential order of disorder onset, however, we were able to assess whether mean ED age of onset was related to OCD prevalence.

Analysis of the lifetime estimates revealed higher comorbidity levels in samples with earlier mean age of ED onset. However, inspection of the data revealed this effect to be driven largely by a univariate outlier characterized by a particularly late mean age of onset (see Figure 8;

Grilo et al., 2009), without which the relation trended in the same direction but was no longer credible, $OR_{excluded}$ = 0.8, $CI_{95\%}$, [0.5 to 1.3]. Analysis of current estimates revealed a non-credible trend in the opposite direction as shown in Figure 8 regardless of whether the outlier was included or excluded, $OR_{excluded}$ = 1.9 $CI_{95\%}$, [0.9 to 4.2]. In either case, this variable appears to suffer from a notable range restriction, with most samples having an average onset of EDs between the ages of 16 and 20. Thus, strong conclusions cannot be drawn and rather it highlights the need for further research in populations with late ED onset.

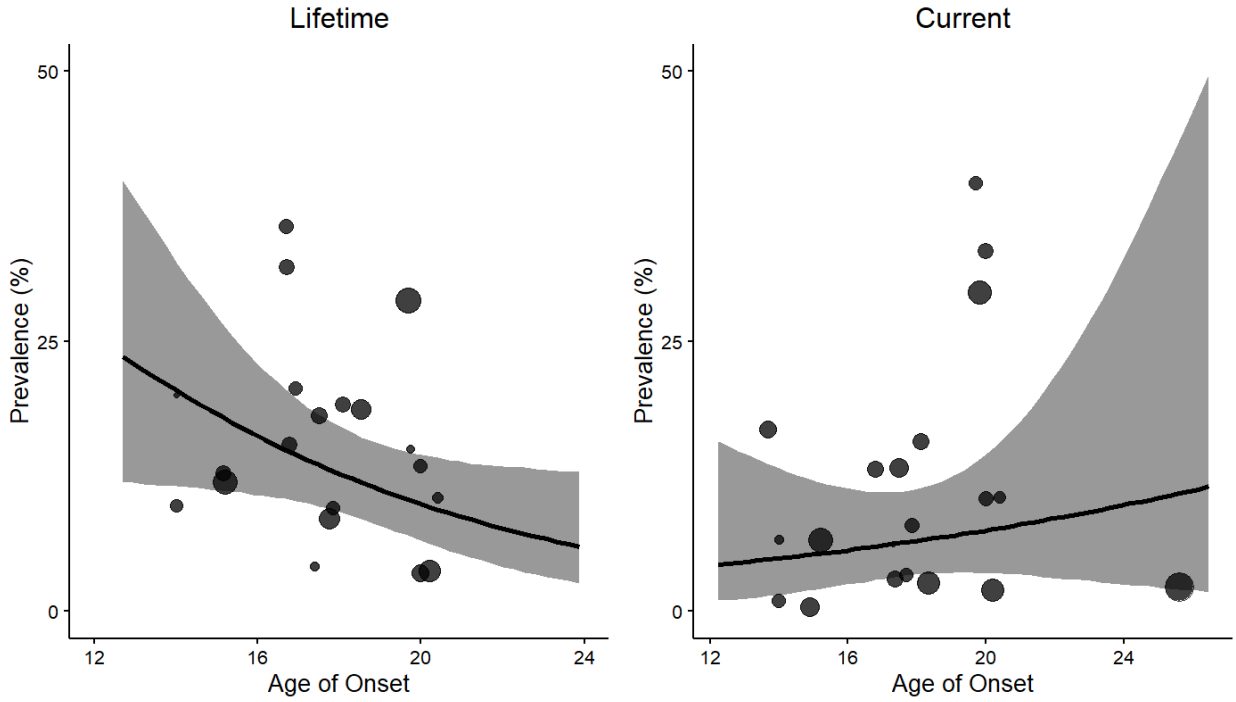

Figure 8. Plot illustrating OCD prevalence (%) by mean age of ED onset for lifetime and current prevalence. Points represent the empirical estimates reported in the publications with the point size reflecting the sample size. The regression line reflects the prevalence predicted by the corresponding moderator model and the 95% confidence intervals are illustrated by the grey overlay.

BMI. As past research has found low body weight to increase symptom severity and comorbidity (with the prognosis improving as body weight returns to a healthy range; Calugi et al., 2018), BMI was explored as a moderator. As depicted in Table 4 and Figure 9, samples with lower mean BMIs predicted credibly higher lifetime and current OCD comorbidity. However, mean BMI also varied systematically as a function of ED diagnosis, making this association difficult to interpret in the present data. For example, the studies with the highest BMI were composed largely of BN and/or BED patients (i.e., Grilo et al., 2009; Striegel-Moore et al., 2001; Wilfley et al., 2000), which tended to be at lower risk of OCD than AN patients (who, on average, would have lower BMIs).

To evaluate whether the effect of BMI was driven by sample composition, we fit separate moderator models for only those with AN and BN diagnoses as we had a sufficient number of samples. A non-credible trend demonstrating higher comorbidity with lower BMI was found for BN samples only for the current measurement window (for which we have 96% confidence in the direction of the relation). None of the remaining AN or BN models produced even a tendency in either direction. We urge caution in interpreting this effect due to it being confounded with ED diagnosis and being driven by two leverage points with particularly high BMIs relative to the other included samples.

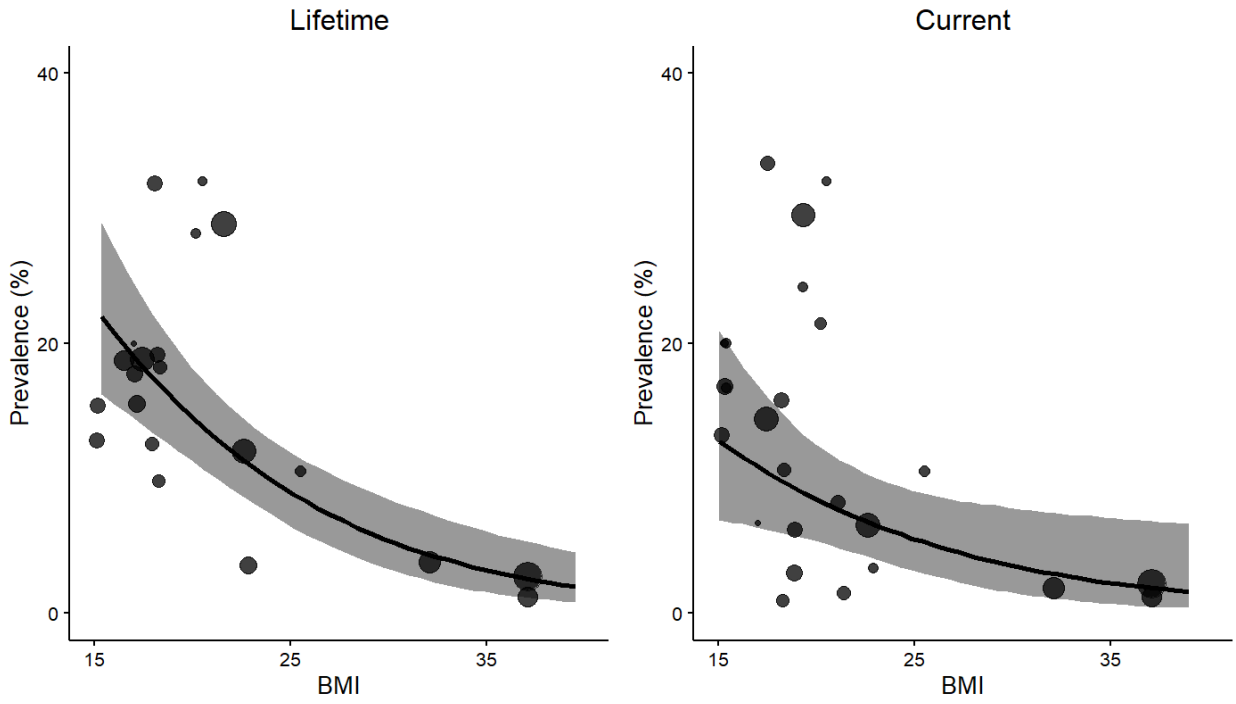

Figure 9. Plot illustrating OCD prevalence (%) by mean BMI for lifetime and current prevalence. Points represent the empirical estimates reported in the publications with the point size reflecting the sample size. The regression line reflects the prevalence predicted by the corresponding moderator model and the 95% confidence intervals are illustrated by the grey overlay.

Quality Ratings. A trend was observed in favour of quality ratings as a predictor of lifetime OCD comorbidity and was a credible moderator in the current measurement window. A similar association was observed across prevalence windows indicating that studies with higher quality ratings tend to report lower comorbidity than studies with lower quality ratings (see Figure 10).

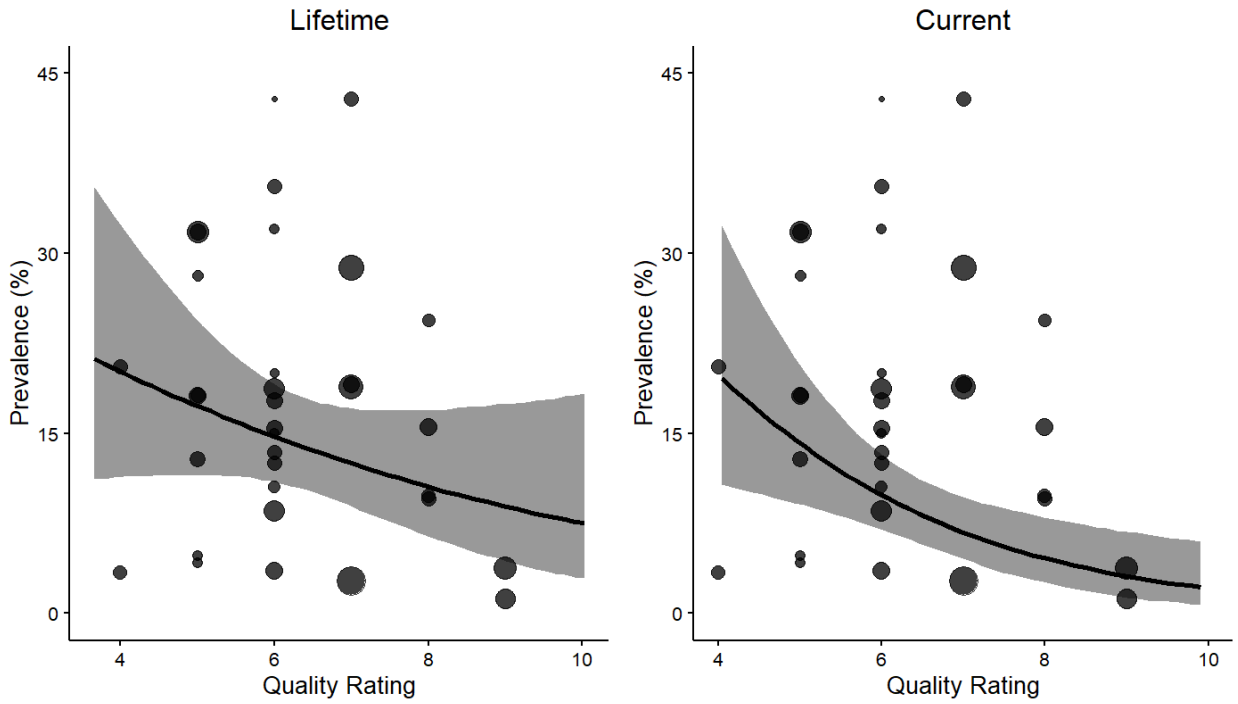

Figure 10. Plot illustrating OCD prevalence (%) by quality rating for lifetime and current prevalence. Points represent the quality ratings assigned with the point size reflecting the sample size. The regression line reflects the prevalence predicted by the corresponding moderator model and the 95% confidence intervals are illustrated by the grey overlay.

Non-Credible Moderator Analyses

Moderator analyses revealed non-credible differences or trends related to ED duration, mean age, OCD diagnostic criteria, OCD diagnostic measure, geographic region, and year. Results for *all* non-credible moderators are summarized below and outlined in Table 4.

ED Duration. ED duration was examined as an exploratory predictor of lifetime and current OCD comorbidity as literature to date suggests the length of living with any psychiatric condition increases the risk for experiencing additional psychiatric conditions (see extensive reviews O'Brien & Vincent, 2003; Swinbourne & Touyz 2007). Previous research has suggested

a significant correlation may exist between overall length of living with an ED and experience of comorbid OCD (Godart et al., 2003). As shown in Figure 11, average duration of EDs across samples trended in a positive direction for lifetime OCD with 94% certainty that as duration increases so does the prevalence. The former is a promising trend but further data are required before strong conclusions may be drawn. The lack of a similar relation for current might be cause for greater caution, however, there are fewer lifetime estimates for ED duration than current estimates.

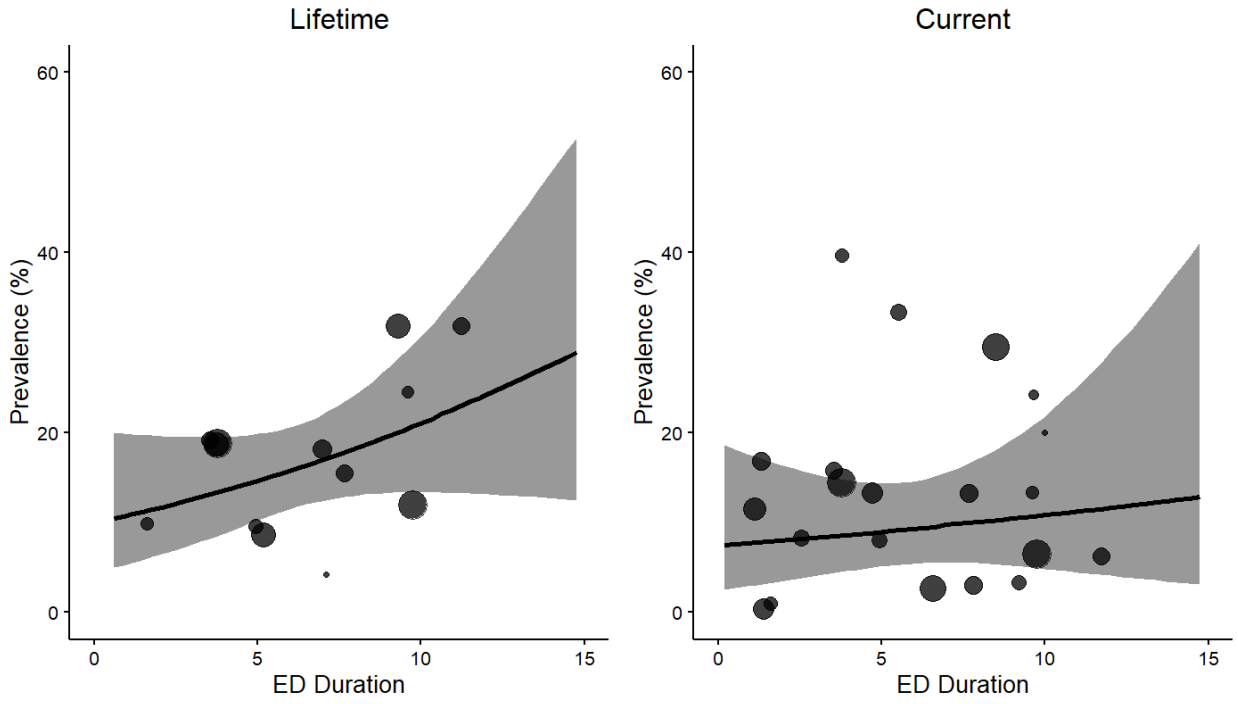

Figure 11. Plot illustrating OCD prevalence (%) by mean ED duration for lifetime and current prevalence. Points represent the empirical estimates reported in the publications with the point size reflecting the sample size. The regression line reflects the prevalence predicted by the corresponding moderator model and the 95% confidence intervals are illustrated by the grey overlay.

Age. The mean age of the sample was probed as a potential continuous moderator for lifetime and current OCD prevalence in EDs but presented no compelling support of an effect as depicted in Figure 12. Furthermore, age of the sample was confounded with age of ED onset and ED duration but did not suffer from the same level of range restriction as observed in age of ED onset.

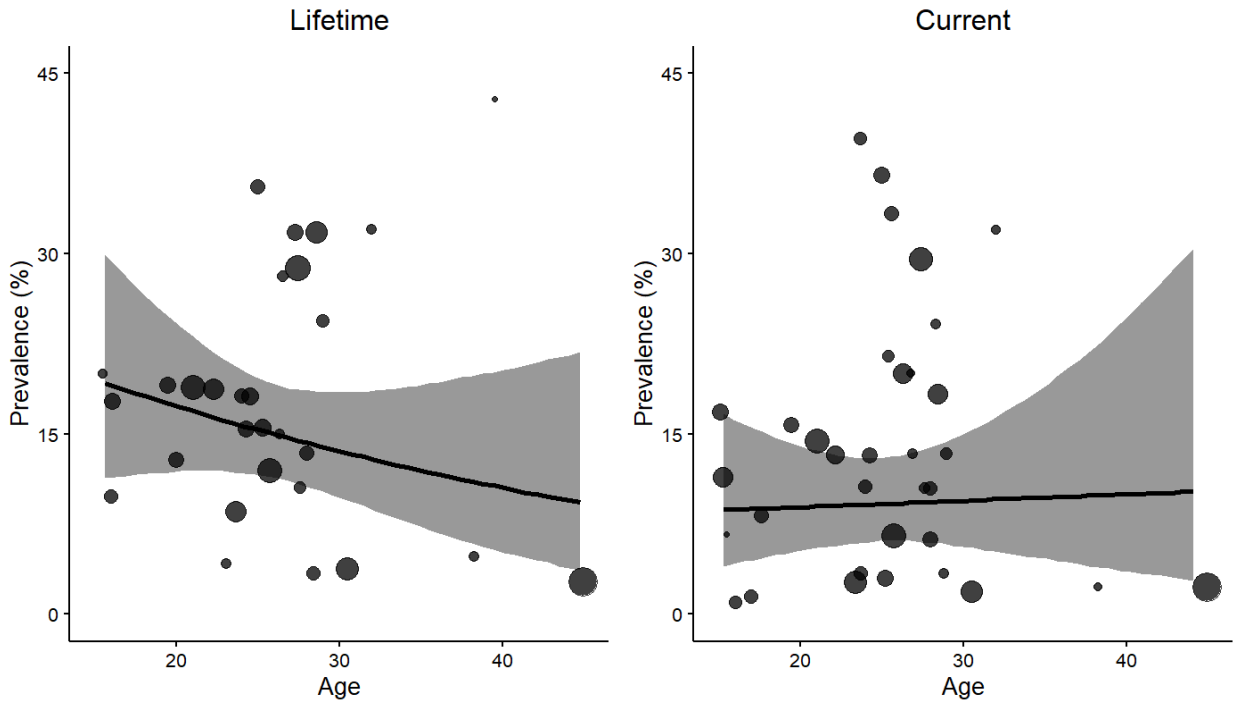

Figure 12. Plot illustrating OCD prevalence (%) by mean age for lifetime and current prevalence. Points represent the empirical estimates reported in the publications with the point size reflecting the sample size. The regression line reflects the prevalence predicted by the corresponding moderator model and the 95% confidence intervals are illustrated by the grey overlay.

OCD Diagnostic Criteria. As each new edition of the *DSM* brought forth new refined subclassification of both OCD and EDs, it was expected that the change in approach may also impact the identification of their co-occurrence (APA, 2013). The diagnostic criteria employed to

assess for the presence and diagnosis of comorbid OCD was coded to determine whether prevalence increased as a function of the criteria used to guide clinician diagnosis. Only one study utilized *ICD-10* criteria and was thus excluded from the lifetime model due to inadequate number of informative estimates (e.g., Anderluh et al., 2009). Diagnostic criteria utilized for OCD diagnosis was not supported as a credible predictor of either lifetime or current comorbidity in EDs. The *DSM-IV* yielded higher prevalence rates than *DSM-III-R* for lifetime OCD and current OCD (Figure 13), however, it is important note that despite the observed differences, the difference was not credible thus not indicative of having a significant impact on comorbid OCD prevalence in EDs.

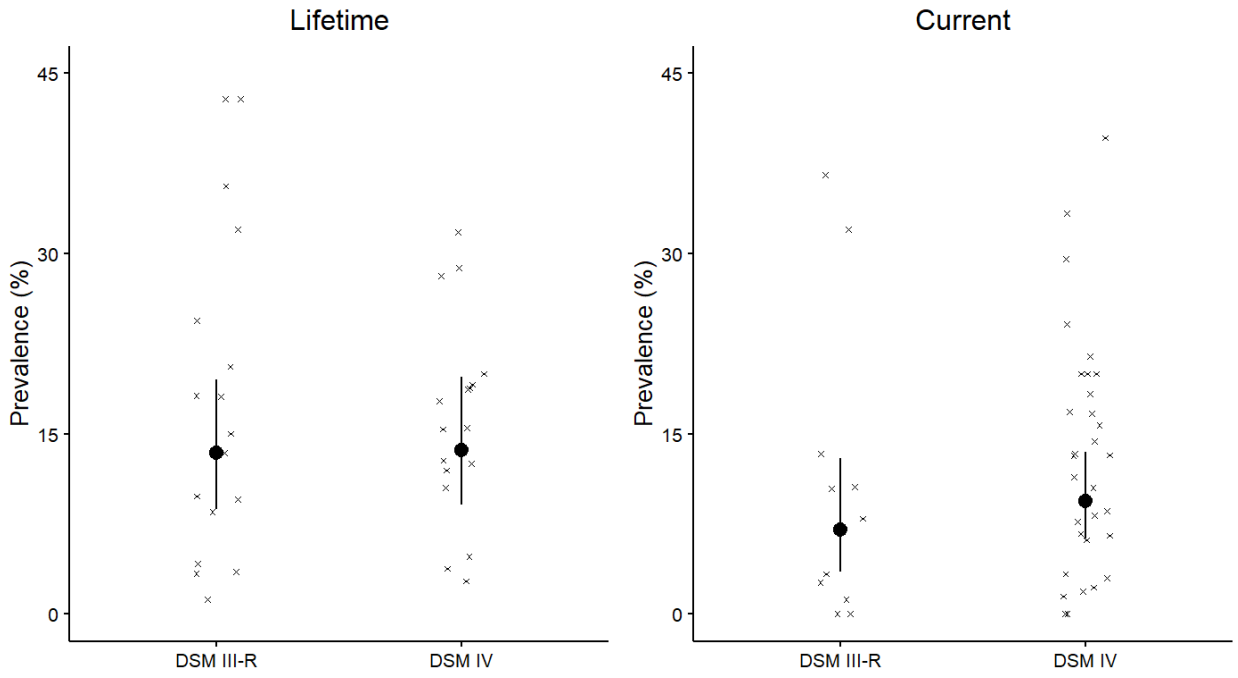

Figure 13. Plot illustrating OCD prevalence (%) by OCD diagnostic criteria employed *(DSM-III-R, DSM-IV)* for lifetime and current prevalence. Points and error bars reflect the estimates from

the Bayesian model with the corresponding 95% confidence intervals. The prevalence reported in each publication is represented by an "x" and were jittered on the x-axis for visibility.

OCD Diagnostic Measure. The type of semi-structured or structured interview clinicians applied to support OCD diagnoses was examined as a potential predictor. A variety of semi-structured and structured interviews were used in the literature to guide the assessment of OCD and EDs alongside the clinicians' expertise. Studies that used the ADIS (e.g., Schwalberg et al., 1992), CIDI (e.g., Godart et al., 2000; Thornton & Russell, 1997), unidentified clinician interview (e.g., Rabe-Jablonska, 2003; Rastam et al., 1992), DIS (Halmi et al., 1991), or EATATE (Anderluh et al., 2009) in the lifetime model and the ADIS (Swinbourne et al., 2012), CIDI (Godart et al., 2000; Salbach-Andrae et al., 2008), unidentified clinician interview (Rastam et al., 1992; Thiel et al., 1995), DIS (Bellodi et al., 2001; Halmi et al., 1991), or EATATE (Schmidt et al., 2008) in the current model were excluded as there was an insufficient number of estimates per measure to reliably include them in the model.

Higher lifetime OCD prevalence was observed in samples assessed using the SADS interview and were 2.1 times $CI_{95\%}$, [0.9 to 4.1] more likely to be diagnosed with lifetime OCD than those assessed with the SCID. Current OCD prevalence was markedly higher when the MINI was employed as opposed to the SADS. Current OCD prevalence with higher when using the MINI than the SADS (see Figure 14); however, the large difference and substantially greater risk must be interpreted with caution as the number of estimates available to make such a comparison are quite low. No convincing support for OCD diagnostic measure as a moderator of lifetime or current OCD prevalence was obtained.

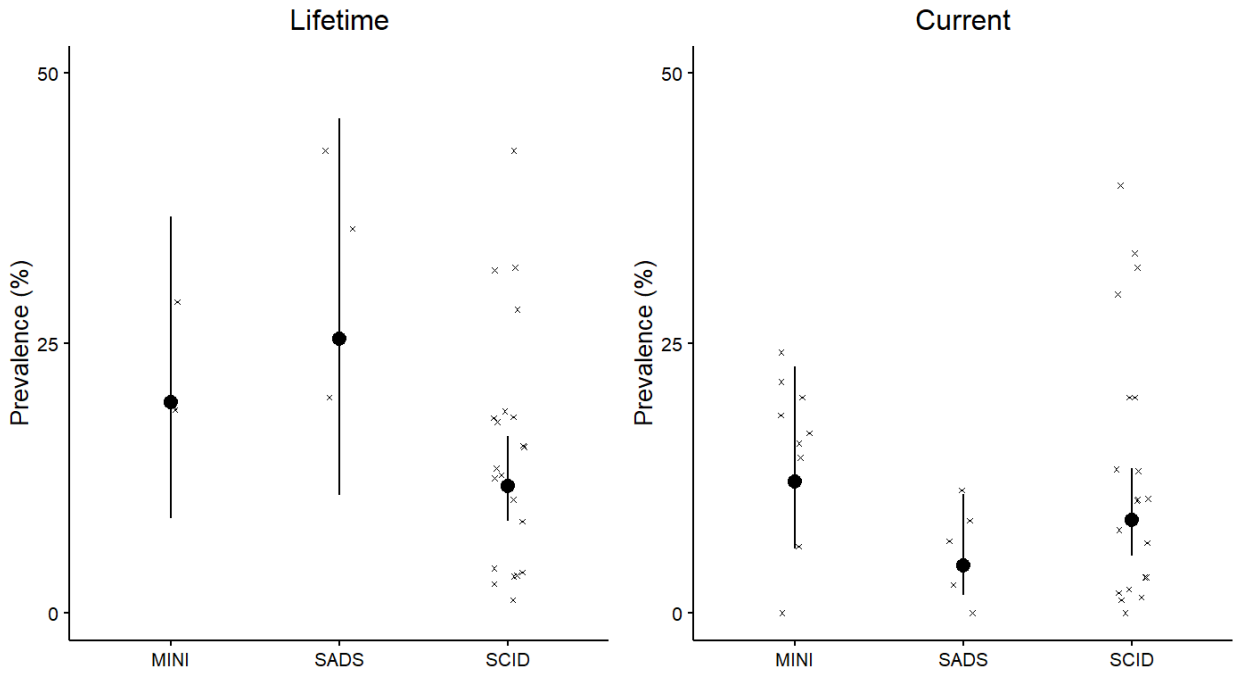

Figure 14. Plot illustrating OCD prevalence (%) by OCD diagnostic measure (MINI, SADS, SCID) used to assess for lifetime and current prevalence. Points and error bars reflect the estimates from the Bayesian model with the corresponding 95% confidence intervals. The prevalence reported in each publication is represented by an "x" and were jittered on the x-axis for visibility. MINI = Mini International Neuropsychiatric Interview; SADS = Schedule for Affective Disorders and Schizophrenia; SCID = Structured Clinical Interview for DSM Disorders.

Geographic Region. Cultural differences and diagnostic practices vary according to geographic region which may also have an impact on the prevalence of comorbid OCD, thus region was examined as a moderator on an exploratory basis. Due to a low number of estimates per country, countries were combined into four geographic regions: North America, Europe, Oceania, and Asia. One study from Oceania (Swinbourne et al., 2012) and two studies from Asia (e.g., Matsunaga et al., 1999a; 1999b) were excluded from the current model due to an

inadequate number of estimates to make reliable comparisons. The analysis did reveal that lifetime OCD prevalence in EDs was higher among Asian samples than other geographic regions. As illustrated in Figure 15, however, there was only a slight increase in current OCD prevalence in EDs amongst European samples than North American samples. Overall, no credible differences were observed and geographic region was not supported as a predictor for either lifetime or current OCD comorbidity in EDs.

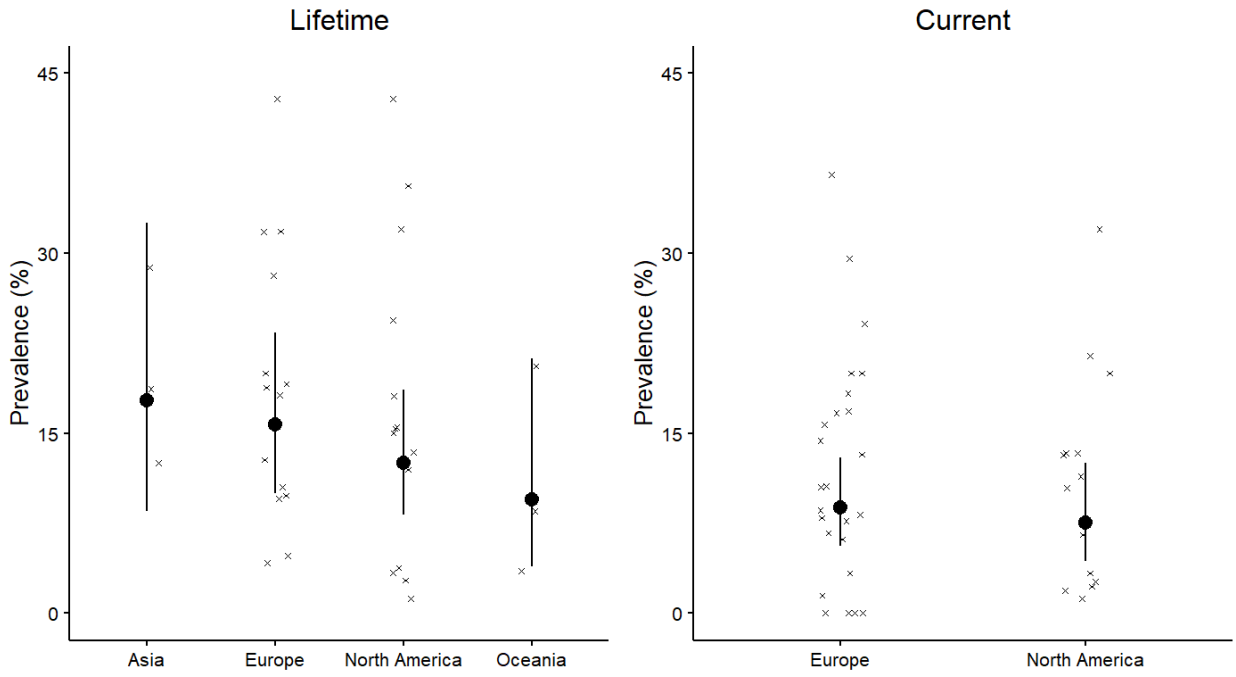

Figure 15. Plot illustrating OCD prevalence (%) by geographic region (Asia, Europe, North America, Oceania) for lifetime and current prevalence. Points and error bars reflect the estimates from the Bayesian model with the corresponding 95% confidence intervals. The prevalence reported in each publication is represented by an "x" and were jittered on the x-axis for visibility.

Year. Publication year was evaluated as a continuous moderator to determine whether the prevalence of comorbid OCD in EDs has changed over time; however, no significant changes in

lifetime or current OCD prevalence in EDs has occurred between 1988 and 2019. Following visual inspection of lifetime OCD prevalence and current OCD prevalence (see Figure 16), it is increasingly evident there was no support for even a trend in either direction for year as a potential moderator of lifetime or current comorbid OCD in EDs as the slopes are straight lines suggesting no change in the prevalence across time.

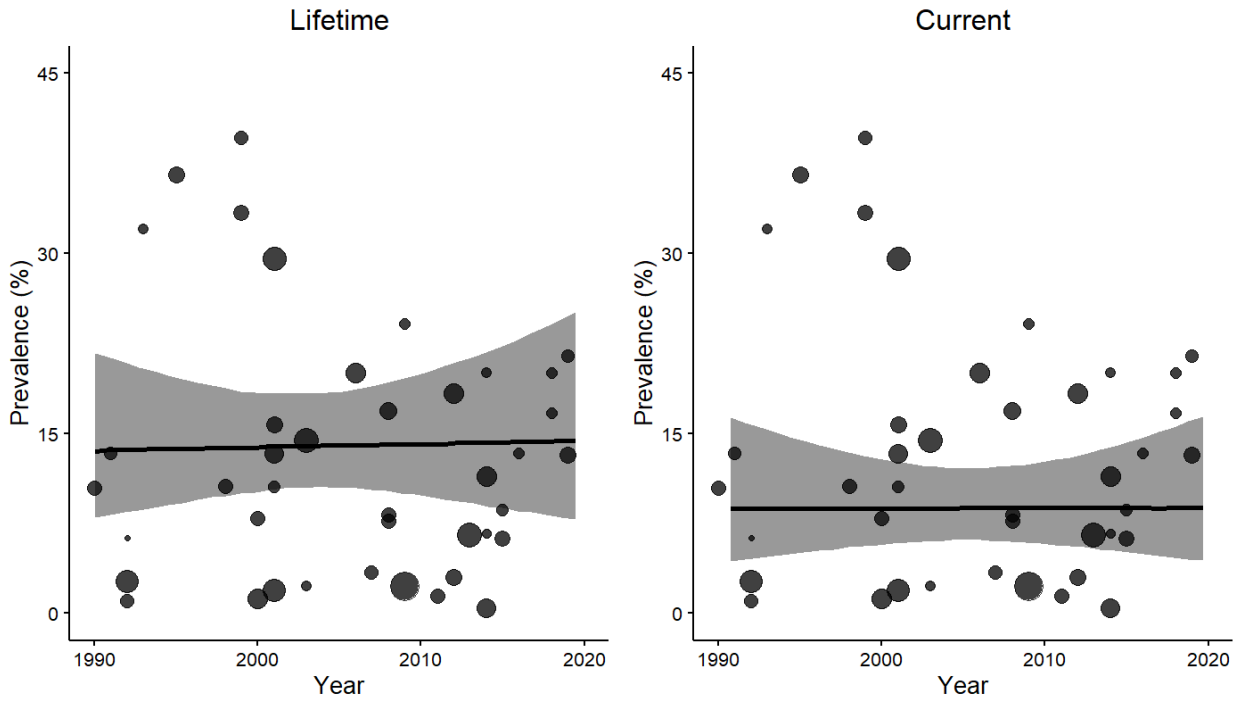

Figure 16. Plot illustrating OCD prevalence (%) by year of publication for lifetime and current prevalence. Points represent the empirical estimates reported in the publications with the point size reflecting the sample size. The regression line reflects the prevalence predicted by the corresponding moderator model and the 95% confidence intervals are illustrated by the grey overlay.

Discussion

The primary goal of the present meta-analysis was to provide a robust estimate of the lifetime and current prevalence of OCD amongst individuals with EDs. A secondary aim was to explore sources of heterogeneity in the presently reported estimates and isolate predictors of OCD and ED comorbidity to inform diagnostic practices and symptom management of both disorders with shared characteristics. The current models synthesized estimates from 59 studies and found the prevalence of OCD among people with EDs to be 13.9% and 8.7% within the lifetime and current measurement windows, respectively. These estimates were characterized by prediction intervals ranging as low as ~1% to as high as ~40% suggesting a great degree of heterogeneity across ED samples. Compared to healthy controls, those with EDs were at 8.9 times greater risk for lifetime OCD and 8 times greater risk for current OCD, relative to healthy controls.

Of the coded moderators, the effects of ED subtype, sample type, ED age of onset, average BMI, and study quality were found to be credible. Among the EDs, ANBP was at greatest risk for comorbid OCD, whereas those with BED were at lowest risk. Diagnosis of any ED elevated the risk for comorbid OCD; however, OCD was most common amongst AN and BN. The pattern observed for age of ED onset was inconsistent across measurement windows and driven largely by a univariate outlier. With respect to sample type, OCD prevalence was found to be lowest in community samples and notably higher among inpatient or outpatient samples. Lower average BMI was associated with higher lifetime and current OCD comorbidity but was confounded with the primary ED diagnosis (e.g., lower BMIs are anticipated to be observed in AN whereas, higher BMIs are associated with BED due to the nature of the conditions). Lastly, studies scoring higher in quality tended to have lower comorbidity than those scoring lower in quality. These findings are discussed in greater detail below.

Greater OCD Prevalence in EDs than Healthy Controls

Whereas the present meta-analysis estimated OCD comorbidity to be roughly 13.9% and 8.7% for the lifetime and current measurement windows, respectively, these values were slightly lower than those reported by another recent meta-analysis. Mandelli et al. (2020) estimated OCD comorbidity among people with EDs to be 18% and 15% for the lifetime and current measurement windows, respectively. The difference between the estimates from these two meta-analyses may be attributable to the present meta-analysis' inclusion of additional articles, use of more lenient search restrictions (e.g., no date or language restrictions were placed on the search conducted for the current meta-analysis), and variations in inclusion criteria (e.g., the present analysis included *DSM-III-R* or newer diagnostic criteria and only prospective investigations). Consistent with past research (e.g., Mandelli et al., 2020), the present meta-analysis found greater risk for lifetime and current OCD among those with AN compared to BN, and among ANBP in particular. The current meta-analysis illustrated that lifetime and current OCD comorbidity was not only numerically greater in ANBP than ANR, as anticipated due to ANBPs higher rate of psychiatric comorbidity generally, but ANBP was also identified as the ED subtype at greatest risk for experiencing comorbid OCD among the EDs (Altman & Shankman, 2009; Margolis et al., 1994).

Identification of ANBP as the ED subtype most vulnerable for comorbid OCD also differs from Mandelli et al. (2020), who found instead a non-significant trend favouring ANR (Mandelli et al., 2020, Figure 5).[2] This difference is likely attributable to variation in the included literature as well as the analytic and coding practices between the meta-analyses. The most notable difference for this outcome was perhaps in the coding of Speranza et al. (2001): Whereas I coded

[2] Note it is believed that the axis label for Figure 5 from Mandelli et al. (2020) has been reversed, such that values below 1 reflect greater OCD in ANBP and values above 1 reflect greater OCD in ANR; as such, their finding of an odds ratio of 1.3 in Panel C would non-significantly favour greater OCD in ANR for the lifetime measurement window.

lifetime prevalence as 16% (*n* = 44; ANR) and 43% (*n* = 14; ANBP, e.g., see Table 4 of Speranza et al., 2001), Mandelli et al. (2020, Figure 5) coded 42.8% (*n* = 14; ANR) and 12.9% (*n* = 31; ANBP) which is the opposite of what is reported in-text by the authors. As such, Speranza et al. (2001) strongly favoured ANBP > ANR in our models but strongly favoured ANR > ANBP in their models. Further, whereas our risk ratios were calculated across samples (using the posterior from our logistic regression model), Mandelli et al. (2020) instead calculated odds ratios within-samples. Nonetheless, the comparison between the AN subtypes favouring ANR for Mandelli et al. (2020) was non-significant and ANBP for the present meta-analysis was also non-credible.

Lifetime and current OCD comorbidity was second most prevalent in BN. One possible interpretation of this finding is that the purging component of ANBP contributed to increased comorbid OCD prevalence as this behaviour may be viewed as similar to compulsive behaviours in OCD. However, as BNP did not present with a similar elevated prevalence for BN subtypes, uncertainty remains as to why ANBP is the ED subtype most at risk for comorbid OCD and what specifically about ANBP contributes to such heightened risk. BED demonstrated the lowest rates of OCD, which was unexpected as BED has been found to be highly comorbid with lifetime and current anxiety related disorders including OCD (Grilo et al., 2009). Therefore, it was anticipated that those with BED would be at greater risk akin to other EDs. However, this particular disorder was also partially confounded with sample type, as the majority of BED estimates were derived from community samples. Likewise, AN and BN estimates were predominantly obtained from inpatient or outpatient samples, thus it is plausible these samples may have been diagnosed in a timelier fashion than those in the community (e.g., BED samples).

Potential Contributors Driving Comorbid OCD in ANBP

There are several possible factors that may explain the heightened risk of comorbid OCD in ANBP including impulse regulation difficulties, frequency and type of comorbid personality disorders, experience of early life stressors/trauma, treatment motivation, and treatment non-compliance. A core tenant to the expression of the AN subtypes involves differences in rigid symptomology such as impulsivity and emotion regulation. ANBP is heavily influenced by lack of impulse control and extreme difficulty regulating emotions compared to what is observed by ANR (Hoffman et al., 2012; Weinbach et al., 2018). For example, the cycle of weight obsession followed by binge eating and purging behaviours closely mirror the cycle of obsessions and compulsions observed in OCD.

Purging behaviors in outpatients with EDs have been linked to increased difficulty with impulse regulation and higher rates of personality disorders (Murakami et al., 2002). Higher rates of obsessive-compulsive personality disorder (OCPD) have been found in AN compared to BN (Martinussen et al., 2017) and may hint at perfectionism as a mutually shared vulnerability factor for OCD and EDs (Vanzhula et al., 2021). An evidence-based synthesis by Young et al. (2013) identified a significant association specifically between OCPD in AN patients who engage in excessive exercise, therefore, serving as a second illustration of the prevalence of OCPD in those with ANR than ANBP. However, another meta-analysis found that individuals with ANBP are most frequently diagnosed with borderline or paranoid personality disorder, with OCPD more common in individuals with ANR (Farstad et al., 2016). Thus, future research is needed to clarify whether impulse regulation difficulties or the frequency or type of comorbid personality disorders may help explain the heightened risk for comorbid OCD in individuals with ANBP.

Another potential explanation for the higher prevalence of comorbid OCD observed in ANBP is with respect to the experience of early life stressors. Reyes-Rodriguez and colleagues (2011) assessed a sample of 753 women with AN according to *DSM-IV* criteria and found greater odds for experiencing comorbid PTSD amongst those with ANBP than ANR. Similarly, a retrospective chart review by Blinder et al. (2006) found similar results further supporting this relation with their ANBP sample being at two times greater risk for PTSD than all other EDs (inclusive of ANR, BN, and EDNOS). The most reported adverse experiences involved instances of sexual assault in childhood and/or adulthood with the majority of traumatic events occurring before AN onset (Reyes-Rodriguez et al., 2011). When looking at the influence of type of childhood trauma on ED psychopathology and comorbid psychiatric disorders, emotional abuse was associated with greater preoccupation with eating, shape, and weight versus sexual or physical abuse, which was correlated only with concerns pertaining to eating (Guillaume et al., 2016). Furthermore, a history of childhood trauma is most frequently observed in those diagnosed with OCD prior to the onset of PTSD than those diagnosed with PTSD before the onset of OCD (Araujo et al., 2018). Individuals who had OCD pre-trauma tended to exhibit higher rates of psychiatric comorbidity with a mean number of lifetime psychiatric disorders of 4.9 (Araujo et al., 2018). As a history of childhood adverse events is significantly correlated with ANBP and OCD (such as cleaning behaviours; Lockwood et al., 2004), this may be a neglected historical factor that further explains the link between higher rates of OCD in ANBP than other EDs.

Childhood trauma has been linked to lower mental health service engagement (Dixon et al., 2016), which may explain why ED patients tend to be more ambivalent about seeking treatment and have a decreased motivation to recover, particularly in AN. Simultaneous treatment for comorbid OCD in EDs often lends to greater improvement in those with BN than AN after 57 days

seeking inpatient care with significant reductions in OCD, ED, and depression symptom expression (Simpson et al., 2013). In severe cases, delayed treatment seeking in AN can result in involuntary hospitalization to preserve life (Douzenis & Michopoulus, 2015).

Nordbø et al. (2012) systematically examined and identified 7 barriers preventing the motivation to recover from AN including but not limited to the denial of the presence of AN, feeling overwhelmed by distress, misperception of judgments about their body or weight, and valuing having AN (e.g., increased social desirability when meeting the thin ideal; Nordbø et al., 2012). For example, a thematic analysis of 20 ED outpatients revealed that patients either perceived maintenance of the ED as a burden that controls them leading to a desire to change versus others who viewed the ED as a new outlet to exert control over one's life hindering desire to change (Reid et al., 2008). When examining motivation to engage in behaviour change, Knowles et al. (2013) reviewed a series of studies specifically examining the impact of motivational therapeutic interventions but found little efficacy for reducing or even having an impact on restrictive or compensatory eating behaviours. Although there is hope for motivating change in ambivalent ED patients with the use of piloted interventions that place a large emphasis on fostering a motivational environment (Feld et al., 2001), rates of attrition remain high in AN in comparison to other EDs.

Treatment non-compliance may also be implicated in the high rates of OCD comorbidity, with ANBP associated with the highest attrition rates for treatment than those with ANR and is further complicated with notable lack of readiness for change especially amongst inpatient samples (Abdelbaky et al., 2013). Similarly, in a randomized control trial that recruited adult women experiencing AN for at least 7 years, those with ANBP were significantly more likely to not complete the intervention (Abdelbaky et al., 2012). They found that early termination of treatment

was strongly predicted by diagnosis with ANBP and extremely low quality of life. Interestingly, the frequency of attrition was not associated with the type of intervention utilized, BMI, or the ED duration. The high rate of attrition in AN outpatient samples is believed in part to be influenced by the lack of incorporation of individual differences into treatment planning (e.g., failure to account for personality or cognitive factors such as perfectionism, rigidity, or obsessionality; Martinez & Craighead, 2015). As those with AN often deny the presence of an ED or the need for additional support, it is plausible that as ED psychopathology becomes more severe so does the risk for and experience of comorbid OCD as observed in the linear model of sample type.

In summary, impulse regulation difficulties, type of comorbid personality disorders, experience of early life stressors/trauma, treatment motivation, and intervention compliance may be contributing factors to heighted risk of comorbid OCD in ANBP. Impulsivity and its associated behaviours are often used as a coping mechanism for trauma to mitigate against intrusive negative cognitions (Lockwood et al., 2004), therefore it is also important to be cognizant of the possibility that those with ANBP who are at greater odds for experiencing comorbid PTSD may be using impulsive and compulsive behaviours to reduce emotional arousal or avoid negative emotions (Lockwood et al., 2004). With respect to mental health treatment engagement and compliance, if individuals with ANBP are less engaged or compliant with treatment, it is plausible that greater symptom severity or greater resistance towards changing shared etiological factors (e.g., perfectionism, rigidity, obsessionality) contribute to greater OCD comorbidity in ANBP compared to AN-R.

Increased OCD Comorbidity in Patient Samples

With respect to sample type, OCD prevalence was found to increase linearly as the level of psychiatric care intensified. Sallet and colleagues (2010) found, in their examination of 92

patients with AN, BN, or BED, those with comorbid OCD were more likely to seek psychiatric treatment in comparison to patients with OCD but no ED. Our findings in combination with the observations made by Sallet and colleagues (2010) suggest that it is plausible that those with comorbid OCD and EDs present a need for greater care, and therefore may be more likely to seek psychiatric support. Individuals with EDs and secondary OCD require more intensive interventions and have poorer treatment outcomes so it is more likely they will also require referral to higher levels of care. Lifetime and current comorbid OCD in the present meta-analysis was lowest amongst community populations and highest amongst patients seeking inpatient or outpatient psychiatric support. Further examination of which populations or subgroups of individuals with EDs are at greatest risk for experiencing comorbid OCD will help inform approaches to clinical assessment and more targeted interventions.

Impact of ED Age of Onset on OCD Comorbidity

Although an initial trend had been observed showing heightened risk of lifetime OCD in populations with earlier mean ED onset, this trend was inconsistent between the measurement windows and driven largely by an outlier with a late age of ED onset and low prevalence (Grilo et al., 2009). The trend was no longer credible once the outlier was removed; therefore, this trend should interpreted with caution. This analysis and the leverage point reveals range restriction within this variable as almost all samples report a mean age of ED onset between 16 and 20 years old. Past research by Patriciello and colleagues (2017) has noted a bimodal distribution for age of ED onset amongst both their AN and BN samples between early versus late onset. The majority of participants (upwards of 83%) experienced early onset AN or BN around the age of 18, which parallels the mean age of ED onset of the present meta-analysis. Although a bi-modal distribution for age of ED onset was not observed in the present meta-analysis, the high rate of early ED onset

does correspond with our data demonstrating a greater number of estimates within the restricted range of 16 to 20 years. Interestingly, they observed no difference between the early and late age of onset groups, which calls into question if there were more data on late age of ED onset would the trends become credible in the present meta-analysis or would they diminish to align with Patriciello et al. (2017).

The scarcity of late age of onset estimates is of great concern. One possible explanation for the lack of late age of onset data in ED samples may be relative to past observations that have found those with late onset tend to exhibit less severe ED psychopathology (e.g., fewer vomiting episodes or less evident personality traits; Bueno et al., 2014). As a result, individuals with late ED onset may not be readily diagnosed or may not be aware that they are coping with an ED due to reduced severity of symptoms and the tendency to fall within a higher body weight range. Therefore, future examination of age of onset as a moderator will require greater exploration of OCD comorbidity with broader age ranges inclusive of later ED onset for stronger conclusions to be able to be drawn.

Lower BMI Predicts Greater OCD Comorbidity

Mean BMI was a credible moderator for lifetime and current comorbid OCD with lower BMIs being associated with higher prevalence rates. Our findings align with past discoveries that found those with comorbid OCD in EDs tend to exhibit significantly lower BMIs than those with an ED in the absence of OCD (Lennkh et al., 1998; Matsunaga et al., 1999b; Speranza et al., 2001) and with similar findings for comorbid AN and GAD (Thornton et al., 2011). In contrast to past research, BMI was not isolated as a predictor of OCD comorbidity for AN despite the findings from Calugi et al.'s (2018) longitudinal treatment study, which observed a significant association between starvation symptoms among AN patients at baseline and increased eating disorder and

general psychopathology. Across the 20 weeks of treatment, marked improvement of general psychopathology occurred in relation to increases in BMI and decreases in starvation symptoms, with greater starvation symptoms resulting in slower improvement of general psychopathology and other relevant variables across treatment (Calugi et al., 2018).

Interestingly, Abramovitch et al. (2019) identified that even OCD without comorbid AN or depression is associated with significantly lower BMI and greater risk for meeting the underweight threshold. This finding is noteworthy as those with AN and OCD both have been found to be associated with lower BMI's, however, no association between BMI and comorbid OCD was observed in the present analyses with AN. The discovery of no relation between BMI and AN was consistent with Mattar et al. (2012) who observed that all indices of psychosocial functioning showed some symptom improvement (e.g., anxiety and depression symptoms) with BMI in AN except for the Y-BOCS. The findings of the present meta-analysis further support the notion that improved BMI is not correlated with significant improvement of obsessive-compulsive symptomatology (and in turn, possibly not for those meeting diagnostic criteria for OCD).

Although BMI was supported as a predictor of OCD prevalence it should be interpreted cautiously as we believe the effect to be confounded with the primary ED diagnosis. Namely, this relation does not survive when the analysis is limited to a single ED subtype (e.g., AN). Furthermore, although there were too few studies to examine it empirically, we believe mean BMI to likewise be confounded with sample type (e.g., inpatient, outpatient, community). Thus, we would expect inpatient samples to have lower mean BMIs but additional research is required to isolate the impact of BMI on OCD prevalence in EDs.

Clinical Implications

OCD comorbidity in EDs presents clinicians with a unique challenge concerning the development of an effective treatment plan and determining which psychotherapeutic intervention will lead to greater symptom reduction. Integration of screening for OCD is crucial when treating those with any ED and is particularly vital for those with ANBP due to the commonality of worsening prognosis. Although the conditions are astonishingly entangled, both psychologically and etiologically, no gold standard treatment approach for their co-occurrence has been identified. Countless studies trialing interventions consistently report that it is imperative that patients not be in an active state of semi-starvation (or a healthy weight be achieved) for any improvement to be observed in either OCD or ED symptomatology (Lewin et al., 2013; Woodside & Staab, 2006).

A review of the most recent trialled approaches by Lewin and colleagues (2013) noted promising findings for combined psychological (e.g., ERP, CBT, family therapy; Fairburn, 2008; Lewin et al., 2013; McCabe & Boivin, 2008; Olatunji et al., 2010; Simpson et al., 2013) and pharmacological approaches for treating OCD in EDs (e.g., SRIs or SSRIs; Lewin et al., 2013; Olatunji et al., 2010; Simpson et al., 2013). Most recently, Lee and colleagues (2020) identified that obsessive-compulsive symptoms such as obsessing and ordering were positively correlated with ED severity at pre-treatment. This suggests that ED patients with OCD may require more intensive interventions. The fact that OCD symptoms do not improve with treatment of the ED suggests that interventions specifically targeting OCD are necessary. It remains to be determined whether these interventions should be delivered concurrently, before, or after ED treatment. In one study, an intervention targeting OCD symptoms and thought-action fusion was associated with a marked reduction in OCD and ED severity post-treatment (Lee et al., 2020). These findings

provide hope for continued refinement and development of psychological interventions targeting shared symptom domains and treatment outcomes.

Strengths, Limitations, and Future Directions

This study had a number of strengths. First, a meta-analytic lifetime prevalence of comorbid OCD of 13.9% and an aggregate estimate of current OCD of 8.7% in EDs were obtained to bring greater clarity to the "true" prevalence of OCD in EDs. Second, ANBP was identified as the ED subtype with the greatest risk for comorbid OCD and those with EDs were at 8.9 times (Lifetime OCD) and 8 times (Current OCD) greater risk for comorbid OCD than the general population. Third, five credible moderators of the co-occurrence of OCD in EDs were identified to equip clinicians with knowledge of the factors that have the potential to increase or decrease the prevalence. Fourth, the six non-credible moderators also highlight factors that at the present time do not exert an influence on the prevalence. Lastly, a spotlight was placed on numerous identified gaps in the literature such as the lack of inclusion of males in ED samples or moderators with potential for credibility with additional data that require future research attention to advance the concurrent treatment of OCD in EDs in many underrepresented samples.

This study also had certain limitations. Firstly, all credible moderators identified were analyzed in separate models to mitigate against the inconsistent reporting of data across the literature and permit inclusion of the maximum number of studies contributing the relevant data whilst maximizing statistical power. As a result, it is important to consider that the probability of any one moderator being credible when modelled separately may be higher than when examined concurrently with other moderators; but whenever possible analyses were conducted as previously described or noted to account for any confounds.

Due to the limited literature available on the non-credible moderators that were exploratory in nature, there was insufficient evidence to be able to draw hypotheses in either direction or to discuss. The literature to date largely examines the moderators in the context of EDs or OCD separately but lacks discussion or evaluation of the predictors impact on their co-occurrence. In terms of study limitations, there was minimal methodological variability within certain moderators, constraining our ability to draw strong conclusions due to the small number of estimates. For example, we were able to locate only 3 lifetime estimates of comorbid OCD in EDs using the MINI and only 6 current estimates with a community sample. As a result, we were unable to conduct exhaustive comparisons between categories for many of the coded moderators. It is recommended that future investigations of OCD comorbidity enhance reporting of sample characteristics and refine diagnostic procedures (e.g., utilization of semi-structured or structured interviews) to inspire future isolation of additional predictors of OCD prevalence in EDs whilst informing approaches for concurrent treatment.

Additionally, it is important for studies to also examine OCD comorbidity in those with current EDs as the assessment of lifetime OCD psychiatric history relies heavily upon retrospective reporting which could result in potential underestimation of comorbid lifetime OCD prevalence. Further, recent cluster and network analyses exploring the relation between obsessive-compulsive symptoms in EDs have found associations between both contamination and intrusions by obsessions (Hasler et al., 2005; Meier et al., 2019). To determine whether specific OCD symptom dimensions significantly overlap with EDs, it is suggested that future studies examine OCD comorbidity in EDs by OCD subtype.

Another limitation was the general lack of inclusion of healthy or representative control groups in research investigating ED-OCD comorbidity. The addition of larger control samples in

future research would allow for further comparison and quantification of more accurate estimates of how much more vulnerable individuals with EDs are for experiencing OCD than the general population. Other diagnostic and demographic populations have also been underrepresented in this research arena, including the exclusion of EDNOS, BED, OSFED, UFED, ARFID, *DSM-5* criteria, and males. This limited the number of available estimates for comparison of disorders such as EDNOS or BED, and further prevented examination of gender as a potential moderator of OCD comorbidity. Similarly, studies examining late age of ED onset and its associated comorbidities are lacking with only one lifetime estimate among those between 20 and 26 years old. Future research with the inclusion of those with late ED onset would assist in teasing apart the differential impact of age of onset as a predictor of either lifetime or current OCD.

Despite 25% of those meeting diagnostic criteria for AN and BN being male (Hudson et al., 2007; Mond et al., 2014), they remain largely underrepresented in eating disorder samples and were largely excluded from recruited samples (Limbers et al., 2018; Mangweth-Matzek & Hoek, 2017; Murray et al., 2017). As a result, this hindered the ability for models to be fit with gender as a predictor of comorbid OCD in EDs or to quantify risk when comparing females to males as they were often excluded from ED samples. Past research has found that those with a primary AN diagnosis were 9.6 times more likely to experience comorbid OCD with the vulnerability being twice as high for males (Cederlöf et al., 2015), and latest trends predicting the prevalence of EDs rising at a more rapid rate in males than females (Smith et al., 2016). As a result, it is increasingly evident that there is a need for greater inclusion of males and reporting of prevalence by gender in this research.

To improve study quality in the ED comorbidity literature, it is recommended future studies recruit more representative and diverse samples with respect to gender, race and ethnicity (e.g.,

inclusion of males and non-Caucasian participants). Methodologically it would be beneficial to incorporate random sampling, calculation of response rates with comparison of responders versus non-responders on demographic variables, report who conducted diagnostic interviews (e.g., trained layperson versus clinical psychologist), and explicitly define the measurement window for assessing comorbid OCD (e.g., current OCD within the past month).

Another noteworthy limitation of the present meta-analysis pertains to the applicability of our findings beyond predominantly Westernized cultures. Although reasonable global representation was obtained across the included studies (see Figure 2), no estimates from Africa, South America, nor Western Europe were available. This not only prevented further examination of geographic region as a predictor of OCD comorbidity in EDs but also limits the breadth of the applicability of the findings to society outside of North America, Asia, Oceania, and Eastern Europe. In recent international collaborations, Brakoulias et al. (2016; 2017) explored the prevalence and pharmacological treatment of comorbid psychiatric conditions in OCD, with the inclusion of samples with primary OCD diagnoses and comorbid AN or BN from Brazil, India, and South Africia. This demonstrates the potential for future research to examine the prevalence of comorbid OCD in EDs in these underrepresented regions of the world and highlights how little is known about the prevalence of comorbid OCD in non-Westernized countries. Estimating the prevalence of comorbid OCD in EDs across all nations would enable future comparisons between geographic regions and examining whether the high prevalence is attributable to Westernized countries or extends across the globe.

Substantial changes pertaining to the classification and diagnosis of both OCD and EDs have been made over the past few decades (Sunday et al., 2001). The change from *DSM-IV* to *DSM-5* criteria for AN resulted in changes to criterion A (e.g., 85th percentile to severity based on

BMI), criterion B (e.g., from underweight to interfering with weight gain), removal of criterion D for amenorrhea, and further specification as ANR and ANBP, but now with the addition of partial or full remission (Substance Abuse and Mental Health Service Administration, 2016a). Substantial changes to OCD diagnostic criteria also occurred from *DSM-IV* to *DSM-5* including the creation of a new category of "obsessive-compulsive and related disorders", and dropping prior diagnostic criteria (e.g., recognizing that the obsessions or compulsions are excessive or unreasonable) and definitional requirements for obsessions (thoughts, impulses, or images are not simply excessive worries about real-life problems; recognition that the obsessional thoughts, impulses, or images are a product of his or her own mind; Substance Abuse and Mental Health Service Administration, 2016b). Given that most of the currently published research focused on OCD used *DSM-IV* or older criteria, newer research using *DSM-5* diagnostic criteria may influence the applicability of the overall findings and bring greater potential for OCD diagnostic criteria to be isolated as a predictor of OCD comorbidity.

Conclusion

The current meta-analysis estimated the comorbidity of OCD across EDs to inform clinical practice by equipping clinicians with empirical support for incorporating OCD screening in ED populations while highlighting credible factors which increase or decrease risk of comorbid OCD in EDs including the ED subtype, sample type, age of ED onset, average BMI, and study quality. Additional research exploring how treatment can best be tailored to target common etiological factors across eating disorder subtypes and OCD subtypes is also essential to improve how treatment plans are devised for these conditions.

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