

It is illegal to post this copyrighted PDF on any website.

Women Are at Greater Risk of OCD Than Men: A Meta-Analytic Review of OCD Prevalence Worldwide

Emily J. Fawcett, PhD^{a,*}; Hilary Power, MA^b; and Jonathan M. Fawcett, PhD^c

ABSTRACT

Objective: To estimate the worldwide prevalence of obsessive-compulsive disorder (OCD), examine whether women are at greater risk than men, and explore other potential moderators of OCD prevalence to explain variability in community-based epidemiologic studies.

Data Sources: An electronic search of PsycINFO and PubMed was conducted until January 2017, without date or language restrictions, using the keywords *OCD*, *epidemiology*, and *prevalence*. The search was supplemented by articles referenced in the obtained sources and relevant reviews.

Study Selection: Studies were included if they reported current, period, and/or lifetime OCD prevalence (diagnosed according to an interview based on *DSM* or *ICD* criteria) in representative community samples of adults aged 18 years or older. A total of 4,045 studies were retrieved, with 34 studies ultimately included.

Data Extraction: OCD prevalence was extracted from each study alongside 9 moderators: gender, year, response rate, region, economic status, diagnostic criteria, diagnostic interview, interviewer, and age.

Results: The overall aggregate current, period, and lifetime OCD prevalence estimates were 1.1%, 0.8%, and 1.3%, respectively. In a typical sample, women were 1.6 times more likely to experience OCD compared to men, with lifetime prevalence rates of 1.5% in women and 1.0% in men. There was also a trend toward younger adults' being more likely to experience OCD in their lifetime than older adults. All findings demonstrated moderate heterogeneity.

Conclusions: Women are typically at greater risk of experiencing OCD in their lifetime than men.

J Clin Psychiatry 2020;81(4):19r13085

To cite: Fawcett EJ, Power H, Fawcett JM. Women are at greater risk of OCD than men: a meta-analytic review of OCD prevalence worldwide. *J Clin Psychiatry*. 2020;81(4):19r13085.

To share: <https://doi.org/10.4088/JCP.19r13085>

© Copyright 2020 Physicians Postgraduate Press, Inc.

^aStudent Wellness and Counselling Centre, Memorial University of Newfoundland, St John's, Newfoundland, Canada

^bDepartment of Psychology, University of Regina, Regina, Saskatchewan, Canada

^cDepartment of Psychology, Memorial University of Newfoundland, St John's, Newfoundland, Canada

*Corresponding author: Emily J. Fawcett, PhD, Student Wellness and Counselling Centre, UC-5000, Memorial University of Newfoundland, St John's, NL, A1C 5S7, Canada (efawcett@mun.ca).

Past research has found anxiety disorders to be the sixth leading cause of disability worldwide,¹ resulting in increased health care service utilization and greater work absenteeism, particularly in women.² One disorder that has gained considerable recognition is obsessive-compulsive disorder (OCD), which is characterized by the presence of obsessions (ie, intrusive thoughts, images, or urges) that are mitigated by compulsive behaviors or mental acts aimed at remedial distress.³ This condition was originally classified as an anxiety disorder but has undergone significant revision with the publication of the *Diagnostic and Statistical Manual of Mental Disorders*, Fifth Edition (*DSM-5*),³ in which it is now its own diagnostic entity (obsessive-compulsive and related disorders). Regardless of its conceptualization, this disorder is now thought to be more common than once imagined⁴—although individual prevalence estimates range anywhere from <0.1%⁵ to 4.6%.⁶

The prevalence of this condition is concerning given that obsessions and compulsions impose serious economic and psychosocial hardships. For example, OCD has been associated with impaired quality of life, lower marriage rate, loss of work productivity or unemployment, and adverse effects on family members.^{7–12} The broad comorbidity and chronicity of OCD further accentuate functional impairments and complicate treatment trajectories.¹³ To make matters worse, OCD rarely occurs in isolation, with up to 92.3% lifetime comorbidity in treatment-seeking individuals,¹⁴ with anxiety, depressive, and eating disorder comorbidity most common in women and psychotic, developmental, and autism spectrum disorders and attention-deficit/hyperactivity disorder most common in men.¹⁵ Comorbidity in OCD is associated with greater anxiety and depressive symptoms, suicidal behaviors, and previous treatment.¹⁴

Although the *DSM-5* points to a slightly greater prevalence rate in women than men,³ past reviews are inconsistent, citing either a slight increase in women,¹⁶ an approximately equal gender ratio,¹⁷ or an inconsistent sex-specific OCD prevalence rate.¹⁸ Results from individual studies have likewise been mixed, with some demonstrating considerably higher prevalence for women than men,^{19–28} others showing only slightly higher prevalence in women,^{5,29–39} others finding no difference,^{40,41} and even some showing slightly higher prevalence in men.^{30,31,42,43} These inconsistencies have frustrated strong conclusions concerning the prevalence of OCD—and particularly whether women are at greater risk.

The present meta-analysis addresses these concerns by meta-analytically synthesizing extant prevalence estimates and quantifying variation across samples. Our primary goal is to

Clinical Points

- This study is the first to provide a meta-analytic estimate of the worldwide prevalence of obsessive-compulsive disorder (OCD) in men and women.
- Women were 1.6 times more likely to suffer from OCD at some point in their lives compared to men, with aggregate lifetime prevalence estimates of 1.5% and 1.0%, respectively, in women and men. Gender differences vary moderately from sample to sample.
- Future research is needed to clarify the genetic and environmental factors driving gender differences in prevalence and symptom expression in OCD and whether these factors are specific to OCD or represent a more general susceptibility to mood and anxiety disorders in women.

estimate the prevalence of OCD in the general populace and to adjudicate whether women are truly at greater risk than men. However, as a secondary goal, we also carry out exploratory moderator analyses evaluating claims as to whether prevalence has been increasing over time, differs across regions, or is impacted by measurement decisions, such as the diagnostic criteria or interview used. We feel these to be important questions as their answers will provide valuable information pertaining to the rates of OCD in community samples independent of treatment seeking.⁴

METHODS

Literature Search

We followed the Preferred Reporting Items for Systematic Review and Meta-Analyses (PRISMA) guidelines.⁴⁴ We conducted a search of the online resources PsycINFO and PubMed using the Boolean search phrase: (“*obsessive compulsive disorder*” OR “*obsessive-compulsive disorder*” OR “*OCD*”) AND (“*epidemiology*” OR “*prevalence*”). The search was conducted until January 11, 2017, without date or language restrictions and was supplemented by all relevant reference sections and epidemiologic reviews within this area. The first author screened all abstracts and read the full text of all articles being considered for study eligibility.

Inclusion and Exclusion Criteria

For inclusion, a study was required to (a) be an original study assessing a representative community sample at either the regional or national level; (b) use a diagnostic interview according to *DSM* or *ICD* criteria (with the whole population interviewed or a 2-step survey methodology); (c) report current (1-week, 1-month), period (6-month, 12-month), or lifetime estimates of OCD prevalence; and (d) include participants aged 18 years or older.

All studies in which diagnoses were based on retrospective chart review, clinical records, or insurance claims were excluded, along with studies diagnosing OCD according to self-report surveys. Studies examining the

prevalence of OCD in children and/or adolescents were excluded as we were interested in the prevalence of OCD in adults. Furthermore, studies in which all respondents were over the age of 65 years were excluded, along with studies with restricted age ranges (eg, 18–24 years, reports from a birth cohort at a specific age). We also excluded studies targeting non-community samples or special populations (eg, outpatients, university students, military populations, patient populations, ethnic subgroups).

Data Extraction

The first author extracted the following data from each article: author name, year of publication, sample size, sample gender makeup (female, male, mixed), OCD prevalence, country/region, diagnostic interview (eg, Composite International Diagnostic Interview [CIDI], Mini-International Neuropsychiatric Interview [MINI], Diagnostic Interview Schedule [DIS]), diagnostic criteria (eg, *DSM-III*, *DSM-III-R*, *DSM-IV*, *ICD-10*), and prevalence measurement window (current, period, lifetime). In cases in which a given study reported only prevalence separated by gender, the aggregate estimate was calculated by combining those groups to produce a mixed estimate used in our secondary analyses, treating samples containing only one gender as a 100% mixture in one direction or the other.

Moderator Analyses

As stated earlier, our primary goal was to estimate the aggregate prevalence of OCD and evaluate its relative risk in women compared to men. However, 8 additional moderators were also considered on an exploratory basis, including (a) year of publication, (b) region in which the study was conducted, (c) response rate for the study, (d) age, (e) interviewer, (f) economic status of the country where the study was conducted, (g) diagnostic criteria, and (h) diagnostic interview.

Following Baxter et al,⁴⁵ response rate was categorized as low (<60%), average (60%–79.9%), or excellent (80% and higher). For age, there was significant variation in the reported ranges, with the most common being either 18–34, 35–54, and 55+ or 18–24, 25–44, and 45–65 years. These age groupings—or other groupings that could be combined to approximate them—were recoded by the first author into coarse categories corresponding to young adults, middle-aged adults, and older adults. The individual administering the interview was coded as either a trained interviewer (eg, lay person), student/allied mental health practitioner (eg, psychology or sociology students, medical students, social workers, psychiatric nurses), or a clinician (eg, physician, psychologist, psychiatry intern or psychiatrist). Finally, World Bank income classifications of developing, emerging, or developed were used to code country economic status.⁴⁵

Due to the small number of studies and variation in reporting standards across variables, each moderator was considered in a separate model. This allowed us to maximize the number of studies incorporated into any given model, thereby maximizing statistical power.

Quality Assessment

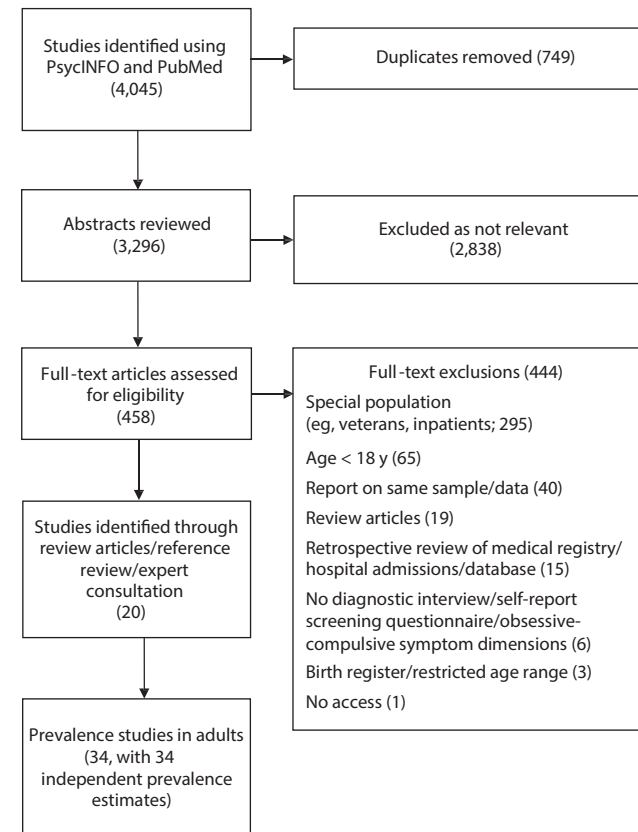
To assess study quality, the first author generated a 10-point checklist to assess bias pertaining to key methodological criteria. Standardized and reliable quality assessment tools specifically designed for epidemiologic studies^{46,47} were modified and expanded to include additional questions pertaining to the specification of eligibility criteria and diagnostic interview administration. Quality ratings from 0 to 10 for each study are provided (see Table 1), with higher scores reflecting higher-quality studies. Below are the exact questions and scoring information:

1. Was the target population clearly defined? Were demographic characteristics of the study population given? (eg, age, sex, ethnicity, income; not reported in the article/only one of the above listed = 0, two or more of the above listed = 1)
2. Were the eligibility criteria clearly specified? (neither specified in the article = 0, inclusion or exclusion criteria clearly specified = 1)
3. Was either of the following ascertainment methods used? (must be one or the other)
 - a. Probability sampling OR
 - b. Entire population surveyed (unclear/no, or convenience sample used = 0, yes = 1)
4. Was the response rate adequate? (eg, below 70%/not reported = 0, 70% or higher = 1)
5. Was information included about people who completed the study versus those who refused? For instance, did they differ on any demographic variables? (no/not reported in the article = 0, yes = 1)
6. Was the sample representative of the target population? (no/unclear = 0, yes = 1)
7. Were data collection methods standardized? (no/unclear = 0, yes = 1)
8. Were validated criteria used to assess for the presence/absence of disorder (OCD)? (eg, validated scale or diagnostic tool; no/unclear = 0, yes = 1)
9. Who administered the diagnostic interview? (trained lay person/not reported = 0, trained clinician/researcher/allied mental health worker or trainee = 1)
10. Were the OCD prevalence estimates given with confidence intervals or standard errors (not reported = 0, reported = 1)

Effect Size Calculation and Analysis

Fully Bayesian multilevel binomial regression models were used, implemented using brms 2.9.0^{56,57} within R 3.5.2.⁵⁸ No effect size calculations were needed prior to analysis as the models themselves were fit using the sample size and the number of individuals with OCD in that sample. Prevalence was estimated within each model as a logit-transformed proportion,⁵⁹ but has been back-transformed and reported as a percentage for ease of interpretation. Each model incorporated random intercepts (and slopes, when appropriate) accounting for variability across samples, which

Figure 1. Flowchart of Studies Included in the Meta-Analysis



were in-turn used to calculate prediction intervals.⁶⁰ For our primary analyses—calculating the overall prevalence of OCD and evaluating gender differences—we desired estimates for each measurement period (current, period, or lifetime). Rather than fitting separate models, we used multivariate models treating each period as a separate dependent measure while accounting for correlations between these estimates. Models addressing gender differences included only samples made entirely of women or entirely of men (ie, excluding mixed samples). The resulting output was used to estimate both the prevalence within each population and the relative risk for women as compared to men.

All other analyses used instead the mixed lifetime prevalence estimates that were reported in text or calculated manually using the reported estimates. Bayes factors were calculated for each moderator analysis evaluating evidence for inclusion of the relevant variable, although we adopt a holistic view and report predictions from the model incorporating the relevant moderator alongside those values. Tests of publication bias were not undertaken owing to the fact that their underlying assumptions are not readily applicable to prevalence estimates (see Borenstein^{61p173}). Further information pertaining to our modeling approach is provided in Supplementary Appendix 1 and described in greater detail elsewhere.^{62–65}

Table 1. Characteristics of OCD Studies

Study (First Author)	Country (Region)	Economic Status	Criteria	Measure	Interviewer	Response Rate	Quality	Prevalence Measurement Window	Prevalence, % (n)		
									Mixed	Male	Female
Canino ³⁰	Puerto Rico (North America)	Developed	DSM-III	DIS	Trained interviewer	Excellent	9	Period	1.8 (1,513)	1.3 (655)	2.3 (859)
Canino ³⁰	Canada (North America)	Developed	DSM-III	DIS	Trained interviewer	Average	8	Lifetime	3.2 (1,513)	3.3 (654)	3.1 (859)
Bland ^{29a}	US (North America)	Developed	DSM-III	ASI	Trained interviewer	Excellent	7	Lifetime	3.0 (3,258)	2.8 (1,330)	3.1 (1,928)
Henderson ²¹	US (North America)	Developed	DSM-III	DIS	Trained interviewer	Average	7	Current	2.8 (497)	2.4 (250)	3.2 (247)
Karno ²²	US (North America)	Developed	DSM-III	DIS	Trained interviewer	Average	7	Current	1.3 (18,571)	1.1 (7,617)	1.5 (10,954)
								Period	1.6 (18,571)	1.4 (7,617)	1.8 (10,954)
								Lifetime	2.5 (18,571)	2.0 (7,617)	2.9 (10,954)
Hwu ^{36b}	Taiwan (Asia-Pacific)	Developed	DSM-III	DIS	Student/AMHP	Excellent	8	Lifetime	0.94, 0.54, 0.3 (5,005, 3,004, 2,995) ^b	0.81, 0.38, 0.37 (2,464, 1,581, 1,582)	1.07, 0.71, 0.22 (2,541, 1,423, 1,413)
Oakley-Browne ^{25c}	New Zealand (Asia-Pacific)	Developed	DSM-III	DIS	Trained interviewer	Average	See Wells ²⁸	Period	1.0 (1,498)	0.6 (504)	1.4 (994)
Wells ^{28c}	New Zealand (Asia-Pacific)	Developed	DSM-III	DIS	Trained interviewer	Average	8	Lifetime	2.2 (1,498)	1.0 (504)	3.4 (994)
Lee ³⁹	South Korea (Asia-Pacific)	Developed	DSM-III	DIS	Student/AMHP	Excellent	8	Lifetime	2.29 (3,134)	2.21 (1,490)	2.38 (1,644)
Ciarlo ^{40d}	US (North America)	Developed	DSM-III	DIS	Trained interviewer	Average	7	Current	0.8 (4,745)	0.8 (2,265)	0.8 (2,479)
Wittchen ⁴⁸	Germany (Europe)	Developed	DSM-III	DIS	Clinician	Average	10	Period	1.79 (483)	1.79 (232)	2.29 (251)
								Lifetime	2.03 (483)		
Chen ³¹	China (Asia-Pacific)	Emerging	DSM-III	DIS	Trained interviewer	Excellent	8	Lifetime	1.05 (7,229)	0.87 (3,443)	1.22 (3,786)
Kolada ^{41a}	Canada (North America)	Developed	DSM-III	DIS	Trained interviewer	Average	See Bland ²⁹	Period	1.6 (3,258)	1.6 (1,330)	1.6 (1,928)
Stein ⁴⁹	Canada (North America)	Developed	DSM-IV	CIDI	Trained interviewer	Average	7	Current	3.1 (2,261)		
Bijl ⁴³	The Netherlands (Europe)	Developed	DSM-III-R	CIDI	Trained interviewer	average	8	Current	0.3 (7,076)	0.3 (3,588)	0.2 (3,488)
								Period	0.5 (7,076)	0.5 (3,588)	0.4 (3,488)
								Lifetime	0.9 (7,076)	0.9 (3,588)	0.8 (3,488)
Szódóczy ⁵⁰	Hungary (Europe)	Developed	DSM-III-R	DIS	Student/AMHP	Excellent	7	Lifetime	2.86 (2,953)		
Grabe ⁵¹	Germany (Europe)	Developed	DSM-IV	CIDI	Trained interviewer	Average	8	Period	0.39 (4,075)	0.02 (2,045)	0.37 (2,030)
								Lifetime	0.5 (4,075)	0.4 (2,045)	0.1 (2,030)
Abou-Saleh ⁵	United Arab Emirates (Middle East)	Developed	ICD-10	CIDI	Student/AMHP	Excellent	9	Lifetime	0.07 (1,394)	0.14 (711)	0 (683)
Kringlen ²³	Norway (Europe)	Developed	DSM-III-R	CIDI	Student/AMHP	Low	9	Period	0.7 (2,066)	0.3 (928)	1.0 (1,138)
								Lifetime	1.6 (2,066)	0.7 (928)	2.3 (1,138)
Andrade ⁴²	Brazil (South America)	Emerging	ICD-10	CIDI	Trained interviewer	Average	8	Current	0.3 (1,464)	0.4 (622)	0.1 (842)
								Period	0.3 (1,464)	0.4 (622)	0.1 (842)
								Lifetime	0.3 (1,464)	0.4 (622)	0.1 (842)
Caraveo-Anduaga ¹⁹	Mexico (North America)	Emerging	ICD-10	CIDI	Trained interviewer	Average	6	Period	1.0 (1,932)	0.7 (871)	1.2 (1,061)
								Lifetime	1.4 (1,932)	0.8 (871)	1.8 (1,061)
Çilli ²⁰	Turkey (Middle East)	Emerging	DSM-IV	CIDI	Clinician	Excellent	8	Period	3.0 (3,012)	2.5 (1,420)	3.3 (1,592)
Jacobi ³⁷	Germany (Europe)	Developed	DSM-IV	CIDI	Clinician	Excellent	9	Current	0.4 (4,181)	0.2 (1,913)	0.6 (2,268)
								Period	0.7 (4,181)	0.6 (1,913)	0.9 (2,268)
Mohammadi ²⁴	Iran (Middle East)	Emerging	DSM-IV	SADS	Clinician	Excellent	8	Lifetime	1.8 (25,178)	0.7 (12,628)	2.8 (12,520)
Crino ³⁵	Australia (Asia-Pacific)	Developed	DSM-IV	CIDI	Trained interviewer	Average	7	Period	0.6 (10,641)	0.6 (4,705)	0.7 (5,936)
								Current	0.5 (10,641)	0.5 (4,705)	0.5 (5,936)
Gureje ⁵²	Nigeria (Africa)	Developing	DSM-IV	CIDI	Trained interviewer	Average	8	Period	0.1 (4,984)		
								Lifetime	0.1 (4,984)		

(continued)

RESULTS

Description of Studies

Of the 4,045 studies initially identified, 34 studies were coded (see Figure 1), including 34 independent estimates of either current, period, or lifetime prevalence. Countries were categorized into 6 geographical regions, including North America (26.5%), South America (2.9%), Africa (2.9%), Middle East (14.7%), Asia-Pacific (32.4%), and Europe (20.6%). Study characteristics are summarized in Table 1, including overall quality scores, which ranged from 6 to 10 (mean [SD] = 7.97 [1.0]).

Aggregate Prevalence Estimate

Prior to evaluating our moderators, we first estimated the aggregate prevalence of OCD within the mixed samples. As depicted in Figure 2, this analysis produced an aggregate back-transformed lifetime prevalence of 1.3%, (95% CI, 0.9% to 1.8%). To evaluate the degree of heterogeneity across samples, we next calculated the corresponding prediction interval, reflecting the range of “true” prevalence estimates that might be expected in a new sample similar to those included in the present model. This prediction interval ranged from 0.1% to 4.9%, suggesting a moderate degree of heterogeneity aligned with the range of observed scores described in the introduction. Estimates for comparable current or period analyses are also provided in Figure 2.

Moderator Analyses

Having established an aggregate estimate for the lifetime prevalence of OCD, we next evaluated evidence for each moderator, as summarized in Table 2. Due to its central role in our theorizing, our analysis of gender used all measurement windows (current, period, lifetime); however, due to inconsistent coding and missing data, it was not possible to do the same for the remaining moderators. For that reason, and due to their exploratory nature, the remaining moderator analyses were conducted only for the lifetime measurement window, for which we have the largest sample of estimates. Of our moderators, only gender and age produced credible effects. For the sake of exposition, all remaining models are described in Supplementary Appendix 1 and summarized in Table 2.

Gender. One of our primary goals was to interrogate evidence favoring greater risk of OCD for women than men. As depicted in Figure 3 and summarized in Table 2, this claim was supported for lifetime estimates: There was a higher lifetime prevalence in women (mean = 1.5%; 95% CI, 1.0%

Table 1 (continued).

Study (First Author)	Country (Region)	Economic Status	Criteria	Measure	Interviewer	Response Rate	Quality	Prevalence Measurement			
								Window	Mixed	Male	Female
Karam ⁵³	Lebanon (Middle East)	Emerging	DSM-IV	CIDI	Trained interviewer	Average	7	Period	0.1 (2,857)		
Cho ³²	South Korea (Asia-Pacific)	Developed	DSM-IV	CIDI	Student/AMHP	Average	10	Period Lifetime	0.6 (6,275) 0.6 (6,275)	0.5 (3,524) 0.6 (3,524)	0.7 (2,751) 1.0 (2,751)
Keqing ³⁸	China (Asia-Pacific)	Emerging	DSM-IV	SCID	Clinician	Excellent	9	Current Lifetime	2.46 (20,716) 2.6 (20,716)	2.29 (10,343)	2.63 (10,373)
Alhasnawi ⁶	Iraq (Middle East)	Emerging	DSM-IV	CIDI	Trained interviewer	Excellent	7	Period Lifetime	3.6 (4,332) 4.6 (4,332)		
Cho ³³	South Korea (Asia-Pacific)	Developed	DSM-IV	CIDI	Trained interviewer	Excellent	8	Period Lifetime	0.5 (6,510) 0.6 (6,510)	0.4 (2,581) 0.5 (2,581)	0.6 (3,929) 0.7 (3,929)
Ruscio ²⁶	US (North America)	Developed	DSM-IV	CIDI	Trained interviewer	Average	6	Period Lifetime	1.2 (2,073) 2.3 (2,073)	0.5 (1,036) 1.6 (1,036)	1.8 (1,037) 3.1 (1,037)
Williams ⁵⁴	Australia (Asia-Pacific)	Developed	DSM-IV	SCID	Clinician	Average	9	Lifetime			1.4 (1,095)
Chong ³⁴	Singapore (Asia-Pacific)	Developed	DSM-IV	CIDI	Trained interviewer	Average	9	Period Lifetime	1.1 (6,616) 3.0 (6,616)	2.8 (3,299)	3.2 (3,317)
Skapinakis ²⁷	Greece (Europe)	Developed	ICD-10	CIS-R	Trained interviewer	Low	8	Current	1.69 (4,894)	1.27 (2,425)	2.1 (2,469)

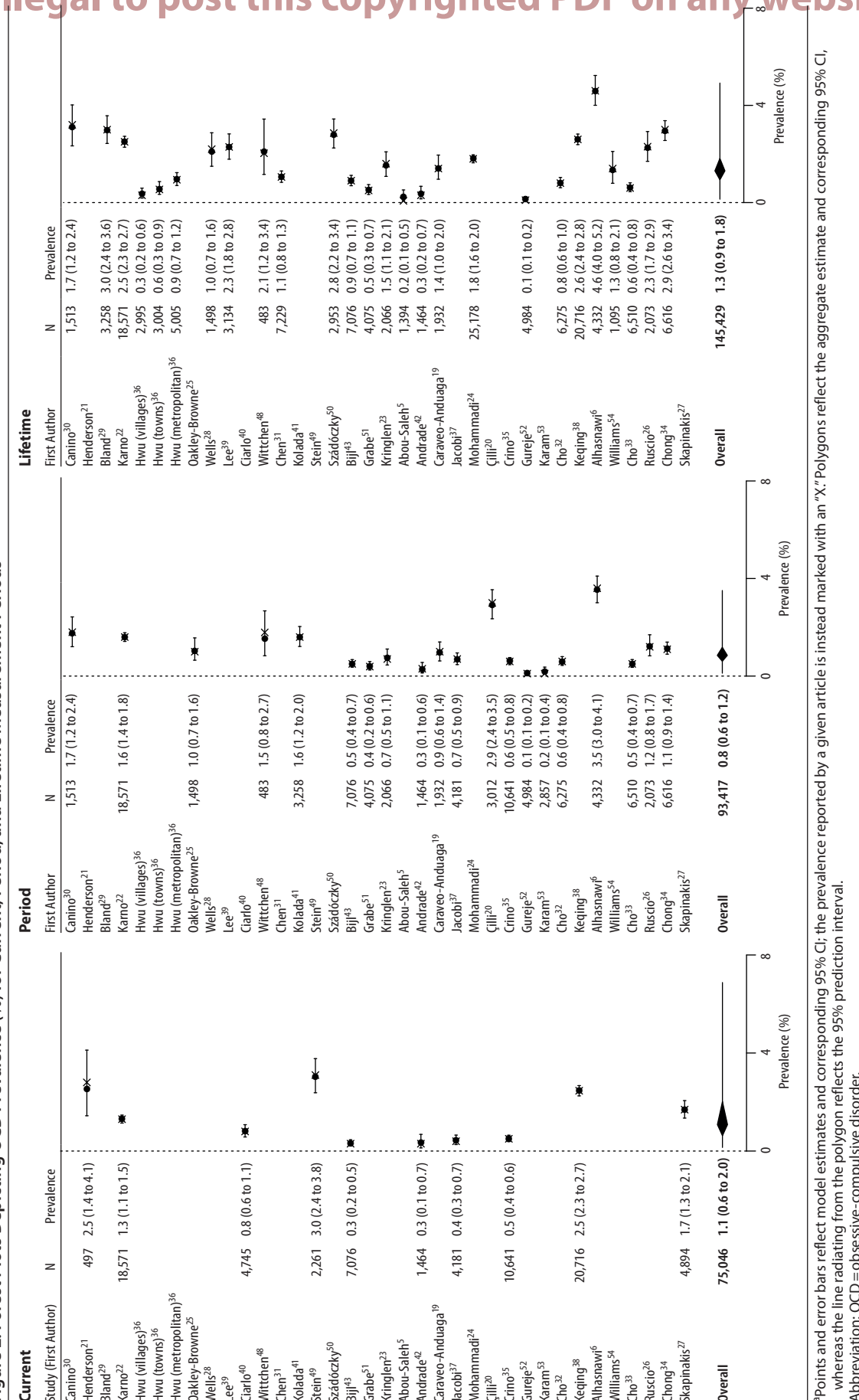
^aBland et al²⁹ and Kolada et al⁴¹ report on the same sample, but report lifetime and period prevalence, respectively.

^bPrevalence rates for Hwu et al³⁶ are separated according to 3 geographical regions: Metropolitan Taipei, 2 small towns, and 6 rural villages, respectively.

^cWells et al²⁸ and Oakley-Browne et al²⁵ report on the same sample, but report lifetime and period prevalence, respectively.

^dOCD estimates were taken from Sachs-Ericsson and Carlo.⁵⁵

Abbreviations: AMHP = allied mental health practitioner; ASI = Anxiety Symptoms Interview; CIDI = Composite International Diagnostic Interview; CIS-R = Clinical Interview Schedule-Revised; DIS = Diagnostic Interview Schedule; DSM = *Diagnostic and Statistical Manual of Mental Disorders*; DSM-III = DSM, Third Edition; DSM-III-R = DSM, Third Edition, Revised; DSM-IV = DSM, Fourth Edition; ICD-10 = *International Classification of Diseases*, Tenth Revision; OCD = obsessive-compulsive disorder; SADS = Schedule for Affective Disorders and Schizophrenia; SCID = Structured Clinical Interview for DSM-IV-TR Axis I Disorders.

Figure 2. Forest Plots Depicting OCD Prevalence (%) for Current, Period, and Lifetime Measurement Periods^a

^aPoints and error bars reflect model estimates and corresponding 95% CI; the prevalence reported by a given article is instead marked with an "X." Polygons reflect the aggregate estimate and corresponding 95% CI, whereas the line radiating from the polygon reflects the 95% prediction interval. Abbreviation: OCD = obsessive-compulsive disorder.

Table 2. Summary of Moderator Analyses for Lifetime OCD Prevalence^a

Moderator	No. of Estimates	Prevalence, % (95% CI)	Difference, % (95% CI)	Odds Ratio (95% CI)	Bayes Factor ^b
Gender (current)					1.0 (+)
Male	9	0.9 (0.5 to 1.8)	
Female	9	1.1 (0.6 to 2.2)	0.2 (0.0 to 0.5)	1.2 (1.0 to 1.5)	
Gender (period)					32.2 (+)
Male	15	0.7 (0.5 to 1.0)	
Female	15	1.0 (0.7 to 1.4)	0.3 (0.1 to 0.6)	1.4 (1.2 to 1.9)	
Gender (lifetime)					44.1 (+)
Male	21	0.9 (0.6, 1.4)	
Female	22	1.5 (1.0 to 2.1)	0.5 (0.2 to 0.9)	1.6 (1.2 to 2.0)	
Year	26	1.00 (0.9 to 1.1)	42.9 (–)
Region					4.9 (–)
North America	5	2.1 (1.1 to 3.8)	
Asia/Pacific	11	1.2 (0.8 to 2.0)	0.8 (–0.4 to 2.5)	0.6 (0.3 to 1.3)	
Europe	5	1.4 (0.7 to 2.9)	0.6 (–1.0 to 2.4)	0.7 (0.3 to 1.7)	
Middle East	3	1.4 (0.6 to 3.4)	0.6 (–1.3 to 2.4)	0.7 (0.3 to 1.9)	
Diagnostic criteria					1.4 (–)
ICD-10	3	0.7 (0.3 to 1.6)	
DSM-III	13	1.5 (0.9 to 2.4)	0.8 (–0.2 to 1.8)	2.3 (0.8 to 6.2)	
DSM-IV	10	1.2 (0.7 to 2.1)	0.5 (–0.5 to 1.5)	1.9 (0.7 to 5.2)	
Diagnostic interview					1.0 (+)
CIDI	12	0.9 (0.5 to 1.6)	
DIS	11	1.6 (0.9 to 2.8)	0.6 (–0.3 to 1.9)	1.7 (0.8 to 3.7)	
Response rate					2.3 (–)
Average (60%–79%)	7	1.5 (0.8 to 2.9)	
Excellent (80%)	10	1.4 (0.8 to 2.5)	0.1 (–1.2 to 1.6)	0.9 (0.4 to 2.2)	
Age ^c					6.9 (–)
Young adult	6	2.7 (2.0 to 3.8)	
Middle age	7	2.6 (2.0 to 3.6)	0.1 (–0.4 to 0.7)	1.0 (0.8 to 1.2)	
Older adult	7	2.0 (1.4 to 2.9)	0.7 (0.0 to 1.4)	0.7 (0.5 to 1.0)	
Interviewer					1.4 (–)
Clinician	4	1.7 (0.7 to 3.8)	
Trained interviewer	10	1.7 (1.0 to 3.1)	0.0 (–1.6 to 2.1)	1.0 (0.4 to 2.7)	
Student/AMHP	6	0.9 (0.4 to 2.0)	0.8 (–0.5 to 2.8)	0.5 (0.2 to 1.5)	
Economic status					2.4 (–)
Developed	19	1.3 (0.9 to 2.0)	
Emerging	6	1.5 (0.8 to 3.0)	0.2 (–0.7 to 1.7)	1.2 (0.5 to 2.5)	

^aCurrent and period estimates are also provided for gender.

^bBayes factors were calculated by comparing models inclusive of each moderator to a comparable model excluding that moderator; “+” means the reported value supports inclusion and “–” means the reported value supports exclusion. Bayes factors for gender were instead calculated using the Savage-Dickey method, permitting separate values for each measurement window.

^cExcludes the outlier identified in text—a comparable model including the outlier is reported in text and corresponds to a Bayes factor of 39.5 in support of the null model.

Abbreviations: AMHP = allied mental health practitioner, CIDI = Composite International Diagnostic Interview, DIS = Diagnostic Interview Schedule, OCD = obsessive-compulsive disorder.

to 2.1%) than in men (mean = 0.9; 95% CI, 0.6 to 1.4), resulting in a difference of 0.5% (95% CI, 0.2% to 0.9%). Supplementary risk ratios calculated for lifetime estimates revealed women are 1.6 (95% CI, 1.2 to 2.0) times more likely than men to suffer from OCD at some point in their lives. However, the prediction interval surrounding this value ranged from 0.7 to 4.0, suggesting variance in the underlying “true” gender effects. As summarized in Table 2, current and period estimates demonstrated a similar pattern, although this pattern was less compelling within the former, owing to the small number of current estimates.

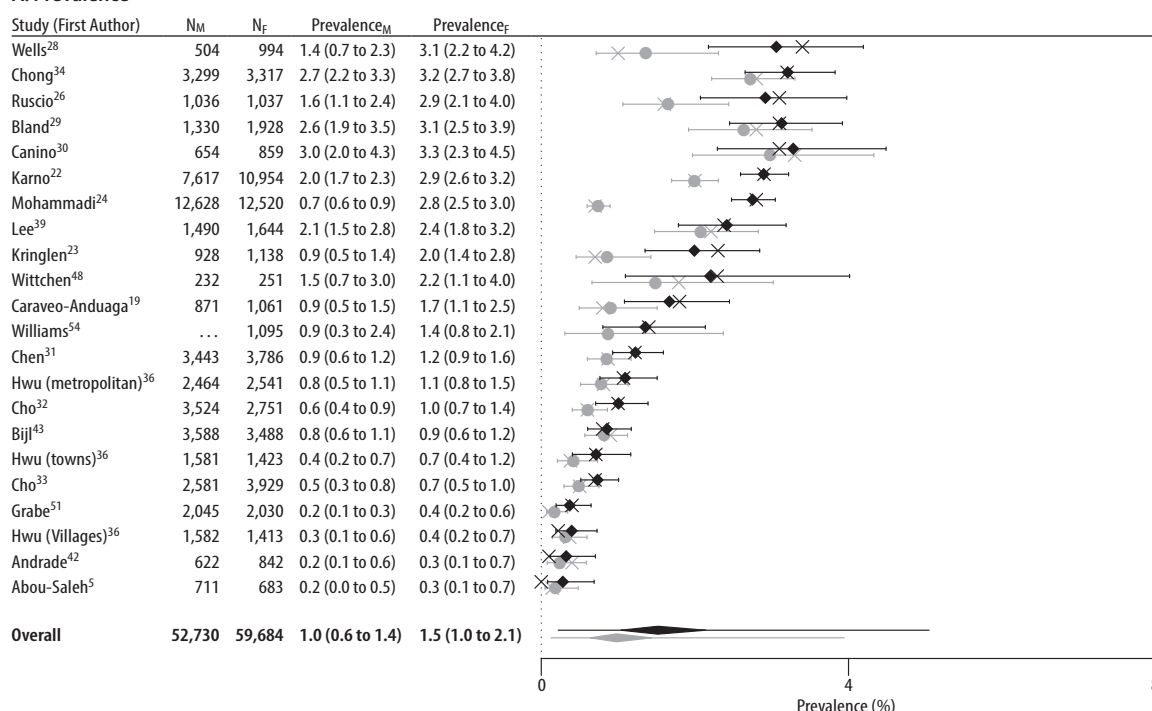
Age group. Due to heterogeneous reporting of age categories across the included studies, estimates were loosely categorized into young adult, middle-aged, and older adult. Whereas the young adult (mean = 2.7%; 95% CI, 2.0% to 3.6%) and middle-aged (mean = 2.6%; 95% CI, 2.0% to 3.4%) groups demonstrated minimal difference in prevalence (mean = 0.1%; 95% CI, –0.4% to 0.7%), the older adult group

presented a slight numerical reduction in lifetime prevalence (mean = 2.3%; 95% CI, 1.5% to 3.5%). Nonetheless, the magnitude of the difference between the younger and older adults was small and highly uncertain (mean = 0.4%; 95% CI, –0.7% to 1.3%). As noted in Table 2, an omnibus Bayes factor comparing this model to a model excluding age found strong support against age as a moderator.

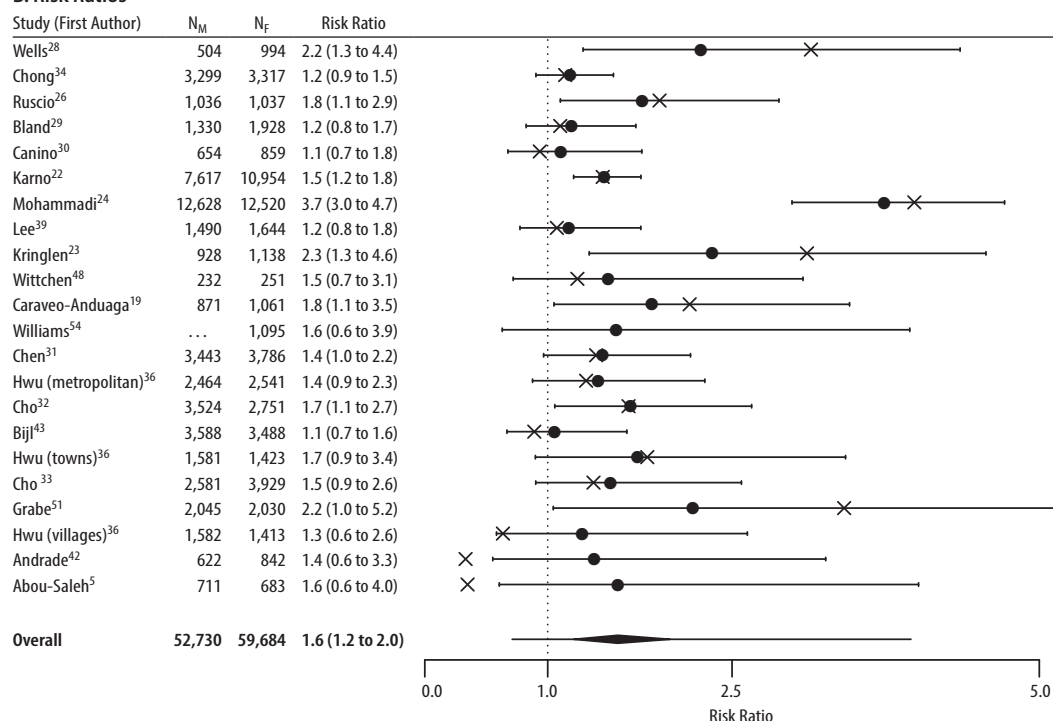
However, inspection of the data contributing to each estimate revealed an apparent outlier within this model: Whereas most studies depict a common pattern with younger adults demonstrating similar lifetime prevalence to middle-aged adults, either of whom demonstrate greater lifetime prevalence than older adults, Canino et al³⁰ present instead a strikingly linear increase in lifetime prevalence, with older adults demonstrating over twice the probability of having suffered from OCD (5.3%) relative to younger (1.9%) or middle-aged (2.5%) participants. This pattern is striking for two reasons. First, all remaining studies in

Figure 3. Forest Plots Depicting (A) OCD Prevalence by Gender and (B) Corresponding Risk Ratios for the Lifetime Measurement Period^a

A. Prevalence



B. Risk Ratios



^aPrevalence_F and dark gray represent OCD prevalence rates (%) for women; Prevalence_M and light gray represent OCD prevalence rates (%) for men. Points and error bars reflect model estimates and corresponding 95% CI; the prevalence or risk ratio reported by a given article is instead marked with an "X." Polygons reflect the aggregate estimate and corresponding 95% CI, whereas the line radiating from the polygon reflects the 95% prediction interval.

Abbreviations: N_F = number of female subjects, N_M = number of male subjects, OCD = obsessive-compulsive disorder.

It is illegal to post this copyrighted PDF on any website.

this model demonstrate at least some degree of reduction in lifetime prevalence for older as compared to younger adults (smallest reported difference = 0.2%^{20,24,28}). Second, the lifetime prevalence reported for older adults in Canino and colleagues³⁰ sample is almost twice the prevalence estimate of its nearest competitor and over 3 times the lowest prevalence estimate despite having one of the lower prevalence estimates within their younger sample; the difference between either the younger or middle-aged adults and older adults is likewise much larger than for other studies, suggesting a multivariate outlier.

For this reason, we refit our model excluding Canino et al.³⁰ A similar pattern was observed within this subsample, with younger (mean = 2.7%; 95% CI, 2.0% to 3.8%) and middle-aged (mean = 2.6%; 95% CI, 2.0% to 3.6%) adults, again demonstrating minimal difference in prevalence (mean = 0.1%; 95% CI, -0.4% to 0.7%), but now with the older adult group presenting a much lower lifetime prevalence estimate (mean = 2.0%; 95% CI, 1.4% to 2.9%). The difference between younger and older adults was also more compelling (mean = 0.7%; 95% CI, 0.0% to 1.4%). Put differently, this model suggests that younger adults are 1.4 (95% CI, 1.0 to 1.8) times more likely to have been diagnosed with OCD in their lifetime than older adults. However, given that this finding is both small and predicated on exclusion of an apparent outlier, we urge caution in its application. In fact, despite the observed difference between younger and older adults in our parameter estimates, the corresponding Bayes factor still supports exclusion of age as a moderator—albeit only weakly. Additional data are necessary before strong conclusions may be drawn.

DISCUSSION

The current study is—to our knowledge—the first to provide a meta-analytic estimate of the worldwide prevalence of OCD in men and women. Our primary goal was to evaluate the prevalence of this condition and to determine whether women are truly at greater risk than men. As a secondary goal, we also evaluated 8 additional exploratory moderators to isolate potential sources of heterogeneity within these prevalence estimates. Our aggregate lifetime prevalence estimate of OCD across the included studies was 1.3%—collapsed across gender—with a moderate degree of variation between samples. Period and current prevalences were lower, with estimates of 0.8% and 1.1%, respectively. Importantly, our analysis of gender revealed women to be 1.6 times more likely to suffer from OCD at some point in their lives compared to men, with aggregate prevalence estimates of 1.5% and 1.0%, respectively. This pattern was observed across all measurement windows. We discuss this finding prior to considering our secondary analyses.

Gender Differences in the Prevalence and Expression of OCD

A significant gender difference in OCD prevalence is consistent with other large-scale studies examining gender

effects in common mental disorders. At least two prior systematic reviews and meta-regressions have identified women as being at greater risk of mood or anxiety disorders than men. In particular, Baxter et al⁴⁵ found women to be twice as likely to have an anxiety disorder, with gender proving to be their most influential moderator. Steel et al⁷⁰ further noted that whereas women were at higher risk of mood and anxiety disorders, men were more prone to substance use disorders compared to women. Importantly, our meta-analysis is the first to link this increase to OCD specifically, rather than an “any anxiety disorder” estimate with the disorders examined varying across studies (for a critique of this approach, see Fawcett et al⁶²).

However, despite evidence supportive of gender differences across anxiety disorders as a whole, past studies comparing OCD in men and women have often found little evidence for such an effect.¹⁷ For example, OCD has been found to be slightly more prevalent in women than men,⁵ comparable,⁴⁰ or even slightly less prevalent in women than men.³⁰ It would be tempting to dismiss these inconsistencies as statistical noise, except for a similar pattern observed within our prediction intervals. Specifically, we observed evidence favoring variation in the “true” prevalence from one sample to the next such that the finding of no “true” difference—or even a slight reversal (ie, men being at greater risk)—is possible within a given sample. The implication is that women are most often at greater risk but might not be across all populations. The isolation of those circumstances under which women are at greater risk than men—and those under which men are at greater risk than women—represents a major goal for future research. Nonetheless, the present findings demonstrate the former to be far more prevalent under those conditions commonly studied within the published literature.

Inspection of the current sample reveals no obvious explanation for this variation; studies showing comparable or lower prevalence estimates for women than men are not characterized by any particular geographic region, assessment measure, or diagnostic criteria. One factor that might explain some variability would be the distribution of pregnant or postpartum women within those samples. The perinatal period is thought to be a time of particular vulnerability for OCD, with pregnant and postpartum women being 1.5 to 2.0 times more likely to experience OCD than women in the general population.⁶⁹ Although we were unable to examine this factor as a moderator—because the percentage of perinatal women is rarely reported—it stands to reason that studies with more perinatal women would show heightened prevalence estimates since those populations are known to be at greater risk.

In line with this idea, hormonal influences in general are a promising avenue of investigation with respect to why women might be at greater risk of OCD compared to men. Such an account is supported—for example—by the existence of clear gender differences in pediatric samples (with boys at greater risk) that dissipate with the onset of puberty.⁷¹ It has therefore been postulated that reproductive

You are prohibited from making this PDF publicly available.

It is illegal to post this copyrighted PDF on any website.

hormones and associated major reproductive events such as menarche, pregnancy, postpartum, and menopause may play a role in the onset or exacerbation of OCD symptoms. Supporting this idea, over 25% of women with OCD report the onset of their diagnosis being related to a major reproductive event.⁷² While menarche has been the most commonly implicated event,⁷³ the perinatal period has also been linked to symptom onset.⁷⁴ Further, whereas some women show no change or even improvement in preexisting symptoms across reproductive events,⁷⁵ approximately 30%–50% of premenstrual, pregnant, postpartum, or menopausal women^{72–74,76–78} have been found to experience exacerbated symptoms, possibly resulting from susceptibility to fluctuations in reproductive hormones.

Gonadal hormones such as estrogen, progesterone, and oxytocin have been found to affect the course of OCD, very likely through their effect on serotonergic, dopaminergic, and glutamatergic neurotransmitter systems.⁷⁹ A “hormone-related” OCD subtype has been proposed, as women with onset or exacerbation of OCD symptoms in the perinatal period were more likely to also experience premenstrual exacerbation of symptoms.⁷⁴ This proposed subtype is consistent with a larger theory of hormonal sensitivity in mood disorders, which posits that some women show heightened sensitivity to intense hormonal fluctuations, with major reproductive events viewed as “windows of vulnerability.”^{80–82} However, numerous other conditions have also shown exacerbation across the menstrual cycle (eg, eating disorders, asthma),⁸³ leading to the proposal of a broader hormonal sensitivity syndrome across the lifespan.⁸⁴

In addition to being more prevalent in women, OCD is also expressed differently within these populations. For example, women are known to present greater contamination and cleaning symptoms compared to men and to demonstrate greater eating disorder and impulse-control comorbidity.⁸⁵ Male patients are in turn more likely to have an earlier age at onset and chronic course, single status, greater tic and substance use disorder comorbidity, and more sexual-religious and aggressive symptoms. Explanations pertaining to these differences in presentation might broadly be categorized as arising from biological, psychological, or sociocultural causes.

The fact that contamination and cleaning symptoms are twice as common among women as among men⁸⁶ and are most associated with perinatal onset⁸⁷ suggests a biological component. Supporting this assertion, genetic studies suggest that contamination/cleaning and hoarding are the two most heritable symptom dimensions,⁸⁸ with contamination linked to specific genes relative to other symptom dimensions.⁸⁹ Furthermore, brain regions such as the fronto-orbital cortex, which show greater activation in individuals with OCD contamination and cleaning symptoms upon provocation,⁹⁰ have a higher density of sex steroid receptors in development⁹¹ and have significantly larger brain volume in women.⁹⁰

Psychological factors are also relevant to understanding gender differences in symptom presentation. For example,

disgust sensitivity is greater in women⁷¹ and mediates gender differences in contamination fears.⁹² Patients with contamination/washing symptoms who view washing-related pictures show activation of the insula, a brain region critical to the recognition and expression of disgust.⁹⁰ Disgust sensitivity has been found to heighten during the first trimester of pregnancy,⁹³ facilitating increased precautionary measures such as food aversion to animal products, which may help to reduce food-borne illness⁹⁴ at a critical time of fetal development.⁹⁵ Therefore, the commonality of contamination and cleaning symptoms and their association with reproductive events is also consistent with an evolutionary account of OCD, in which heightened parental preoccupations and harm avoidance are adaptive mechanisms that increase the likelihood of infant survival and successful reproduction.⁹⁶

Lastly, cross-cultural research has demonstrated differences in OCD presentation, including contamination cognitions.⁹⁷ Thus, cultural norms and societal gender roles may influence attitudes, beliefs, and customs around cleanliness, increasing the risk of contamination OCD in women. Societal influences may also affect the reporting of OCD symptoms in men. With sexual, religious, and aggressive symptoms more common in men, prevalence may be underestimated due to stigma or shame, potentially delaying diagnosis or treatment seeking.⁹⁸ For instance, in a large survey,⁹⁹ over 50% of individuals with self-reported OCD reported delaying or avoiding receiving treatment due to feelings of shame about having a problem or needing help.

Greater Prevalence in Younger Than in Older Adults

Beyond gender, the only moderator to produce a credible difference was age, which revealed (on removal of a multivariate outlier) that younger adults were 1.4 times more likely to be diagnosed with OCD in their lifetime than older adults. This finding is consistent with Baxter and colleagues⁴⁵ systematic review and meta-regression of the global prevalence of anxiety disorders, which found that older adults were 20% less likely to suffer from such disorders. Indeed, this trend may be true of mental disorders more broadly. For instance, a large nationally representative survey in the United States found the 12-month and lifetime prevalence of mood, anxiety, and substance disorders to be lower for older adults compared to younger adults.¹⁰⁰ While physical health and cognitive function deteriorate at an accelerated rate across the adult lifespan, self-reported mental health measures suggest a linear increase or better mental health among older adults.¹⁰¹

Numerous methodological explanations have been proposed for the reduction of mental illness such as mood and anxiety disorders across the lifespan. For instance, cross-sectional studies are particularly at risk for cohort effects, such that earlier-born cohorts may have been raised in a unique historical period with different cultural circumstances than later cohorts.^{102,103} It is possible that older cohorts are less willing to report psychiatric symptoms due to stigma or that attitudes toward mental health problems differ between

You are prohibited from making this PDF publicly available.

It is illegal to post this copyrighted PDF on any website.

age groups.^{100,102} Furthermore, there may be genetic or environmental risk factors exerting greater influence in recent cohorts,¹⁰⁰ although there is limited evidence of environmental risk factors for OCD.¹⁰⁴ Survivor effects may also be present, such that mortality rates are higher for those with mental illness or that those who survive longer into older age have better mental health outcomes.^{101,102} Life expectancy differs depending on the presence of mental illness, with a recent meta-analysis¹⁰⁵ concluding that the mortality rate is 2.2 times higher in people with mental disorders. Finally, sampling bias may also drive these effects, as older adults with chronic conditions leading to hospitalization, long-term care placement, or institutionalization are rarely sampled by community surveys.^{100,102}

However, setting aside methodological concerns, psychological factors may also contribute to the lower risk of mental disorders in older samples. For instance, older adults may show increased resilience due to the development of greater coping skills or an immunity to stressful life experiences,^{100,106} although this finding should speak more to current than lifetime prevalence. Lastly, cognitive functioning may exert an influence on lifetime prevalence rates, as older adults may have poor recall of historical psychiatric symptoms due to memory difficulties.^{100–102}

Importantly, the finding that younger adults are at greater risk of OCD relies on a small number of estimates and exclusion of an outlier; further, this difference emerges only in the contrast conducted between the younger and older age groups and is not supported by the corresponding Bayes factor. Future studies should report age in a more consistent fashion to allow a more powerful test of this hypothesis.

Other Moderators

None of our remaining moderator analyses produced compelling results. The fact that economic status failed to show any difference is in contrast to Baxter et al,⁴⁵ who found a higher prevalence of anxiety disorders in emerging and developed countries compared to developing countries. However, this contrast is very likely due to our exclusion of developing countries, as we had only a single estimate. It is also possible that the moderation of anxiety disorder prevalence by economic status is not specific to OCD.

Weak regional effects were found, with a trend showing relatively higher OCD prevalence in North America compared to Asia-Pacific, Europe, or the Middle East, but none of these comparisons were credible. This is in contrast to the meta-regression of Baxter et al,⁴⁵ in which Euro-Anglo cultures showed greater risk for anxiety compared to all other cultures. Although higher prevalence of OCD may be predicted in North America, these findings are difficult to contrast as the present study compared North America to Europe, Asia-Pacific, and the Middle East, whereas the Euro-Anglo cultural categorization in Baxter et al⁴⁵ included Western Europe, North America, and Australasia.

There was likewise no evidence to suggest that OCD prevalence changed over the past 26 years. Although community-based epidemiologic evidence shows no increase

in the prevalence of anxiety disorders over time,⁴⁵ a recent Finnish study¹⁵ found increased incidence for the treatment of OCD in specialist mental health care settings. Similarly, age-specific prevalence of childhood psychiatric diagnoses including OCD have increased over 20 years across different Scandinavian birth cohorts, according to medical discharge registries.¹⁰⁷ However, increases in reported diagnoses may be more reflective of greater awareness and recognition of OCD, improvements in availability of services, and/or increases in treatment referrals.^{15,107}

Strengths, Limitations, and Future Directions

The current study is the first to meta-analytically estimate the prevalence of OCD worldwide and also the first to explore gender differences in the prevalence of this disorder. Representative community samples were used along with the gold standard of semistructured or structured diagnostic interviews for OCD diagnosis. Concerns have been raised about the overdiagnosis of OCD according to lay person-administered interviews such as the CIDI and DIS,⁴⁹ especially as one study⁴⁹ showed a precipitous drop from 3.1% to 0.6% in OCD prevalence with clinical reappraisal interviews. However, the current study found high concordance between prevalence estimates derived by clinicians and trained interviewers. While it is possible that OCD prevalence may be reduced through clinician-administered semistructured interviews such as the Structured Clinical Interview for DSM, we were unable to resolve this question in the current study given the preponderance of estimates using the CIDI and DIS to the exclusion of other instruments. Although future research may help to resolve this question, large-scale epidemiologic studies employing clinicians rather than lay interviewers would be considerably more cumbersome.

Methodological limitations should also be considered, although they are not unique to this meta-analysis. Some of the moderator analyses in the current study may have been underpowered, with small sample sizes in terms of subcomparisons. For instance, in our diagnostic interview moderator analysis, we had to collapse *DSM-III* and *DSM-III-R* into a single estimate, and only 3 studies used *ICD-10* diagnostic criteria. Similarly, in our geographic region moderator analysis, we only had 5 studies from the Middle East versus as many as 11 studies from Asia-Pacific. There are quite likely other significant variables that may moderate OCD prevalence that were not assessed in the present study, including variables that are difficult to quantify, such as awareness and stigma associated with OCD across regions and cultures. Higher prevalence of OCD in women compared to men may be driven by additional confounding demographic factors, such as marital status, employment/job status, ethnicity, and age, as when in the Epidemiologic Catchment Area survey¹⁰⁸ gender differences disappeared after controlling for these variables. However, basic demographic information such as employment status, income, and ethnicity are reported rarely and inconsistently across studies, making meta-analysis difficult. Further reporting of demographic variables such as marital status and

You are prohibited from making this PDF publicly available.

ethnicity broken down by their respective OCD prevalence rates would allow future meta-analyses to examine risk ratios. This information could be included in an appendix or online supplement, or authors could be more responsive through e-mail to requests for additional data. Finally, there may be interaction effects between the moderators examined in the study, differentially affecting gender, although there was insufficient power to investigate these higher-order interactions. Such interaction effects may be an important area for future research, as the number of prevalence studies focusing on the new *DSM-5* criteria expands.

There is currently a scarcity of literature examining gender differences in the prevalence and expression of different OCD spectrum disorders (eg, body dysmorphic disorder), making this an important area for future research. Continuing to examine gender differences in OCD prevalence and symptomatology is of central importance to understanding the underlying etiology of OCD and

the relative contribution of genetic and environmental influences.

CONCLUSION

The current study confirms that women are typically at greater risk for OCD compared to men. While the aggregate lifetime prevalence estimate of OCD was below 2%, this condition is highly comorbid with other psychiatric and neurodevelopmental disorders. Age was the most compelling exploratory moderator, showing younger adults to be at greater risk than older adults. Future research is needed to explain additional factors contributing to heterogeneity in prevalence estimates across samples and to elucidate whether the genetic and environmental factors driving gender differences in prevalence and symptom expression are specific to this condition or represent a more general susceptibility to mood and anxiety disorders in women.

Submitted: September 14, 2019; accepted February 25, 2020.

Published online: June 23, 2020.

Author contributions: E.J.F. and J.M.F. developed the project. E.J.F. conducted the literature search and coded the studies. J.M.F. conceived of the statistical model. J.M.F. implemented, conducted, and presented all statistical analyses. E.J.F. took the lead on the manuscript, but all authors contributed.

Potential conflicts of interest: The authors report no potential conflicts of interest.

Funding/support: This research received no direct financial support from any granting agency.

Acknowledgments: We would like to thank and acknowledge Maryam Michael (undergraduate research volunteer at the Student Wellness and Counselling Centre, Memorial University) for her help with the literature search.

Supplementary material: Available at PSYCHIATRIST.COM.

REFERENCES

- Baxter AJ, Vos T, Scott KM, et al. *Psychol Med*. 2014;44(11):2363–2374.
- McLean CP, Asnaani A, Litz BT, et al. *J Psychiatr Res*. 2011;45(8):1027–1035.
- American Psychiatric Association. *Diagnostic and Statistical Manual for Mental Disorders*. Fifth Edition. Washington, DC: American Psychiatric Association; 2013.
- Horwath E, Gould F, Weissman MM. Epidemiology of anxiety disorders. In: Tsuang MT, Tohen M, Jones PB, eds. *Textbook in Psychiatric Epidemiology*. 3rd ed. Chichester, UK: John Wiley & Sons Ltd; 2011:311–328.
- Abou-Saleh MT, Ghubash R, Daradkeh TK. *Soc Psychiatry Psychiatr Epidemiol*. 2001;36(1):20–28.
- Alhasnawi S, Sadik S, Rasheed M, et al; Iraq Mental Health Survey Study Group. *World Psychiatry*. 2009;8(2):97–109.
- Calvocoressi L, Lewis B, Harris M, et al. *Am J Psychiatry*. 1995;152(3):441–443.
- Koran LM, Thienemann ML, Davenport R. *Am J Psychiatry*. 1996;153(6):783–788.
- Leon AC, Portera L, Weissman MM. *Br J Psychiatry suppl*. 1995;166(2):19–22.
- Magliano L, Tosini P, Guarneri M, et al. *Eur Psychiatry*. 1996;11(4):192–197.
- Rasmussen SA, Eisen JL. *Psychiatr Clin North Am*. 1992;15(4):743–758.
- Nestadt G, Samuels J. *Int Rev Psychiatry*. 1997;9(1):61–72.
- Pinto A, Mancebo MC, Eisen JL, et al. *J Clin Psychiatry*. 2006;67(5):703–711.
- Torres AR, Shavitt RG, Torresan RC, et al. *Compr Psychiatry*. 2013;54(7):1042–1052.
- Rintala H, Chudal R, Leppämäki S, et al. *BMC Psychiatry*. 2017;17(1):64.
- Weissman MM, Bland RC, Canino GJ, et al; The Cross National Collaborative Group. *J Clin Psychiatry*. 1994;55(suppl):5–10.
- Lochner C, Stein DJ. *Arch Women Ment Health*. 2001;4(1):19–26.
- Somers JM, Goldner EM, Waraich P, et al. *Can J Psychiatry*. 2006;51(2):100–113.
- Caraveo-Anduaga JJ, Bermúdez EC. *Salud Ment*. 2004;27(2):1–6.
- Çilli AS, Telcioglu M, Aşkin R, et al. *Compr Psychiatry*. 2004;45(5):367–374.
- Henderson JG Jr, Pollard CA. *J Clin Psychol*. 1988;44(5):747–752.
- Karno M, Golding JM, Sorenson SB, et al. *Arch Gen Psychiatry*. 1988;45(12):1094–1099.
- Kringlen E, Torgersen S, Cramer V. *Am J Psychiatry*. 2001;158(7):1091–1098.
- Mohammadi MR, Ghanizadeh A, Rahgozar M, et al. *BMC Psychiatry*. 2004;4(1):2.
- Oakley-Browne MA, Joyce PR, Wells JE, et al. *Aust N Z J Psychiatry*. 1989;23(3):327–340.
- Ruscio AM, Stein DJ, Chiu WT, et al. *Mol Psychiatry*. 2010;15(1):53–63.
- Skapinakis P, Bellis S, Koupidis S, et al. *BMC Psychiatry*. 2013;13(1):163.
- Wells JE, Bushnell JA, Hornblow AR, et al. *Aust N Z J Psychiatry*. 1989;23(3):315–326.
- Bland RC, Orn H, Newman SC. *Acta Psychiatr Scand suppl*. 1988;338:24–32.
- Canino GJ, Bird HR, Shrout PE, et al. *Arch Gen Psychiatry*. 1987;44(8):727–735.
- Chen CN, Wong J, Lee N, et al. *Arch Gen Psychiatry*. 1993;50(2):125–133.
- Cho MJ, Kim JK, Jeon HJ, et al. *J Nerv Ment Dis*. 2007;195(3):203–210.
- Cho MJ, Chang SM, Lee YM, et al. *Asian J Psychiatr*. 2010;3(1):26–30.
- Chong SA, Abidin E, Vaingankar JA, et al. *Ann Acad Med Singapore*. 2012;41(2):49–66.
- Crino R, Slade T, Andrews G. *Am J Psychiatry*. 2005;162(5):876–882.
- Hwu HG, Yeh EK, Chang LY. *Acta Psychiatr Scand*. 1989;79(2):136–147.
- Jacobi F, Wittchen HU, Höltling C, et al. *Psychol Med*. 2004;34(4):597–611.
- Keqing L, Ze C, Lijun C, et al. *Asian J Psychiatry*. 2008;1(2):51–55.
- Lee CK, Kwak YS, Yamamoto J, et al. *J Nerv Ment Dis*. 1990;178(4):242–246.
- Ciarlo JA, Shern DL, Tweed DL, et al. *Eval Program Plann*. 1992;15(2):133–147.
- Kolada JL, Bland RC, Newman SC. *Acta Psychiatr Scand suppl*. 1994;376:24–35.
- Andrade L, Walters EE, Gentil V, et al. *Soc Psychiatry Psychiatr Epidemiol*. 2002;37(7):316–325.
- Bijl RV, Ravelli A, van Zessen G. *Soc Psychiatry Psychiatr Epidemiol*. 1998;33(12):587–595.
- Moher D, Liberati A, Tetzlaff J, et al; PRISMA Group. *Ann Intern Med*. 2009;151(4):264–269, W64.
- Baxter AJ, Scott KM, Vos T, et al. *Psychol Med*. 2013;43(5):897–910.
- Giannakopoulos NN, Rammelsberg P, Eberhard L, et al. *Clin Oral Investig*. 2012;16(3):781–788.
- Knight T, Steeves T, Day L, et al. *Pediatr Neurol*. 2012;47(2):77–90.
- Wittchen HU, Essau CA, von Zerssen D, et al. *Eur Arch Psychiatry Clin Neurosci*. 1992;241(4):247–258.
- Stein MB, Forde DR, Anderson G, et al. *Am J Psychiatry*. 1997;154(8):1120–1126.
- Szádóczky E, Papp Zs, Vitrai J, et al. *J Affect Disord*. 1998;50(2–3):153–162.
- Grabe HJ, Meyer C, Hapke U, et al. *Eur Arch Psychiatry Clin Neurosci*. 2000;250(5):262–268.
- Gureje O, Lasebikan VO, Kola L, et al. *Br J Psychiatry*. 2006;188(5):465–471.
- Karam EG, Mneimneh ZN, Karam AN, et al. *Lancet*. 2006;367(9515):1000–1006.
- Williams L, Jacka F, Pasco J, et al. *Australas Psychiatry*. 2010;18(3):250–255.
- Sachs-Ericsson N, Ciarlo JA. *Sex Roles*. 2000;43:605–628.
- Bürkner PC. *J Stat Softw*. 2017;80(1):1–28.
- Bürkner PC. *R J*. 2018;10(1):395–411.
- R Core Team. *R: A Language and Environment for Statistical Computing*. Vienna, Austria: R Foundation for Statistical Computing; 2018.
- Cooper H, Hedges LV, Valentine JC. *The Handbook of Research Synthesis and Meta-Analysis*. 2nd ed. New York, NY: Russell Sage Foundation; 2009.
- Inthout J, Ioannidis JP, Rovers MM, et al. *BMJ*

It is illegal to post this copyrighted PDF on any website.

- Open. 2016;6(7):e010247.
61. Borenstein M. *Common Mistakes in Meta-Analysis and How to Avoid Them*. Englewood, NJ: Biostat, Incorporated; 2019.
 62. Fawcett JM, Fairbrother N, Fawcett EJ, et al. *Int J Methods Psychiatr Res*. 2018;27(4):e1742.
 63. Fawcett EJ, Fairbrother N, Cox ML, et al. *J Clin Psychiatry*. 2019;80(4):18r12527.
 64. Fawcett JM, Lawrence MA, Taylor TL. *J Exp Psychol Gen*. 2016;145(1):56–81.
 65. Fawcett JM, Ozubko JD. *Can J Exp Psychol*. 2016;70(2):99–115.
 66. Gelman A, Carlin JB, Stern HS, et al. *Bayesian Data Analysis*. Vol. 2. Boca Raton, FL: Chapman; 2014.
 67. Gelman A, Hill J. *Data Analysis Using Regression and Multi-Level/Hierarchical Models*. Cambridge, UK: Cambridge University Press; 2007.
 68. Lewandowski D, Kurowicz D, Joe H. *J Multivariate Anal*. 2009;100(9):1989–2001.
 69. Russell EJ, Fawcett JM, Mazmanian D. *J Clin Psychiatry*. 2013;74(4):377–385.
 70. Steel Z, Marnane C, Iranpour C, et al. *Int J Epidemiol*. 2014;43(2):476–493.
 71. McLean CP, Anderson ER. *Clin Psychol Rev*. 2009;29(6):496–505.
 72. Guglielmi V, Vulink NC, Denys D, et al. *Depress Anxiety*. 2014;31(12):979–987.
 73. Labad J, Menchón JM, Alonso P, et al. *J Clin Psychiatry*. 2005;66(4):428–435, quiz 546.
 74. Forray A, Focseneanu M, Pittman B, et al. *J Clin Psychiatry*. 2010;71(8):1061–1068.
 75. Uguz F, Gezginc K, Zeytinci IE, et al. *Compr Psychiatry*. 2007;48(6):558–561.
 76. Maina G, Albert U, Bogetto F, et al. *Psychiatry Res*. 1999;89(1):49–58.
 77. Vulink NC, Denys D, Bus L, et al. *Int Clin Psychopharmacol*. 2006;21(3):171–175.
 78. Williams KE, Koran LM. *J Clin Psychiatry*. 1997;58(7):330–334, quiz 335–336.
 79. Karpinski M, Mattina GF, Steiner M. *Neuroendocrinology*. 2017;105(1):1–16.
 80. Deecher D, Andree TH, Sloan D, et al. *Psychoneuroendocrinology*. 2008;33(1):3–17.
 81. Soares CN. *Arch Women Ment Health*. 2010;13(1):15–16.
 82. Soares CN, Zitek B. *J Psychiatry Neurosci*. 2008;33(4):331–343.
 83. Pinkerton JV, Guico-Pabia CJ, Taylor HS. *Am J Obstet Gynecol*. 2010;202(3):221–231.
 84. Pope CJ, Oinonen K, Mazmanian D, et al. *Med Hypotheses*. 2017;102:69–77.
 85. Mathis MA, Alvarenga Pd, Funaro G, et al. *Br J Psychiatry*. 2011;33(4):390–399.
 86. Labad J, Menchón JM, Alonso P, et al. *Depress Anxiety*. 2008;25(10):832–838.
 87. Labad J, Alonso P, Segalas C, et al. *Arch Women Ment Health*. 2010;13(1):75–81.
 88. Brakoulis V, Starcevic V, Martin A, et al. *Psychiatry Res*. 2016;239:315–319.
 89. van Grootheest DS, Boomsma DI, Hettema JM, et al. *Am J Med Genet B Neuropsychiatr Genet*. 2008;147B(4):473–478.
 90. Mataix-Cols D, Wooderson S, Lawrence N, et al. *Arch Gen Psychiatry*. 2004;61(6):564–576.
 91. Goldstein JM, Seidman LJ, Horton NJ, et al. *Cereb Cortex*. 2001;11(6):490–497.
 92. Olatunji BO, Sawchuk CN, Arrindell WA, et al. *Pers Individ Dif*. 2005;38(3):713–722.
 93. Fessler DM, Eng SJ, Navarrete CD. *Evol Hum Behav*. 2005;26(4):344–351.
 94. Flaxman SM, Sherman PW. *Q Rev Biol*. 2000;75(2):113–148.
 95. Lienard P. *Neurosci Biobehav Rev*. 2011;35(4):1067–1074.
 96. Eilam D, Izhar R, Mort J. *Neurosci Biobehav Rev*. 2011;35(4):999–1006.
 97. Williams MT, Abramowitz JS, Olatunji BO. *J Behav Ther Exp Psychiatry*. 2012;43(1):632–637.
 98. Thorsen AL, Kvale G, Hansen B, et al. *Curr Treat Options Psychiatry*. 2018;5(1):182–194.
 99. Marques L, LeBlanc NJ, Weingarden HM, et al. *Depress Anxiety*. 2010;27(5):470–475.
 100. Gum AM, King-Kallimannis B, Kohn R. *Am J Geriatr Psychiatry*. 2009;17(9):769–781.
 101. Thomas ML, Kaufmann CN, Palmer BW, et al. *J Clin Psychiatry*. 2016;77(8):e1019–e1025.
 102. Byers AL, Yaffe K, Covinsky KE, et al. *Arch Gen Psychiatry*. 2010;67(5):489–496.
 103. Satre DD, Knight BG, David S. *Prof Psychol Res Pr*. 2006;37(5):489–498.
 104. Brander G, Pérez-Vigil A, Larsson H, et al. *Neurosci Biobehav Rev*. 2016;65:36–62.
 105. Walker ER, McGee RE, Druss BG. *JAMA Psychiatry*. 2015;72(4):334–341.
 106. Jorm AF. *Psychol Med*. 2000;30(1):11–22.
 107. Atladottir HO, Gyllenberg D, Langridge A, et al. *Eur Child Adolesc Psychiatry*. 2015;24(2):173–183.
 108. Martin P. *Dialogues Clin Neurosci*. 2003;5(3):281–298.

Editor's Note: We encourage authors to submit papers for consideration as a part of our Focus on Women's Mental Health section. Please contact Marlene P. Freeman, MD, at mfreeman@psychiatrist.com.

See supplementary material for this article at PSYCHIATRIST.COM.



THE JOURNAL OF CLINICAL PSYCHIATRY

THE OFFICIAL JOURNAL OF THE AMERICAN SOCIETY OF CLINICAL PSYCHOPHARMACOLOGY

Supplementary Material

Article Title: Women Are at Greater Risk of OCD Than Men: A Meta-Analytic Review of OCD Prevalence Worldwide

Author(s): Emily J. Fawcett, PhD; Hilary Power, MA; and Jonathan M. Fawcett, PhD

DOI Number: <https://doi.org/10.4088/JCP.19r13085>

List of Supplementary Material for the article

1. [Appendix 1](#) Model Details and Moderator Analyses

Disclaimer

This Supplementary Material has been provided by the author(s) as an enhancement to the published article. It has been approved by peer review; however, it has undergone neither editing nor formatting by in-house editorial staff. The material is presented in the manner supplied by the author.

Appendix 1

Modelling Details

Models were fit and evaluated for convergence using standard metrics (e.g., $R\text{-hat} < 1.01$)^{66,67} and practices (e.g., visual inspection) described in greater detail elsewhere.⁶²⁻⁶⁵ The only deviation is that we employed a mildly informative prior on the intercept for each model, reflecting the reasonable assumption that the probable average prevalence in a typical sample should range somewhere between 0.6% and 27.0%; within logit-space, this corresponds to a normal distribution with a mean of -3 and a standard deviation of 1. Slopes and standard deviations for random effects were fit with a mildly regularizing prior corresponding to a normal distribution with a mean of 0 and a standard deviation of 1 and correlations amongst random effects were fit with a prior corresponding to an LKJ distribution⁶⁸ with an eta value of 4 (to discourage correlations away from extreme values such as -.9 or .9). Together, these priors reflect the belief that after accounting for variability across samples the “true” prevalence within any given sample might vary anywhere from <0.1% to 73%.

Moderator Analyses

Year. As a continuous predictor, year of publication was centred and standardized prior to fitting the model. Although an increased frequency of OCD diagnosis in psychiatric hospitals has been reported,¹² this conclusion does not appear to be supported in community samples, with the model excluding this moderator being 42.9 times more likely than the model including it. Within our North American samples the older studies^{22,29,30} produced – if anything – numerically higher estimates (2.5%, 3.0%, and 3.2%, respectively) than our more recent samples (2.3%²⁶ and 1.4%¹⁹). In short, there is little evidence in the current data that OCD prevalence has changed over the past 26 years.

Region. Past research has found the prevalence of OCD within pregnant and postpartum women to vary as a function of geographical region.⁶⁹ For that reason, we expected a similar pattern within the general population. Two studies were excluded^{42,52} on the basis that they pertained to regions (Africa and South America) for which there were insufficient estimates to produce reliable results (i.e., one study

each). The remaining four regions (North America, Europe, Asia Pacific and the Middle-East) were dummy coded with North America as the intercept. Despite a trend favouring relatively higher prevalence in North America, $M = 2.1\%$, $CI_{95\%} [1.1\%, 3.8\%]$, than Asia-Pacific, $M = 1.2\%$, $CI_{95\%} [0.8\%, 2.0\%]$, Europe, $M = 1.4\%$, $CI_{95\%} [0.7\%, 2.9\%]$, or the Middle East, $M = 1.4\%$, $CI_{95\%} [0.6\%, 3.4\%]$, these effects were weak and the model excluding this moderator is 4.9 times more likely than the model including it.

Response Rate. No estimates of lifetime prevalence fell within the low category, so our model instead compared average to excellent response rates. Despite prevalence being numerically lower in the excellent category, $M = 1.4\%$, $CI_{95\%} [0.8\% \text{ to } 2.5\%]$, than in the average category, $M = 1.5\%$, $CI_{95\%} [0.8\% \text{ to } 2.5\%]$, we were unable to draw strong conclusions with respect to this difference, $M = 0.6\%$, $CI_{95\%} [-0.3\% \text{ to } 1.9\%]$.

Interviewer. To evaluate the effect of interviewer, we compared the prevalence of OCD as a function of whether the diagnosis had been made by a trained interviewer, clinician, or student/allied mental health practitioner. Prevalence estimates based on clinicians, $M = 1.7\%$, $CI_{95\%} [0.7\%, 3.8\%]$, and trained interviewers, $M = 1.7\%$, $CI_{95\%} [1.0\%, 3.1\%]$, were consistent with one another, $M = 0.0\%$, $CI_{95\%} [-1.6\%, 2.1\%]$. Prevalence estimates based on interviews conducted by students or allied mental health practitioners were lower, $M = 0.9\%$, $CI_{95\%} [0.4\%, 2.0\%]$, but the difference between these estimates and those based on diagnoses made by clinicians was not credible overall, $M = 0.8\%$, $CI_{95\%} [-0.5\%, 2.8\%]$.

Country Economic Status. Because there was only a single estimate from a developing country,⁵² we instead compared only those estimates from developed and emerging countries. Prevalence estimates were similar for developed, $M = 1.3\%$, $CI_{95\%} [0.9\%, 2.0\%]$, and emerging countries, $M = 1.5\%$, $CI_{95\%} [0.8\%, 3.0\%]$, with a difference of only 0.2% , $CI_{95\%} [-0.7\%, 1.7\%]$.

Diagnostic Criteria. With respect to diagnostic criteria, we collapsed DSM-III and DSM-III-R into a single estimate, resulting in a model comparing DSM-III, DSM-IV and ICD-10. There was a trend favouring lower prevalence estimates based on the ICD-10, $M = 0.7\%$, $CI_{95\%} [0.3\%, 1.6\%]$, than on the

DSM-III, $M = 1.5\%$, $CI_{95\%} [0.9\%, 2.4\%]$, or the DSM-IV, $M = 1.2\%$, $CI_{95\%} [0.7\%, 2.1\%]$. However, neither the contrast between the ICD-10 and DSM-III, $M = 0.8\%$, $CI_{95\%} [-0.2\%, 1.8\%]$, nor the contrast between the ICD-10 and DSM-IV, $M = 0.5\%$, $CI_{95\%} [-0.5\%, 1.5\%]$, produced compelling evidence.

Diagnostic Interview. Five estimates were excluded from the model evaluating the influence of diagnostic interview^{21,24,27,38,54} on the basis that there were too few studies using the measures in question (2 SCID, 1 CIS-R, 1 ASI, and 1 SADS). The resulting model therefore compared prevalence estimates derived from the CIDI to those derived from the DIS. Despite a trend favouring higher prevalence estimates based on the DIS, $M = 1.6\%$, $CI_{95\%} [0.9\%, 2.8\%]$, than those based on the CIDI, $M = 0.9\%$, $CI_{95\%} [0.5\%, 1.6\%]$, this difference was not convincing, $M = 0.6\%$, $CI_{95\%} [-0.3\%, 1.9\%]$.