Journal of Psychiatric Research 141 (2021) 176–191

Contents lists available at ScienceDirect

Journal of Psychiatric Research

journal homepage: www.elsevier.com/locate/jpsychires

Review article

Comorbid obsessive-compulsive disorder in individuals with eating disorders: An epidemiological meta-analysis

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ARTICLE INFO

Keywords:
Obsessive-compulsive disorder
Comorbidity
Eating disorder
Anorexia
Bulimia
Binge eating

ABSTRACT

The present study aimed to provide a precise, meta-analytic estimate of the prevalence of obsessive-compulsive disorder (OCD) amongst those with a current primary eating disorder (ED) diagnosis, and to isolate its predictors. 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 references coded from related review articles 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 were calculated. OCD prevalence was extracted from each study for each ED diagnostic category, along with eleven additional potential moderators. 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 and risk for OCD in EDs to be greatest in anorexia nervosa binge-eating purging type (ANBP). Further, OCD is most prevalent amongst patient samples than samples recruited from the community.

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 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 and Eisen, 1992; Samuels and 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 across disorders (Pinto et al., 2006; Swinbourne and Touyz, 2007; Torres et al., 2013).

Individuals with comorbid conditions often experience increased symptom severity attributable to the reduced effectiveness of treatment (Swinbourne and Touyz, 2007). Quantification of how common comorbid OCD truly is across eating disorder (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 and 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.

Relatively high rates of comorbidity between EDs and OCD may be expected due to the shared genetic and psychological 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

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https://doi.org/10.1016/j.jpsychires.2021.06.035
Received 1 December 2020; Received in revised form 5 May 2021; Accepted 15 June 2021
Available online 19 June 2021

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).

Shared psychological features across conditions, central to both OCD and EDs, are traits such as perfectionism, impulsivity, neuroticism, conscientiousness, harm avoidance, and concern over mistakes (Altman and Shankman, 2009; Dahlenburg et al., 2019; Hoffman et al., 2012; Levinson et al., 2019b; Sallet et al., 2010). Overlap between these disorders is evidenced by the obsessive and compulsive tendencies commonly displayed by eating-disordered individuals (e.g., preoccupation with body weight and shape paired with compulsive weighing or food restriction). However, an additional diagnosis of OCD is only given if the individual also exhibits obsessions and compulsions unrelated to food or weight (APA, 2013). Even in individuals without an ED, a state of semi-starvation can elicit obsessive behaviour (Keys et al., 1950; Phillipou et al., 2018; Swinbourne and Touyz, 2007). Despite differences in how obsessions and compulsions are experienced across the disorders, the purpose of the obsessive and compulsive behaviours observed in both EDs and OCD is to reduce levels of apprehension, anxiety, and overall negative affect (Altman and Shankman, 2009; Swinbourne and Touyz, 2007). Therefore, it may be that common etiological factors explain the high comorbidity between these conditions.

1. Review of current comorbidity estimates

The lifetime prevalence of comorbid OCD in those with primary ED diagnoses is currently ambiguous, with estimates ranging from as low as 0% (Herpertz-Dahlmann et al., 1996) to as high as 79.1% (Halmi et al., 2003). Variation in these estimates may be influenced by methodological differences across studies, including diagnostic criteria or assessment methods, year of study, mean age, or the sampling procedure for the population (e.g., community, outpatient, or inpatient sample). Another recently identified possible contributing factor is whether the co-occurrence of OCD in those afflicted by EDs may vary with earlier age of onset and longer duration of the condition (Milos et al., 2001; Thornton and Russell, 1997). Similarly, differences in developmental factors such as age may play a role as OCD is more likely to be diagnosed in younger than older adults (Fawcett et al., 2020).

Understanding comorbid disorders is important because living with comorbid psychiatric or physical health conditions has a detrimental impact on the prognosis of all conditions, exacerbating symptoms and diminishing treatment efficacy (Sartorious, 2013). For example, the likelihood of attempting suicide is already heightened in those with eating disorders (Ahn et al., 2019; Udo et al., 2019), exacerbated further amongst those coping with OCD (Torres et al., 2006), and leads to elevated risk of ED relapse amongst those with greater severity of OCD symptoms (Carter et al., 2004). Further, the co-occurrence of an anxiety disorder (including OCD in past versions of the DSM) has been shown to be associated with longer hospital stays in those receiving inpatient care for anorexia nervosa (AN; Lievers et al., 2008).

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). Research to date has found that OCD comorbidity is greatest in AN patient samples (Mandelli et al., 2020; Watson et al., 2019) and more prominent in those with AN binge-eating purging type (ANBP) than those with AN restricting type (ANR; Altman and Shankman, 2009). 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 (Margolis et al., 1994).

2. The present study

Given the heterogeneity in estimates 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 logistic regression modelling approach. A secondary goal was to isolate predictors of their co-occurrence and identify those populations most at risk of experiencing comorbid OCD. Although our focus was on samples with a current ED diagnosis, control samples not diagnosed with a current eating disorder (or any other primary psychiatric diagnosis) identified via our search were also recorded so that we could evaluate directly heightened risk in those with eating disorders relative to the general populace.

3. Method

3.1. 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.

3.2. Inclusion and exclusion criteria

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 Fig. 1). Concerns regarding specific studies were resolved through discussion between the first, second, and fourth author.

3.3. Data extraction

The first author screened each article by title and abstract, extracting the following data from those having met inclusion criteria: year of publication, sample size, gender (male, female, mixed), OCD measure (e.g., Structured Clinical Interview for *DSM-IV* [SCID; First et al., 2002], Mini International Neuropsychiatric Interview [MINI; Sheehan et al., 1998], Anxiety and Related Disorders Interview Schedule [ADIS; Grisham et al., 2004]), OCD diagnostic criteria (*DSM-III-R*, *DSM-IV*, *DSM-5*, *ICD-10*), mean age, population (inpatient, outpatient, community, mixed, healthy controls), country, mean age of ED onset, mean duration of illness, OCD measurement window (current or lifetime), mean age of OCD onset, mean BMI, and OCD prevalence separated by ED subtype.

3.4. Quality ratings

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 1

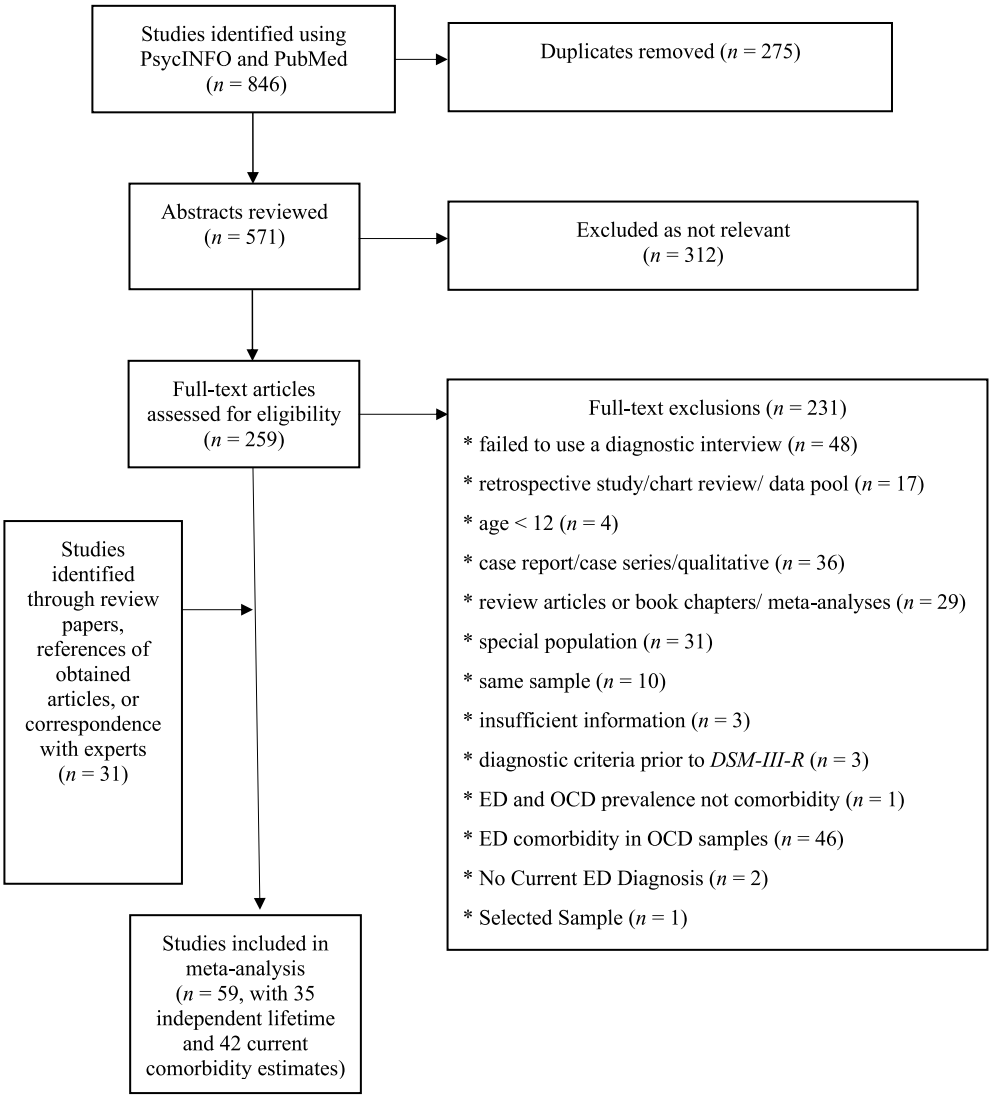

Fig. 1. Meta-analysis inclusion flowchart.

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 E.J.F. with 98.8% agreement and with all disagreements resolved through discussion.

4. 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 *RStudio1.3.1073* (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. Separate models were fit to the lifetime and current data. Further details pertaining to our modelling approach have been provided in the Online Supplement (for other examples, see Fawcett et al., 2020; Fawcett et al., 2019; Fawcett et al., 2016).

For each moderator model, odds ratios (ORs) were reported as a measure of effect size, along with risk ratios for categorical predictors indicating the greater risk associated with one condition over the other. In most cases, either have been calculated directly based on the back-transformed posterior prevalence estimates. Due to variability in reporting of potential moderators, each was analyzed separately to allow for the maximum number of studies to be included, maximizing statistical power. For continuous moderators (e.g., mean age), estimates were centred and scaled prior to fitting the model, with ORs reported for the slope.

The following 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) ED duration, (i) mean BMI, (j) ED subtype, and (k) study quality.

5. Results

5.1. Description of studies

The search initially identified 846 studies, of which we coded 59 studies (see Fig. 1). This process produced 35 estimates of lifetime and 42 estimates of current OCD prevalence among those with a current primary ED diagnosis (see Table 2 for study characteristics). Studies were distributed geographically across North America (Lifetime: 40%, Current: 33.3%), Europe (Lifetime: 40%, Current: 59.5%), Oceania (Lifetime: 8.6%, Current: 2.4%), and Asia (Lifetime: 11.4%, Current: 4.8%). Samples were predominantly female (95.9%), with a mean age of

Table 1
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).*

25.6 years, average ED onset at 18 years, mean ED duration of 6.1 years, and mean age of OCD onset at 13 years. 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, 1992; Schmidt et al., 2008; Stein et al., 2012). All remaining ED samples recruited participants with primary ED diagnoses within the age range of 12 and 60 years. Quality ratings ranged from 4 to 9 with a mean quality score of 6.2 (*SD* = 1.3, see Table 2). 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 1).

5.2. Aggregate prevalence estimate of lifetime and current OCD comorbidity

As depicted in Fig. 2, the aggregate lifetime and current prevalence in a typical sample was estimated to be 13.9% $CI_{95\%}$ [10.4 to 18.1] and 8.7% $CI_{95\%}$ [5.8 to 11.8], respectively. Nonetheless, prediction intervals revealed clear heterogeneity, with the "true" prevalence expected for a new sample with similar demographics and methods to range anywhere from 2.5% to 39.9% (Lifetime) or 1%–37.6% (Current).

5.3. 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 for the sake of brevity shall not be discussed further in-text. Results for all moderators are summarized in Table 3.

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, BN, BED, EDNOS, Any ED, Control); or (c) including all ED subtypes (AN, ANBP, ANR, ANBN, BN, BNP, BNNP, BED, EDNOS, Any ED, Control). An overview of acronym definitions and differentiation between ED subtypes has been outlined in Table 4. 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 3 as (a) population, (b) primary diagnosis, and (c) ED subtype, respectively and depicted in Fig. 3.

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 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

Table 2
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 and 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)	…

(*continued on next page*)

Table 2 (*continued*)

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)
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 and 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. (2012) [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. (2012) [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)	…

(*continued on next page*)

Table 2 (*continued*)

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)
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. (2016)	Norway (Europe)	*DSM-IV*	MINI	CUR	IO	28.0	…	11.7	18.9	8	85.9	6.2 (81)	…

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.*
[†]*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.*

ED. This was particularly true of those diagnosed with AN, *RR* = 12.9 $CI_{95\%}$ [5.7 to 41.9] (Lifetime) and *RR* = 11.9 $CI_{95\%}$ [5.8 to 37.2] (Current), or BN, *RR* = 7.7 $CI_{95\%}$ [3.2 to 25.8] (Lifetime) and *RR* = 6.4 $CI_{95\%}$ [2.8 to 21.0] (Current), 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) and *RR* = 1.9 $CI_{95\%}$ [0.7 to 6.7] (Current) 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.

The analysis broken down by ED subtype supported similar conclusions. 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 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.

Sample Type. We next analyzed the setting from which ED participants were recruited, coded 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 outpatient/inpatient sample. Outpatient/community and mixed inpatient/outpatient/community samples were excluded from the current measurement window analyses (e.g., Boujut et al., 2012; Milos et al., 2001; Wilfley et al., 2000) as there were too few estimates (<3).

Our 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 3, 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 requiring further psychological intervention than those with EDs in the community. Next, the model was re-fit to examine the prevalence of OCD for each population. Supporting our earlier analysis, OCD was least prevalent in community samples, with prevalence increasing for outpatient and again for inpatient samples.

To better evaluate this apparent 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).

[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 diagnoses.*

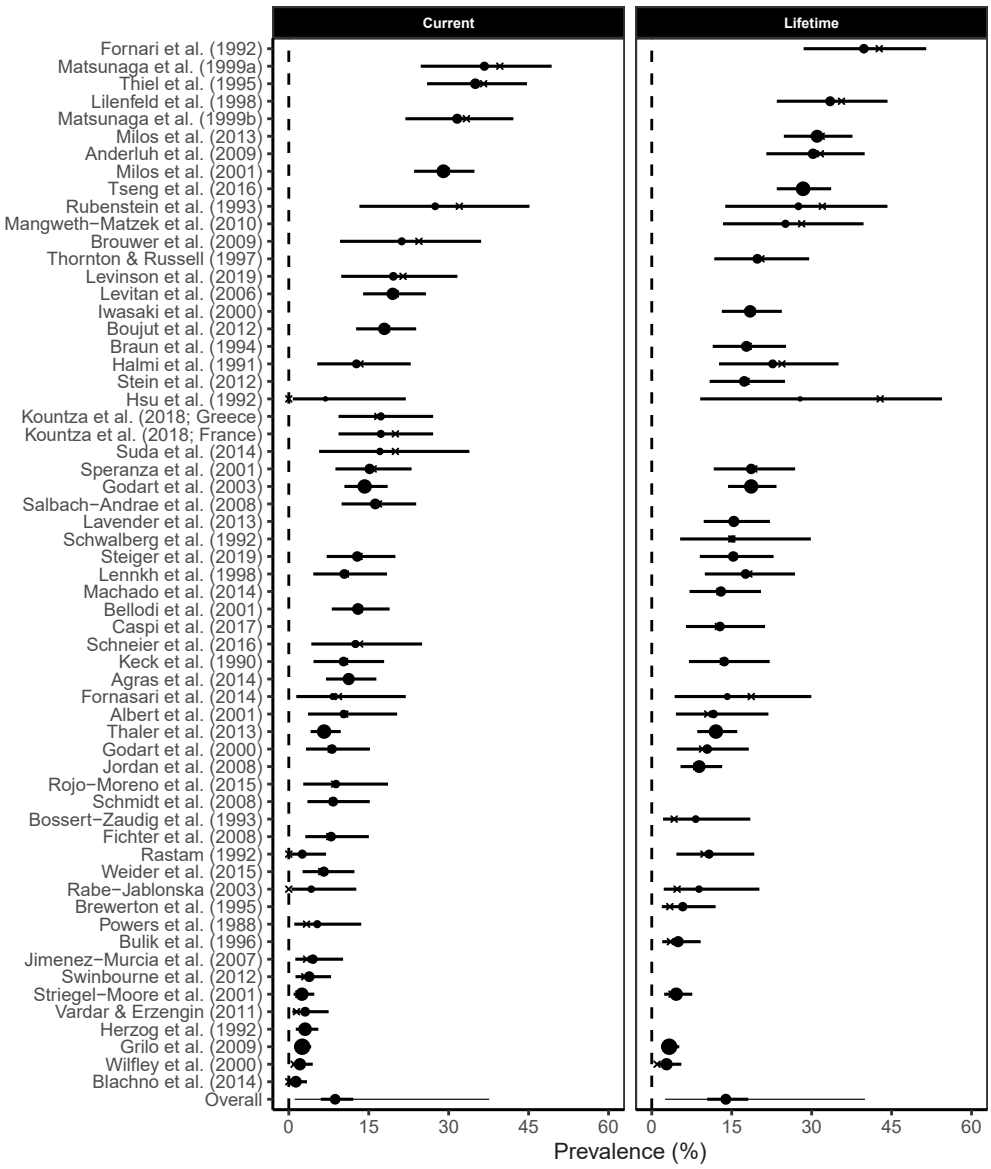

Fig. 2. Forest plots representing OCD prevalence (%) for current and lifetime measurement windows. Note. Points and errors bars reflect model estimates with corresponding 95% confidence intervals. The prevalence reported in each publication is marked with an "X" and the aggregate estimate is illustrated by the final entry labelled "overall." The 95% prediction interval is denoted by the thin grey line radiating from the aggregate point estimate.

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 most broad among outpatients. As a result, comorbid OCD prevalence may increase as ED psychopathology becomes more severe.

ED Age of Onset. Finally, the literature remains ambiguous with respect to the order of ED and OCD occurrence, with some samples showing nearly all cases (~94%) experienced anxiety disorder onset 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 (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 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 ED onset 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.

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 3, 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

Table 3
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 (0.7–1.4)	…	42	…	…	1.0 (0.7–1.5)	…
Geographic Region										
N. America	14	12.5 (8.2–18.6)	…	…	…	14	7.5 (4.3–12.5)	…	…	…
Europe	14	15.7 (10.0–23.4)	3.1 (−5.0 to 11.8)	0.8 (0.4–1.5)	1.3 (0.7–2.2)	25	8.8 (5.6–13.0)	1.3 (−4.4 to 6.3)	1.2 (0.6–2.4)	1.2 (0.6–2.2)
Oceania	3	9.5 (3.9–21.2)	2.9 (−8.8 to 10.8)	1.4 (0.5–3.7)	1.5 (0.5–3.3)	…	…	…	…	…
Asia	4	17.7 (8.5–32.5)	5.1 (−5.5 to 19.9)	0.7 (0.3–1.7)	1.5 (0.6–2.8)	…	…	…	…	…
Age	29	…	…	0.8 (0.6–1.2)	…	35	…	…	1.0 (0.7–1.6)	…
ED Age of Onset	21	…	…	0.7 (0.5–1.0)	…	20	…	…	1.2 (0.7–2.4)	…
ED Duration (Years)	13	…	…	1.3 (0.9–1.8)	…	21	…	…	1.1 (0.7–2.0)	…
BMI	21	…	…	0.5 (0.4–0.6)	…	26	…	…	0.6 (0.4–0.9)	…
OCD Diagnostic Criteria										
DSM-III-R	17	13.4 (8.7–19.5)	…	…	…	11	7.0 (3.5–13.0)	…	…	…
DSM-IV	17	13.6 (9.1–19.7)	0.2 (−7.3 to 7.7)	1.0 (0.5–1.8)	1.0 (0.6–1.8)	31	9.4 (6.3–13.5)	2.3 (−4.2 to 7.5)	0.7 (0.3–1.6)	1.3 (0.7–2.8)
OCD Diagnostic Measure										
SCID	22	11.8 (8.5–16.4)	…	…	…	20	8.6 (5.3–13.4)	…	…	…
MINI	3	19.5 (8.8–36.7)	7.6 (−3.7 to 24.7)	1.8 (0.7–4.5)	1.6 (0.7–3.3)	9	12.2 (6.0–22.8)	3.5 (−3.8 to 14.3)	1.5 (0.6–3.6)	1.4 (0.6–3.0)
SADS	3	25.4 (10.9–45.8)	13.4 (−1.2 to 34.0)	2.5 (0.9–6.5)	2.1 (0.9–4.1)	5	4.4 (1.7–11.0)	4.1 (−2.7 to 9.4)	0.5 (0.2–1.4)	1.9 (0.7–5.3)
Population										
Controls	8	1.6 (0.5–3.5)	…	…	…	7	1.1 (0.4–2.0)	…	…	…
EDs	72	14.2 (10.6–18.5)	12.6 (8.5–17.0)	10.3 (4.2–35.2)	8.9 (3.8–30.1)	89	8.8 (6.2–12.1)	7.7 (5.0–11.0)	8.7 (4.3–26.2)	8.0 (4.0–23.7)
Primary Diagnosis										
Controls	8	1.6 (0.5–3.4)	…	…	…	7	1.1 (0.4–2.0)	…	…	…
AN	31	20.3 (15.9–25.0)	18.6 (14.0–23.5)	16.0 (6.8–52.9)	12.9 (5.7–41.9)	39	13.0 (9.2–17.0)	11.9 (7.9–16.0)	13.6 (6.5–43.2)	11.9 (5.9–37.2)
ANBN	3	23.1 (5.3–54.4)	21.4 (3.6–52.8)	19.0 (3.0–112.2)	14.5 (2.8–61.1)	…	…	…	…	…
Any ED	3	30.1 (15.0–44.7)	28.5 (13.2–43.1)	26.8 (8.4–101.1)	18.9 (6.9–64.2)	3	10.0 (2.0–29.2)	8.9 (0.0–28.1)	10.4 (1.7–58.2)	9.4 (1.7–43.6)
BN	28	12.1 (8.2–17.2)	10.4 (6.1–15.6)	8.6 (3.4–30.0)	7.7 (3.2–25.8)	31	7.0 (3.9–10.9)	5.9 (2.7–9.8)	6.9 (2.9–22.8)	6.4 (2.8–21.0)
BED	4	5.9 (1.8–18.5)	4.2 (−0.2 to 16.8)	4.0 (0.9–21.9)	3.8 (0.9–18.7)	6	2.0 (1.0–4.0)	1.0 (−0.4 to 3.1)	1.9 (0.7–6.9)	1.9 (0.7–6.7)
EDNOS	3	16.7 (4.3–33.8)	15.0 (2.6–32.2)	12.4 (2.4–56.4)	10.4 (2.3–41.2)	8	4.1 (1.2–9.6)	3.0 (0.0–8.6)	3.9 (1.0–16.1)	3.8 (1.0–14.8)
ED Subtype										
Controls	8	1.6 (0.5–3.4)	…	…	…	7	1.1 (0.4–2.0)	…	…	…
AN	9	19.5 (10.4–32.0)	17.9 (8.5–30.4)	14.9 (5.1–56.7)	12.1 (4.6–42.3)	17	9.7 (5.2–15.3)	8.6 (4.0–14.2)	9.8 (4.0–31.3)	8.9 (3.8–27.8)
ANBP	10	22.2 (13.4–31.7)	20.5 (11.5–30.2)	17.3 (6.5–62.1)	13.6 (5.5–46.0)	10	19.2 (13.9–24.7)	18.0 (12.8–23.6)	21.4 (10.5–65.4)	17.4 (9.0–51.9)
ANR	12	18.8 (13.8–23.8)	17.1 (11.9–22.3)	14.0 (6.1–47.0)	11.5 (5.3–37.9)	12	14.2 (8.0–20.7)	13.1 (6.9–19.6)	14.9 (6.3–47.7)	12.9 (5.7–39.7)
ANBN	3	23.3 (5.4–54.3)	21.6 (3.6–52.6)	18.9 (2.9–115.9)	14.4 (2.7–62.9)	…	…	…	…	…
Any ED	3	30.2 (14.7–44.8)	28.5 (12.9–43.0)	26.0 (8.1–98.8)	18.3 (6.6–62.8)	3	10.0 (2.0–29.0)	8.9 (0.8–28.0)	10.4 (1.7–53.9)	9.4 (1.7–40.7)
BED	4	5.8 (1.8–18.6)	4.2 (−0.3 to 16.9)	3.9 (0.9–21.2)	3.7 (0.9–18.1)	6	2.0 (1.0–3.8)	0.9 (−0.4 to 2.9)	1.9 (0.7–6.6)	1.9 (0.7–6.4)
BN	18	11.8 (7.1–18.4)	10.1 (5.0–16.9)	8.2 (3.1–29.5)	7.3 (2.9–25.5)	19	6.4 (3.0–11.7)	5.3 (1.7–10.6)	6.2 (2.3–21.5)	5.9 (2.2–19.7)
BNNP	5	15.4 (7.6–26.5)	13.7 (5.7–24.9)	11.2 (3.7–42.3)	9.6 (3.4–34.1)	6	9.9 (2.8–19.3)	8.8 (1.7–18.3)	10.0 (2.4–37.7)	9.0 (2.3–32.0)
BNP	5	12.0 (6.7–19.4)	10.3 (4.7–17.8)	8.3 (3.0–30.1)	7.4 (2.9–25.7)	6	6.9 (3.7–12.1)	5.8 (2.5–11.0)	6.8 (2.8–22.4)	6.4 (2.7–20.5)
EDNOS	3	16.8 (4.8–34.2)	15.1 (2.9–32.6)	12.3 (2.5–55.7)	10.3 (2.4–40.9)	8	4.1 (1.3–9.8)	3.0 (0.0–8.7)	4.0 (1.0–15.7)	3.8 (1.0–14.5)
Sample Type (Collapsed)										

(*continued on next page*)

Table 3 (*continued*)

	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)
Patient	26	16.5 (12.7–19.9)	…	…	…	33	10.6 (7.5–14.2)	…	…	…
Community	3	5.0 (2.5–10.0)	11.1 (5.1–15.6)	3.7 (1.6–7.8)	3.5 (1.5–6.7)	6	2.7 (1.2–6.2)	7.7 (3.4–11.7)	4.2 (1.7–10.3)	3.9 (1.6–9.2)
Sample Type										
Inpatient	6	14.2 (8.2–23.1)	…	…	…	5	6.7 (2.7–14.5)	…	…	…
Inpatient and Outpatient	8	15.3 (9.6–22.9)	1.0 (−9.3 to 10.7)	0.9 (0.4–2.0)	1.1 (0.6–2.1)	12	11.1 (6.3–17.9)	4.2 (−4.4 to 12.2)	0.6 (0.2–1.6)	1.6 (0.6–4.5)
Outpatient	12	16.6 (10.9–23.6)	2.4 (−8.1 to 11.6)	0.8 (0.4–1.8)	1.2 (0.6–2.2)	16	10.7 (6.4–16.4)	3.9 (−4.7 to 10.9)	0.6 (0.2–1.6)	1.6 (0.6–4.2)
Outpatient and Community	3	5.3 (2.3–11.4)	8.7 (0.5–18.1)	3.0 (1.1–8.2)	2.7 (1.1–6.8)	…	…	…	…	…
Community	3	7.7 (4.2–14.2)	6.4 (−2.0 to 15.6)	2.0 (0.8–4.5)	1.8 (0.8–3.8)	6	3.8 (1.9–7.8)	2.8 (−2.3 to 10.3)	1.8 (0.6–5.1)	1.7 (0.6–4.6)
Inpatient and Outpatient and Community	3	26.5 (13.7–43.5)	12.0 (−3.3 to 30.2)	0.5 (0.2–1.3)	1.9 (0.8–3.9)	…	…	…	…	…
Sample Type (Linear)	32	…	…	1.8 (1.1–3.0)	…	41	…	…	1.9 (1.0–3.3)	…
Study Quality	34	…	…	0.8 (0.6–1.1)	…	41	…	…	0.6 (0.4–0.8)	…

Note. Risk ratios have been inverted to reflect increased risk for the higher prevalence category.
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.*

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 its being confounded with ED diagnosis and being driven by two leverage points with particularly high BMIs relative to the other included samples.

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.

6. Discussion

The primary goal of our 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 reported estimates and isolate predictors of OCD and ED comorbidity to inform diagnostic practices and symptom management. Our 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%. 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, only 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 with 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. 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.

6.1. Greater OCD prevalence in EDs than healthy controls

Whereas we 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 reported by another recent meta-analysis: Mandelli et al. (2020) estimated OCD comorbidity to be instead 18% and 15% for the lifetime and current measurement windows, respectively. The difference in our estimates may be attributable to our inclusion of additional articles, use of more lenient search restrictions (e.g., we used no date or language restrictions), and variation in inclusion criteria (e.g., we included *DSM-III-R* or newer diagnostic criteria and only prospective investigations). Consistent with past research (Mandelli et al., 2020), the present meta-analysis found greater risk for lifetime and current OCD among those with AN compared to BN. We also observed 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, but that ANBP was also identified as the ED subtype at greatest risk for experiencing comorbid OCD (Altman and Shankman, 2009; Margolis et al., 1991).

Our identification of ANBP as the most vulnerable ED subtype for comorbid OCD also differs from Mandelli et al. (2020), who found

Table 4
Overview of ED Subtypes and Acronyms.

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).

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

instead a non-significant trend favouring ANR (Mandelli et al., 2020, Fig. 5).[2] This difference is likely attributable to variation in the included literature as well as the analytic and coding practices between our meta-analyses. The most notable difference for this outcome was perhaps in the coding of Speranza et al. (2001): Whereas we coded 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, Fig. 5) coded 42.8% ($n = 14$; ANR) and 12.9% ($n = 31$; ANBP). 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.

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. 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, those who engage in binge eating and purging behaviours tend to exhibit disordered eating behaviours (e.g., preoccupation with weight or shape followed by ritualistic eating) that closely mirror the cycle of obsessions and compulsions observed in OCD.

Purging behaviors in outpatients with EDs have more generally 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), hinting at perfectionism as a mutually shared vulnerability factor for OCD and EDs (Vanzhula et al., 2021), although 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.

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 subtype 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).

6.2. 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 et al. (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 et al. (2010) suggest that it is plausible 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 with 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.

6.3. Impact of ED age of onset on OCD comorbidity

Although the model for age of ED onset showed heightened risk of lifetime OCD in populations with earlier mean ED onset, this trend was

[2] *Note that we believe the axis label for Fig. 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.*

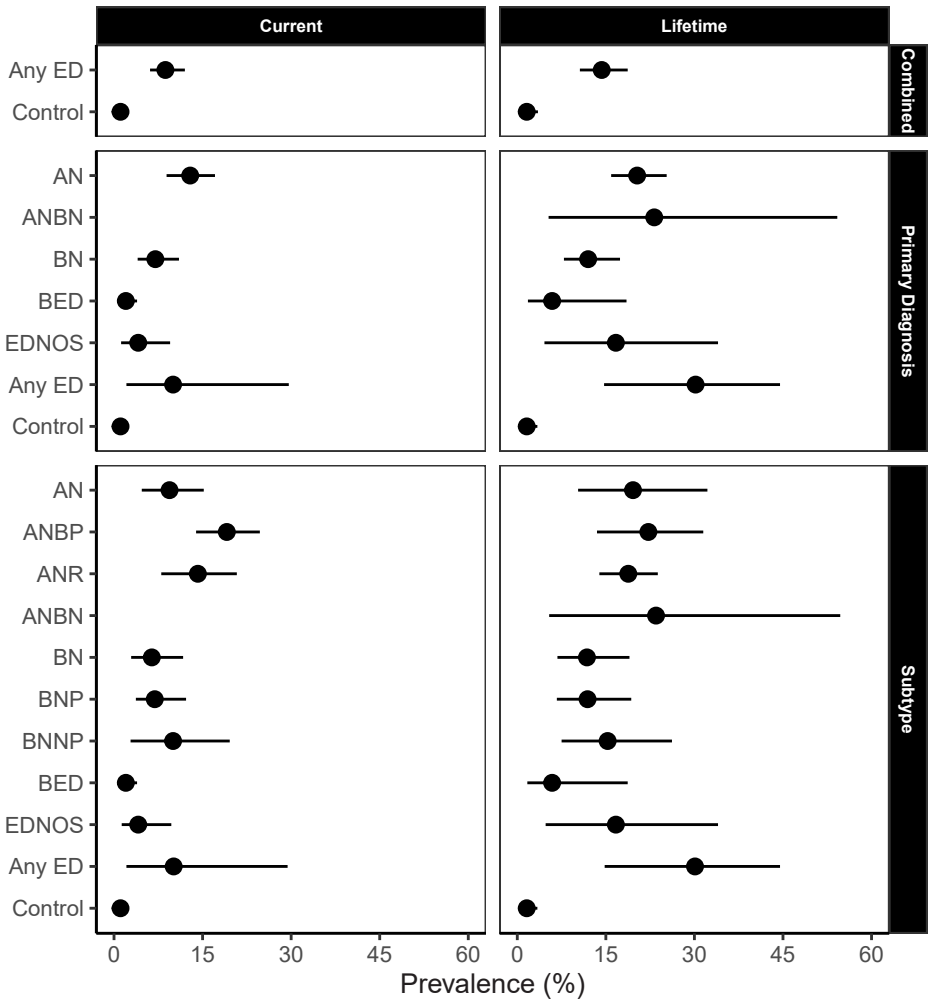

Fig. 3. Forest plots illustrating OCD prevalence (%) by ED subtype for current and lifetime prevalence windows presented separately for the (a) population, (b) primary diagnosis, and (c) ED subtype models. Note. Points and errors bars reflect model estimates with corresponding 95% confidence intervals. The classification "ANBN" was excluded from all of the current models.

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 not be over-interpreted. Our analysis revealed range restriction within this variable as almost all samples report a mean age of ED onset between 16 and 20 years. Stronger conclusions require additional samples focusing on patients with relatively late ED onset.

6.4. *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).

Although BMI was supported as a predictor of OCD prevalence it should be interpreted carefully as we believe the effect to be confounded with the primary ED diagnosis. Specifically, 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.

6.5. *Clinical implications*

OCD 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. Although the conditions are astonishingly entangled, both psychologically and etiologically, no gold standard 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 and Staab, 2006).

A review of the most recent trialled approaches by Lewin et al.

(2013) noted promising findings for combined psychological (e.g., ERP, CBT, family therapy; Fairburn, 2008; Lewin et al., 2013; McCabe and 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 et al. (2020) identified that obsessive-compulsive symptoms such as obsessing and ordering were positively correlated with ED severity at pre-treatment. After participation in an intervention tailored to the strongest obsessive-compulsive symptoms and thought action fusion, a marked reduction in OCD and ED severity was observed at post-treatment (Lee et al., 2020). These findings provide hope for continual refinement and development of psychological interventions targeting shared symptom domains and treatment outcomes.

6.6. Limitations and future directions

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 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. 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 more with EDs, it is suggested that future studies examine OCD comorbidity in EDs by OCD subtype.

There was a lack of general inclusion of healthy or representative control groups in research investigating ED-OCD comorbidity, therefore, the addition of larger control samples in future research would allow for further comparison and quantification of how truly 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, or males from study design and analysis. 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, accessible literature examining late age of ED onset and its associated comorbidities is quite stark with only one lifetime estimate falling 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 and 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 are 9.6 times more likely to experience comorbid OCD with vulnerability being twice as high for males (Cederlöf et al., 2015). 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 literature.

To improve study quality in the ED comorbidity literature, it is recommended future research recruit more representative and diverse samples with respect to gender 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).

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 our overall findings and bring greater potential for OCD diagnostic criteria to be isolated as a predictor of OCD comorbidity.

7. 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.

Funding support

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jpsychires.2021.06.035.

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