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REVIEW

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Prevalence of personality disorders in adults with binge eating disorder—A systematic review and Bayesian meta-analysis

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Summary

Binge eating disorder (BED) is a complex mental health problem entailing high risk for obesity, overweight, and other psychiatric disorders. However, there is still unclear evidence of the prevalence of personality disorders (PDs) in BED patients. We conducted a systematic review and a Bayesian meta-analysis for studies examining the prevalence of any PD in adult BED patients. Data sources included PubMed, Cochrane library, EBSCO, PsycINFO, and Science Direct. A Bayesian meta-analysis was conducted to estimate effect sizes for the prevalence of any PD in BED patients. Twenty eligible articles were examined with a total of 2945 BED patients. Borderline personality disorder and "Cluster C" PD, particularly obsessive-compulsive and avoidant PD, were the most frequent PD found in BED patients. BED diagnosis was associated with 28% probability of a comorbid diagnosis of any PD (0.279, 95%CrI: [0.22, 0.34]), with high levels of between-study heterogeneity ($\tau = 0.61$, 95% CrI [0.40, 0.90]). Sensitivity analysis suggested effect sizes ranging from 0.27 to 0.28. The high comorbidity of PDs in BED patients draws attention to the potential complexity of BED clinical presentations, including those that might also be comorbid with obesity. Clinical practice should address this complexity to improve care for BED and obesity patients.

KEYWORDS
Bayesian estimation, binge-eating, eating disorders, meta-analysis, personality disorders

1 | INTRODUCTION

Binge eating disorder (BED) has been highlighted as an important risk factor for long-term overweight and obesity.[1–4] Although BED and obesity can be comorbid conditions, they are known to have distinct phenotypes[5,6] and clinical presentations.[1,2,4] BED patients are prone to have obesity because BED is characterized by recurring episodes of consuming large amounts of food in a short period of time, over a period of at least 3 months.[1,2] These patients often experience feelings of lack of control, guilt, embarrassment, and emotional distress. A study conducted with a US representative sample of adults revealed elevated rates of obesity in people with BED for whom an estimated mean body mass index of 34.3 was found.[4] The same study highlighted the association between a lifetime or 12-month diagnosis of BED and an increased risk of obesity and extreme obesity. Previous studies have highlighted an elevated risk to have co-occurring BED in individuals seeking weight loss treatment,[7] and in individuals seeking or undergoing bariatric surgery (at pre-operative stage).[8] In a study conducted with 502 individuals with overweight/obesity nearly one third of the sample met criteria for BED, food addiction, or both.[9]

Finally, in a 10-year prospective study conducted with 1383 individuals, having eating disorders was associated with a threefold increase in lifetime obesity, with BED patients having the highest rate of obesity (88%).[10]

A recent large population-based study conducted with adults in the US (N = 36,309) highlighted the high comorbidity of BED with other mental health problems such as depression, anxiety, personality disorders (PDs), and substance abuse.[4] Major depressive disorder, alcohol use disorder, and borderline personality disorder were the most prevalent mental health problems in BED patients, and about 23% of these patients had history of suicide attempt. The most recent meta-analysis examining the prevalence of comorbid PD in BED patients was published in 2014 and included nine studies, five of them adopting the DSM-III-R criteria to diagnose BED and PD.[11] The estimated prevalence of any PD was 29% (0.29; 95% CI: 0.24–0.33; N = 838), with Cluster C being the most frequent (0.3 [95% CI: 0.21–0.41]), particularly avoidant and obsessive-compulsive PD, followed by Cluster B (0.11 [95% CI: 0.02–0.39]), with borderline being the most frequent PD in Cluster B. However, the evidence of the prevalence of PD in BED patients is still unclear due to the small sample of studies included in the previous meta-analysis, and also considering the substantial amount of literature on BED that has been published since 2014.

According to the most recent version of the DSM,[12] PD can be described as an enduring and inflexible pattern of long duration that results in significant distress or impairment and is not due to substance use or another medical condition. These maladaptive patterns begin by late adolescence or early adulthood, are evident in many contexts and deviate from the accepted patterns in the individual's culture. The diagnostic approach used in the DSM-5-TR still assumes that PD are qualitatively distinct clinical syndromes, with the alternative of a dimensional model of PD discussed in passing. These clinical syndromes define constellations of traits that characterize all different types of PDs (e.g., borderline personality disorder, avoidant personality disorder). The 11th revision of the ICD[13] includes a fundamentally new approach that focuses on common features that apply to all PD, including personality traits and personality pathology, representing a paradigm shift in the PD diagnosis.[14] The ICD-11 conceptualizes PD as entailing different levels of severity for individual's impairments in self and interpersonal functioning, with pathological traits being part of the individual's dysfunction at self-functioning and interpersonal domains. It is, therefore, more focused on assessing the dynamics in terms of general functioning at individual (identity) and interpersonal level. The diagnosis criteria for PD based on either DSM-5 or ICD-11 is very similar and is commonly based on validated clinical interviews (e.g., structured clinical interview (SCID); Eating Disorder Examination (EDE); Operationalized Psychodynamic Diagnosis (OPD-2)) that provide clinical guidance for assessing each corresponding diagnostic criteria.

With BED being an important risk factor for obesity, overweight,[1–4] and other mental health problems,[4,7–9] many obesity patients seen at obesity clinics are likely to belong to the sub-group of multimorbidity patients (obesity + BED + mental health). The potential comorbidity of PD with BED (and indirectly with obesity, via BED) poses a serious health risk for patients as PD is an enduring and complex mental health problem, often entailing episodes of self-harm, suicide ideation, and uncontrolled impulsivity.[12,13] It is, therefore, of clinical interest to estimate the prevalence of PD among BED patients, to inform clinicians working with obesity and BED patients about the actual magnitude of this comorbidity.

This paper presents findings of a Bayesian meta-analysis examining the prevalence of any PD in adults with a diagnosis of BED, assuming as the alternative hypothesis of PD being comorbid to BED in adult patients. Advantages of the Bayesian estimation in comparison with the frequentist approach to meta-analysis include[15–17]: a more precise estimation of the effects and the heterogeneity, when providing credible intervals within which the true estimates (μ and τ) lie in (with 95% probability); a more robust estimation for meta-analysis of small samples; a direct modeling of the uncertainty of the estimates; and the integration of prior knowledge on the parameters to be estimated, the effect (μ), and the between-study heterogeneity (τ). Finally, recent statistical literature has highlighted the suitability of Bayesian models and generalized linear mixed models (the same models used in the current meta-analysis) for meta-analysis of proportions in epidemiological research, as they are regraded to be more accurate and to lead to less bias in comparison with other methods, particularly those using arcsine-based transformations of proportions.[18–20]

2 | METHOD

2.1 | Design

The current systematic review and meta-analysis followed the preferred reporting items for systematic reviews and meta-analyses (PRISMA) guidelines for reporting systematic reviews and meta-analysis.[21] This review is registered with the International Prospective Register of Systematic Reviews (PROSPERO: CRD42022307424).

2.2 | Search strategy

Two researchers (C.G. and H.S.) systematically conducted a search of electronic databases (PubMed, Cochrane library, EBSCO, PsycINFO, and Science Direct) to retrieve all articles published up to September 21, 2023 (first search completed on February 14, 2022, with the most recent updated search made on September 21, 2023). These databases were searched using terms that are often used in the literature to address PD in binge eating disorder patients: "binge eating" [All Fields] OR "binge eating" [All Fields] OR "binge" [All Fields] OR "eating disorder" [All Fields] OR "eating" [All Fields] OR "obesity" [All Fields] OR "overweight" [All Fields] OR "anorexia" [All Fields] OR "bulimia" [All Fields] AND "personality disorder" [All Fields] OR "personality" [All Fields] OR "borderline personality" [All Fields] OR "borderline" [All Fields] OR "obsessive" [All Fields] OR "avoidant" [All Fields] OR "psychopathology" [All Fields] OR "psychiatric" [All Fields] OR "mental health" [All Fields] OR "comorbid" [All Fields] OR "comorbidity" OR "psychopathology" [All Fields]. Manual search of articles was also conducted to identify any articles that could potentially meet the inclusion criteria for our review.

TABLE 1 Studies characteristics.

Reference	Country	Study design	Setting	Sample [MA (SD)]	F (%)	Outcome measures for BED	Diagnostic criteria for BED	Outcome measures for PD	Diagnostic criteria for PD	BED patients with PD (events %)	Most frequent types of PD among BED patients with any PD (%)[a]
Becker, 2015[22]	US	OCS	General population	N = 347 MA (SD): 44.7 (9.2)	74.5	EDE-Q; DSM-IV SCID	DSM-IV	DSM-IV SCID	DSM-IV	146/347 (42%)	Borderline (14%); avoidant (55%); obsessive-compulsive (46%) paranoid (16%)
Boswell, 2020[23]	US	OCS	University-based clinic	N = 343 MA (SD): 45(9.2)	79	DSM-IV SCID	DSM-IV	DIPD-IV DSM-IV SCID	DSM-IV	143/343 (41.7%)	Cluster A: 18% Cluster B: 20% Cluster C: 82%
De Jonge, 2003[24]	UK	OCS	Outpatient mental health services	BED: N = 15; MA (SD) = 36.9 (11.9)	100	EDE	DSM-IV	DSM-IV; PDE	DSM-IV	5/15 (33%)	Avoidant: 60%; obsessive-compulsive: 40%; borderline: 20%
Grilo&M, 2002[25]	US	OCS	Outpatient mental health services	N = 116 MA (SD): 44 (8.9)	77.5	DSM-IV SCID	DSM-IV	DSM-IV SCID	DSM-IV	35/116 (30.2%)	Avoidant (60%); obsessive-compulsive (54%)
Grilo, 2002[26]	US	OCS	Outpatient mental health services	N = 145 MA (SD): 43.7 (9.1)	77.9	DSM-IV SCID	DSM-IV	DSM-IV SCID	DSM-IV	42/145 (28.9%)	Avoidant (62%); obsessive-compulsive (47%); borderline (23%)
Grilo, 2004[27]	US	OCS	Outpatient mental health services	N = 228 MA (SD): 44(9.2)	79	DSM-IV SCID	DSM-IV	DIPD-IV; DSM-IV SCID	DSM-IV	40/228 (17.5%)	Avoidant[b]
Masheb, 2008[28]	US	OCS[c]	General population	N = 75 MA (SD): 46(9.1)	100	EDE-Q; DSM-IV SCID	DSM-IV	DSM-IV SCID	DSM-IV	18/75 (24%)	Any PD from the Cluster C: 21.3%; any PD from the Cluster B: 1.3%; any PD from the Cluster A: 0%
Minnick, 2017[29]	US	OCS	General population	S1 (BED): N = 17; MA (SD) = 28.7 (6.1)	100	EDE SCID-DSM-IV-TR	DSM-IV	SCID/DSM-IV-TR; MCMI-III	DSM-IV	S1: 9/17 (53%)	Depressive: 65%; dependent: 53%; avoidant: 47%; borderline: 29%; obsessive-compulsive: 0%
Noma, 2015[30]	Japan	OCS	Outpatient university hospital	N = 76; MA (SD): 30.6(9.4) F: 100%	100	DSM-IV-SCID; EDI	DSM-IV	DSM-IV SCID	DSM-IV	7/76 (9.2%)	All Borderline
Picot, 2003[31]	US	OCS	General population	N = 50 MA (SD): 41.3(8.8)	92	EDE	DSM-IV	SCID-II-DSM-IV	DSM-IV	15/50 (30%)	Avoidant: 40%; obsessive-compulsive: 33%; depressive: 20%; borderline: 6.6%

(Continues)

TABLE 1 (Continued)

Reference	Country	Study design	Setting	Sample [MA (SD)]	F (%)	Outcome measures for BED	Diagnostic criteria for BED	Outcome measures for PD	Diagnostic criteria for PD	BED patients with PD (events %)	Most frequent types of PD among BED patients with any PD (%)[a]
Sansone 2011[32]	US	OCS	S1:psychiatric impatient service; S2: internal medicine outpatient	S1: N = 126; MA (SD): 34.8 (12.2) S2: N = 419; MA (SD): 49.5 (15.3)	90	PDQ-4; DSM-IV	DSM-IV	PDQ-4; DSM-IV	DSM-IV	S1: 87 (69%); S2: 94 (22%) Total: 181/545 (33.2%)	Borderline[b]
Schmit, 1990[33]	US	OCS	General population	N = 69; MA (SD): 19(1.5) BED: N = 23	100	SCID; DSM-III-R	DSM-III-R	PDE; SCID; DSM-III-R	DSM-III-R	3/23 (13%)	Obsessive-compulsive: 67%; dependent: 33%; avoidant: 0%; borderline: 0%
Shenoy, 2019[34]	India	OCS	University students	N = 500 MA:19.9 (1.9) BED sample: 50	63	BEDS-7	DSM-5	MSI-BPD	DSM-5	17/50 (22.4%)	Borderline[b]
Specker, 1994[35]	US	OCS	General population	N = 100; MA: 39.2 (range 20–55); BED: N = 43	100	BES; EDI; DSM-III-R-SCID	DSM-IV	PDQ-R DSM-III-R SCID	DSM-III-R	14/43 (33%).	Borderline: 30%; histrionic: 47%; avoidant: 26%; obsessive-compulsive: 26%
Stice, 2001[36]	US	OCS	General population	N = 159 MA = 40	100	EDE-Q; YBC-EDS DSM-III-R SCID	DSM-IV	DSM-III-R SCID	DSM-III-R	36/159 (22.6%)	Borderline: 41.7%; avoidant: 39%; self-defeating: 27.8%; obsessive-compulsive: 17%
Telch, 1998[37]	US	OCS	General population	BED: N = 61; MA (SD): 43.5 (8.7)	100	QEWP; EDE-Q; BES	DSM-IV	DSM-III-R-SCID	DSM-III-R	12/61 (20%)	Borderline: 33%; avoidant: 33%; self-defeating: 25%; obsessive-compulsive: 25%
Udo, 2019[4]	US	OCS	General population	N = 36,309; MA (SD): 45.6(17.5) BED sample: N = 318	56	AUDADIS-5; DSM-5	DSM-5	AUDADIS-5; DSM-5	DSM-5	S1: 178/328 (56%)	Borderline: 49%; schizotypal: 28%; antisocial: 16%
Van Riel, 2020[38]	Netherlands	OCS	General population	BED Women: N = 129; MA (SD) = 40(12)	100	SCID-DSM-5; EDE-Q	DSM-5	SCID; DSM 5; TCI	DSM-5	13/129 (10%)	NR
Wilfley, 2000[39]	US	OCS	University-based clinic	N = 162 MA (SD): 45.2 (9.6)	83	EDE	DSM-IV	DSM-III-R SCID	DSM-III-R	37/162 (22.8%)	Borderline (40%); obsessive-compulsive (62%); self-defeating (35%); avoidant (24%)

TABLE 1 (Continued)

Reference	Country	Study design	Setting	Sample [MA (SD)]	F (%)	Outcome measures for BED	Diagnostic criteria for BED	Outcome measures for PD	Diagnostic criteria for PD	BED patients with PD (events %)	Most frequent types of PD among BED patients with any PD (%)[a]
Yanovski, 1993[40]	US	OCS	General population	N = 43 (with BED) MA (SD): 36.8 (7.6)	77	BES; QEWP BEDCI	DSM-IV	DSM-III-R SCID	DSM-III-R	15/43 (35%)	Borderline (40%); avoidant (26%); obsessive-compulsive (20%)

Abbreviations: AUDADIS-5, alcohol use disorder and associated disabilities interview schedule-5; BED, binge eating disorder; BEDCI, binge eating disorder clinical interview based on DSM-IV criteria; BEDS-7, binge eating disorder screener-7; BES, binge eating scale; DIPD-IV: diagnostic interview for DSM-IV personality disorders; DSM, Diagnostic and Statistical Manual of Mental Disorders; EDE, eating disorder examination—semi-structured interview format used to examine the complete range of psychopathology and behavior specific to eating disorders; EDE-Q, eating disorder examination questionnaire; EDI, eating disorder inventory; F, females; MA, study mean age; MCMI-III, 175-item Millon Clinical Multiaxial Inventory; MSI-BPD, MacLean screening instrument for borderline personality disorder; NR, not reported; OCS, observational cross-sectional study; PD, personality disorder; PDE, personality disorder examination—semi-structured clinical interview for the examination of personality disorders; PDQ-4, personality diagnostic questionnaire-4; PDQ-R, personality diagnostic questionnaire; QEWP, questionnaire on eating and weight patterns; SCID, DSM structured clinical interview; SD, standard deviation for age; TCI, temperament and character inventory; YBC-EDS, Yale-Brown-Cornell Eating Disorder Scale.

[a]% of type of PD diagnosis within the sample with any PD diagnosis;

[b]sample composed only of that personality disorder;

[c]secondary data extracted from a randomized controlled trial previously conducted, although the data of interest for our meta-analysis were collected and analyzed cross-sectionally.

2.3 | Inclusion and exclusion criteria

We selected all studies meeting the following inclusion criteria: (1) original research reported in English; (2) studies assessing adult patients aged 18 or older with the primary diagnosis of BEDs (DSM or ICD criteria for BED); (3) studies assessing PD (DSM or ICD criteria for PD) in BED patients; (4) studies quantifying the proportion of BED patients with a formal diagnosis of any PD. All types of articles and study designs were considered for this review. We considered studies assessing BED and PD using validated clinical interviews and/or standardized questionnaires, as along as the diagnostic criteria has been based on DSM or ICD. We excluded articles lacking sufficient detail to determine whether all inclusion criteria were met.

2.4 | Data extraction

We followed Cochrane guidelines for systematic reviews to select studies. Two authors (C.G. and H.S.) independently reviewed titles and abstracts and then the full-text articles to identify eligible studies and to extract data from articles. Results of both researchers were compared, and non-eligible studies and duplicates were excluded. Any disagreements were solved by re-reading articles, double-checking the eligibility criteria, and asking the opinion of a third author (H.U.). Abstracts providing sufficient detail for exclusion were removed, and the remaining full-text articles were retrieved to be fully analyzed. Full-text articles were read to determine inclusion, and disagreements were resolved via consensus. The primary outcome measure for this meta-analysis included the proportion of patients with a primary diagnosis of BED who met the DSM or ICD criteria for any PD. Measures of BED and PD were considered for data extraction if they have been based on validated standardized diagnostic questionnaires (e.g., Eating Disorder Examination Questionnaire [EDE-Q]; Personality Diagnostic Questionnaire-4 [PDQ-4]); and/or structured and semi-structured clinical interviews (e.g., the DSM-5 Structured Clinical Interview [SCID]). The following data were extracted: country in which the study was conducted; year of publication; study design; study setting; study sample size, with the corresponding mean age and standard deviation; proportion of female BED patients; outcome measures used for BED; outcome measures used for PD; proportion of BED patients with the diagnosis of any PD; and the most frequent types of PD among BED patients who had the comorbid diagnosis of any PD (see Table 1).

2.5 | Study quality and risk of bias assessment

Methodological quality and risk of bias were independently assessed by three researchers (H.U., C.G., and H.S.) using the Quality Assessment Tool for Observational Cohort and Cross-Sectional Studies from the National Heart, Lung and Blood Institute (NHLBI).[41] Any disagreements were solved by re-reading articles and checking them against the NHLBI assessment criteria (done by H.U., C.G., and H.S.). Areas of

methodological quality assessment include: the research question addressed; study population and case comparability; groups recruited from the same population and uniform eligibility criteria; sample size justification; exposure assessed prior to outcome measurement; sufficient timeframe to see an effect; different levels of the exposure of interest; exposure measures and assessment; repeated exposure assessment; blinding of outcome assessors; follow-up rate; rigor of data analysis; ethical issues; and clear statement of findings. The risk of bias dimension was adjusted to the nature of studies included in our meta-analysis, considering that the great majority of them were observational cross-sectional studies. We have, therefore, regarded "blinding of outcome assessors" as whether the outcomes assessors had been blind to the study aims (e.g., to investigate the prevalence of PD in a sample of BED patients). The tool allowed us to appraise whether the methodological quality/risk of bias for each source of bias, is low, high, or with some concerns.

2.6 | Statistical analysis

The Bayesian meta-analysis was computed with a generalized multivariate (non-linear) multilevel model, to estimate effect sizes (μ) for the prevalence of any PD in adults (age >18) with a diagnosis of BED, and the between-study deviation estimate (heterogeneity; Equation 1). Logarithmic odds and pooled proportions (on a probability scale) were computed as the estimates of the true effect sizes, based on data extracted from studies reporting the proportion of BED patients with a diagnosis of any PD. Bayesian inference entails the specification of prior beliefs on the parameters to be estimated. In Bayesian estimation, prior distributions for model parameters are commonly set and categorized as being non-informative, weakly-informative, or informative.[42] Recent literature has highlighted the potential overestimation of the magnitude of effects leading to overstated evidence on its sign resulting from the use of non-informative flat priors,[43–45] which contradicts the "non-informative" assumption inherent to these priors. More informative priors have, therefore, been recommended as a more appropriate and potentially less biased approach for Bayesian estimation.[46] In the current meta-analysis, weakly-informative priors were chosen, considering the paucity of prior knowledge on the topic regarding the actual comorbidity of BED and PD.[15,47] A normal distribution with a mean = 0 and a SD = 1.81, $\mu \sim N(0,1.81)$, was adopted as prior for the for the effect (μ), as suggested by Rover et al.[47] A half-Cauchy distribution was chosen for the between-study deviation estimate ($\tau \sim$ half-Cauchy (0,0.5)), which has been considered an appropriate choice to model between-study variance in meta-analysis.[15] Different prior distributions for the effect (μ) were run as part of the sensitivity analysis, adopting the strategy suggested by Korner-Nievergelt et al.,[48] when using weakly-informative priors, which includes re-running the primary analysis with progressively narrower prior distributions. The principle is that the best informative prior will result from the narrowest prior distribution that does not affect the results[49] (see Section 2.7 for more details).

2.6.1 | Equation 1—Bayesian generalized multilevel model

Model parameters: $xT_i \sim$ Bin $(nT_i, \pi T_i)$; logit $(\pi T_i) = \mu_i$ μ_i, θ, and $\tau \sim$ priors.

Priors: μ, $\tau \sim$ Normal (μ, τ^2).

The Bayesian multilevel hierarchical model was implemented to run a meta-analysis containing *k* studies with proportions (number of events/sample size). Study *i* has xT_i events, which are assumed to follow a binomial distribution with sample size nT_i within each study. πT_i are underlying true event rates in I, with μ_i representing the underlying true log proportion within studies, θ the overall effect size (resulting from all log proportions), and τ the between-study heterogeneity. For both parameters (μ, τ), we assume prior distributions to be normally distributed. The posterior distribution is, therefore, expressed as p (μ, τ|θ).

$$\textbf{Model Equation}: \gamma = X\tau_k + Z\mu_k + \sum_{k=1}^{k} s_k(x_k), \quad (1)$$

where τ and u are the respective coefficients at the group-level effects (between-study heterogeneity), and population-level effects (effect size), that is, they are the model main parameters, for *k* studies. X, Z are the corresponding design matrices. The terms s_k (x_k) represent smooth functions of unspecified form based on covariates x_k fitted via splines. The coefficients τ and u also represent the fixed and random effects, with the random component being allocated to study (*k*). In the *brms* R package, this equation is computationally expressed as: *brm* $(x_i|trials\ (n_i) \sim 0 + Intercept + (1|Study))$.

Between-study heterogeneity was measured by an estimate for group-level effects (τ), including the corresponding standard error and 95% credible interval. Values of τ ranging from 0.1 to 0.5 are commonly interpreted as indicating reasonable heterogeneity, whereas values above 0.5 and 1.0 suggest fairly high and fairly extreme heterogeneities, respectively.[47]

Statistical analysis was conducted using RStudio software, with the *brms* package used for the Bayesian meta-analysis, Bayesian meta-regression, and for sensitivity analysis with Bayesian estimation (the full R script included in the supplementary materials). The *brms* R package allows us to fit a wide range of Bayesian multilevel and hierarchical regression models, using the Stan probabilistic programming language, which implements the No-U-Turn Sampler (NUTS) extension of the Hamiltonian Monte Carlo algorithm.[50,51] All Bayesian model analyses included in the current work were set with 4000 iterations. The frequentist meta-analysis (as part of the sensitivity analysis) was performed using *metaphor* and *meta* R packages.

2.7 | Meta-regression and sensitivity analysis

A Bayesian meta-regression was undertaken to investigate the potential factors underpinning variance and heterogeneity in our main meta-analysis. A Bayesian meta-regression random-effects model was

conducted to evaluate joint and marginal posterior probability distributions for the covariates included in our model (β) and for the heterogeneity (τ). Priors used for the meta-regression included $\mu \sim N(0,1.5)$ and $\tau \sim$ half-Cauchy(0,0.5) to reflect results from the sensitivity analysis after adopting the narrowing strategy suggested by Korner-Nievergelt et al.[48] Mean age, percentage of female patients in the sample, study setting (clinical vs. non-clinical), and the diagnostic criteria for PD (DSM-III vs. DSM-IV/DSM-5) were included as covariates for our meta-regression model. 95% credible intervals were used for both estimates (μ, τ).

2.7.1 | Bayesian meta-regression multilevel model

$$y_i \mid x_{i,}\, \mu, \tau \sim \text{Normal}\left(\mu_1 x_{i,1} + \ldots + \mu_d x_{i,d}, + \tau^2\right)$$

The data (y) as $y_{i,}$ with the $_i$-th estimate, with i = 1, … k (studies), and d + 1 unknown parameters (coefficients μ), including the four covariates ($x_{i,1\ldots4}$), which are the fixed effects component, and the heterogeneity (τ). In the *brms* R package, the meta-regression model is computationally expressed as *brm (*x_i*|trials (*n_i*) ~ femf + meanage + setf + criteriaf + (1 |Study)*; the equation is identical to the meta-analysis main model, but here, the main parameter (effect size − μ) is associated with the a set with fixed effects (covariates) plus the random component *(1|Study)*.

2.7.2 | Sensitivity analysis

1. Five additional Bayesian meta-analyses using different weakly-informative priors for μ (effect size) and for τ (heterogeneity), following the narrowing strategy suggested by Korner-Nievergelt et al.[48] Additionally, we also tested different scenarios for priors showing greater variance for the parameter τ (between-study heterogeneity), to investigate the potential effect of having greater levels of variance across studies. Models with alternative weakly-informative priors were, therefore, computed as (a) $\mu \sim N(0,1.5)$, $\tau \sim$ half-Cauchy(0,0.5); (b) $\mu \sim N(0,1)$, $\tau \sim$ half-Cauchy(0,0.5); (c) $\mu \sim N(0,1.81)$, $\tau \sim$ half-Cauchy(0,1); (d) $\mu \sim N(0,1.5)$ and $\tau \sim$ half-Cauchy(0,1); (e) $\mu \sim N(0,1)$ and $\tau \sim$ half-Cauchy(0,1);
2. Bayesian meta-analysis using an uninformative flat prior for μ (a prior with the precision = 0 and variance = ∞), and a very weakly-informative prior for τ ($\tau \sim$ half-Cauchy(0,0.5)), to investigate a more non-informative/improper scenario for priors;
3. Additional meta-analysis excluding a study identified as an outlier for reporting a higher prevalence of PD based on a small sample[29];
4. Finally, a frequentist meta-analysis of the same 20 studies included in our main meta-analysis was also computed, using a random intercept logistic regression model. The analysis used the Clopper–Pearson confidence interval for individual studies, and the maximum-likelihood estimator for the between-study heterogeneity parameter tau-squared (τ^2). The logit-transformed proportions were computed as summary measures of the effect size.

2.8 | Publication bias

Publication bias was inspected by running a random-effects funnel plot to check plot asymmetry on the effect estimates (y_i) versus their standard errors (σ_i), using the *funnel.bayesmeta* function of the *bayesmeta* R package.

3 | RESULTS

3.1 | Study selection and characteristics

The study selection process is described in the PRISMA flowchart (Figure 1). We identified 812 articles from the databases and other sources, of which 725 were excluded on the basis of title and abstract review, including exclusion of duplicate articles, leaving 87 articles for full-text revision. After checking the full-texts, we excluded 67 for not meeting eligibility criteria, with a final list of 20 articles to be reviewed.[4,22–40] The main reasons for not meeting the eligibility criteria were: the main studied sample was not composed of adults with a valid diagnosis of BED; data on personality traits but not on PD; and no available data to calculate the proportion of BED patients with PD.

Table 1 summarizes the main characteristics of the 20 studies included in this meta-analysis, which assessed a total of 2945 BED patients. All studies assessed the prevalence of PD in patients with a primary diagnosis of BED. The majority of studies were observational cross-sectional, examining BED patients at an outpatient clinic. The great majority of studies assessed patients aged from 35 to 45 years old, were conducted in the United States (N = 16), had samples mostly composed of female patients, and used the Diagnostic and Statistical Manual of Mental Disorders (DSM) Structural Clinical Interview to diagnose BED and PD. Only three studies[32,34,37] have used questionnaires to diagnose BED, two of which[32,34] also used questionnaires to diagnose PD. In terms of the DSM version adopted to diagnosed BED, three studies[4,34,38] adopted the DSM-5 criteria, sixteen studies[22–32,35–37,39,40] adopted the DSM-IV criteria, and one study adopted the DSM-III-R criteria.[33] Regarding the DSM version adopted to diagnose PD, three studies adopted the DSM-5,[4,34,38] eleven studies adopted the DSM-IV criteria,[22–32] and six studies adopted the DSM-III-R criteria.[33,35–37,39,40] No study used the International Classification of Diseases (ICD) criteria for psychiatric diagnosis. The raw prevalence of any PD in BED patients across all studies ranged from 9.2% to 53%. In general, the most frequent comorbid PD found across studies were borderline personality disorder (mean prevalence [mp] = 0.247; sd = 0.149) and the Cluster C PD, particularly obsessive-compulsive (mp = 0.354; sd = 0.196) and avoidant PD (mp = 0.369; sd = 0.188).

3.2 | Risk of bias within studies

The risk of bias assessments are presented in Table 2. No study was rated as having high risk of bias. However, all studies were rated as

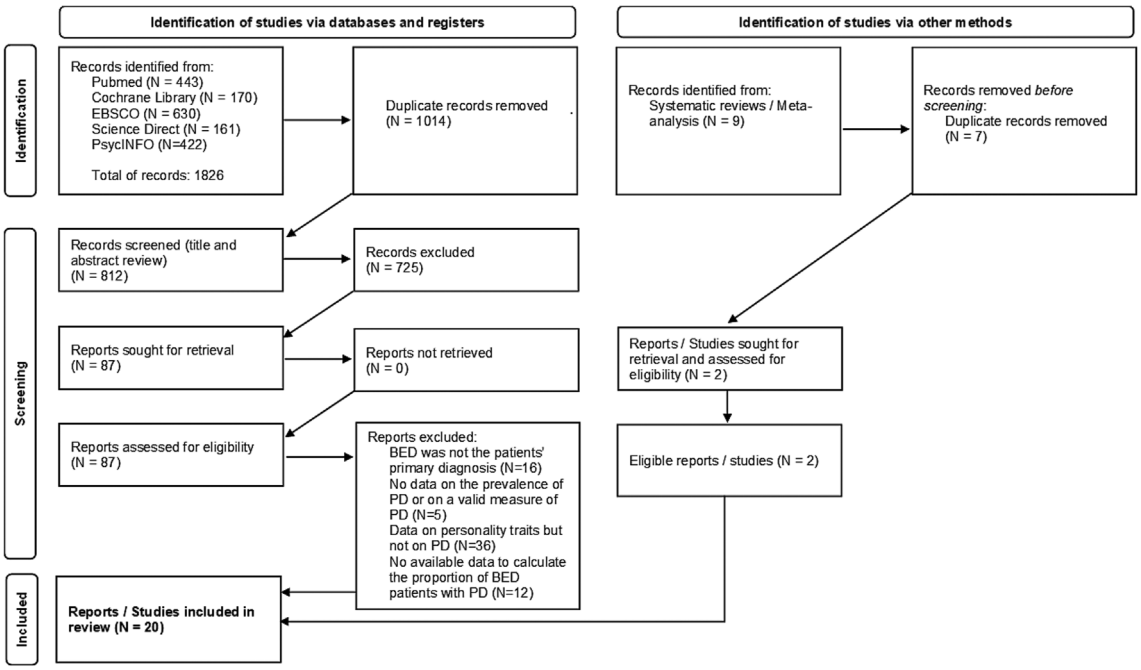

FIGURE 1 Preferred reporting items for systematic reviews and meta-analyses (PRISMA) flow diagram.

entailing some concerns for study quality/risk of bias. Main sources of bias across studies include: lack of rationale or justification for sample size, including information on statistical power calculation; lack of information on whether the outcome assessors were blinded to study aims; and key potential confounding variables not been measured and/or adjusted for in the statistical model(s), which was detected in one study.[26] Most studies did not score positively in Dimensions 6, 7, 8, 10, and 13 due to their study design being cross-sectional.

3.3 | Synthesis of results

Summary of estimates for heterogeneity (τ) and the true effect (μ) for the main meta-analysis performed using weakly-informative priors to estimate the effect size ($\mu \sim N(0,1.81)$), and the between-study heterogeneity ($\tau \sim$ half-Cauchy(0,0.5)) are presented in Table 3. Sensitivity analysis results are also presented in Table 3.

The 95% highest posterior density intervals, and short credible intervals (CrI), for both parameters (μ, τ), did not cross zero, which suggests: the weighted effect size from these data differs from zero and the alternative hypothesis (of an effect, i.e., PD is comorbid to BED) can be accepted; and there is between-study heterogeneity. The 95% CrI for the parameter of effect (μ) do not contain values close to zero, which also suggests that the magnitude of the effect might not be small. The forest plot for the main meta-analysis (Figure 2) illustrates the estimates of the true effect sizes with the corresponding CrI, and the overall effect size (μ) in a probability scale, which might be interpreted as a pooled proportion of PD for BED patients. BED is estimated to be associated with 27.9% probability of a comorbid diagnosis of any PD (0.279, 95% CrI: [0.22, 0.34]). The range of the credible interval in which the true effect is expected to lie in (μ), together with the values for the τ parameter ($\tau = 0.61$, 95% CrI [0.40, 0.90]), suggest moderate effect size variability and high levels of between-study heterogeneity. The joint posterior predictive density plot (for μ and τ parameters) is graphically presented in Figure 3 and suggests higher density in the area closer to the value of 0.3 for the effect size and the value of 0.6 for the heterogeneity.

The model diagnostics are illustrated in Figure S1. The graphical representation for the posterior predictive density of μ and τ parameters suggests normality of posterior distribution of μ and a slight positively skewed distribution for τ (Figure S1). The assessment of model convergence, with Rhat $= 1$ (Table 3), indicates that the algorithm found the optimal solution and the model has converged, which suggests that the model parameters are trustworthy, as illustrated in the Trace Plot (Figure S1) and in the PPcheck plot (Figure S2).

3.4 | Sensitivity analysis

Sensitivity analysis investigated different scenarios for our meta-analysis of the prevalence of PD in BED patients. The results obtained by using different weakly-informative and non-informative priors for μ (effect size) and for τ (heterogeneity) suggest consistency and reliability across all models because the results are quite similar across all meta-analyses, ranging from 0.61 to 0.63 for τ, and being always around 0.28 for μ (Table 3). Regarding the 95% HPDI intervals, they were very similar across models, which suggests model consistency. The HPDI intervals did not cross zero for both parameters (μ, τ), which suggests the existence of an effect associated with the comorbidity of PD in BED patients, and also suggests the presence of between-study

TABLE 2 Risk of bias and study quality assessment (National Heart, Lung and Blood Institute [NHLBI] checklist).

	D1	D2	D3	D4	D5	D6	D7	D8	D9	D10	D11	D12	D13	D14	Overall
Becker,2015	+	+	+	+	×	×	×	N/A	+	×	+	?	N/A	+	−
Boswell, 2020	+	+	+	+	×	×	×	N/A	+	×	+	?	N/A	+	−
De Jonge, 2003	+	+	+	+	×	×	×	N/A	+	×	+	?	N/A	+	−
Grilo, 2002	+	+	+	+	×	×	×	N/A	+	×	+	+	N/A	×	−
Grilo, 2004	+	+	+	+	×	×	×	N/A	+	×	+	?	N/A	+	−
Grilo& M, 2002	+	+	+	+	×	×	×	N/A	+	×	+	+	N/A	+	−
Masheb, 2008	+	+	+	+	×	+	+	N/A	+	+	+	?	N/A	+	−
Minnick, 2017	+	+	+	+	+	×	×	N/A	+	×	+	?	N/A	+	−
Noma, 2015	+	+	+	+	×	×	×	N/A	+	×	+	?	N/A	+	−
Picot, 2002	+	+	+	+	+	×	×	N/A	+	×	+	?	N/A	+	−
Sansone 2011	+	+	+	+	×	×	×	N/A	+	×	+	?	N/A	+	−
Schmit, 1990	+	+	+	+	×	×	×	N/A	+	×	+	?	N/A	+	−
Shenoy, 2019	+	+	+	+	+	×	×	N/A	+	×	+	?	N/A	+	−
Specker, 1994	+	+	+	+	×	×	×	N/A	+	×	+	+	N/A	+	−
Stice, 2001	+	+	+	?	×	×	×	N/A	+	×	+	?	N/A	+	−
Telch, 1998	+	+	+	+	×	×	×	N/A	+	×	+	?	N/A	+	−
Udo, 2018	+	+	+	+	+	×	×	N/A	+	×	+	?	N/A	+	−
Van Riel, 2020	+	+	+	+	×	×	×	N/A	+	×	+	?	N/A	+	−
Wilfley, 2000	+	+	+	+	×	×	×	N/A	+	×	+	?	N/A	+	−
Yanovski, 1993	+	+	+	+	×	×	×	N/A	+	×	+	?	N/A	+	−

D1. Was the research question or objective in this paper clearly stated?
D2. Was the study population clearly specified and defined?
D3. Was the participation rate of eligible persons at least 50%?
D4. Were all the subjects selected or recruited from the same or similar populations (including the same time period)? Were inclusion and exclusion criteria for being in the study prespecified and applied uniformly to all participants?
D5. Was a sample size justification, power description, or variance and effect estimates provided?
D6. For the analyses in this paper, were the exposure(s) of interest measured prior to the outcome(s) being measured?
D7. Was the timeframe sufficient so that one could reasonably expect to see an association between exposure and outcome if it existed?
D8. For exposures that can vary in amount or level, did the study examine different levels of the exposure as related to the outcome (e.g., categories of exposure, or exposure measured as continuous variable)?
D9. Were the exposure measures (independent variables) clearly defined, valid, reliable, and implemented consistently across all study participants.
D10. Was the exposure(s) assessed more than once over time?
D11. Were the outcome measures (dependent variables) clearly defined, valid, reliable, and implemented consistently across all study participants?
D12. Were the outcome assessors blinded to the exposure status of participants?
D13. Was loss to follow-up after baseline 20% or less?
D14. Were key potential confounding variables measured and adjusted statistically for their impact on the relationship between exposure(s) and outcome(s)?

Quality Assessment Score Criteria: Poor / High Risk of Bias: 0-4 out of the 14 dimensions rated as "Good"; Fair Quality / some Risk of Bias Concerns: 5-13 out of the 14 dimensions rated as "Good"; Good / Low Risk of Bias: 14 out of 14 dimensions rated as "Good". NA: Not Applicable.

Good/Low Risk of Bias +

Fair / some concerns for risk of bias −

Poor / high risk of bias ×

Not reported ?

Not applicable N/A

TABLE 3 Posterior summary of estimates for the main meta-analysis and for all sensitivity analyses.

		$\tau \sim$ half-Cauchy(0,0.5)					$\tau \sim$ half-Cauchy(0,1)				
	Parameter	Estimate	SE	95% CrI[c]	95% HPDI[d]	Rhat[b]	Estimate	SE	95% CrI[c]	95% HPDI[d]	Rhat[b]
$\mu \sim$ N(0,1.81)	τ	0.61	0.13	[0.40, 0.90]	[0.40, 0.87]	1	0.63	0.13	[0.42, 0.92]	[0.41, 0.89]	1
	μ	0.28	0.54	[0.22, 0.34]	[0.22, 0.34]	1	0.28	0.54	[0.22, 0.34]	[0.22, 0.34]	1
$\mu \sim$ N(0,1.5)	τ	0.61	0.13	[0.41, 0.90]	[0.39, 0.87]	1	0.62	0.13	[0.42, 0.92]	[0.40, 0.88]	1
	μ	0.28	0.54	[0.22, 0.35]	[0.22, 0.34]	1	0.28	0.54	[0.22, 0.34]	[0.22, 0.34]	1
$\mu \sim$ N(0,1)	τ	0.61	0.12	[0.41, 0.89]	[0.39, 0.85]	1	0.63	0.13	[0.43, 0.92]	[0.40, 0.88]	1
	μ	0.28	0.54	[0.23, 0.34]	[0.23, 0.34]	1	0.28	0.54	[0.23, 0.35]	[0.23, 0.35]	1
$\mu \sim$ inf(0,∞)	τ	0.61	0.12	[0.41, 0.89]	[0.39, 0.85]	1	0.63	0.13	[0.42, 0.91]	[0.41, 0.88]	1
	μ	0.28	0.54	[0.22, 0.34]	[0.22, 0.34]	1.01	0.28	0.54	[0.22, 0.34]	[0.22, 0.35]	1
$\mu \sim$ N(0,1.81)[a]	τ	0.60	0.12	[0.41, 0.88]	[0.39, 0.84]	1	0.62	0.13	[0.42, 0.91]	[0.39, 0.87]	1
	μ	0.27	0.54	[0.21, 0.34]	[0.21, 0.33]	1	0.27	0.54	[0.21, 0.34]	[0.21, 0.34]	1

Abbreviations: SE, standard error; μ, estimate of effect; τ, estimate of heterogeneity.
[a]Main meta-analysis without the outlier study[29]; μ values in probability scale;
[b]Rhat refers to model convergence;
[c]credible intervals;
[d]highest posterior density intervals.

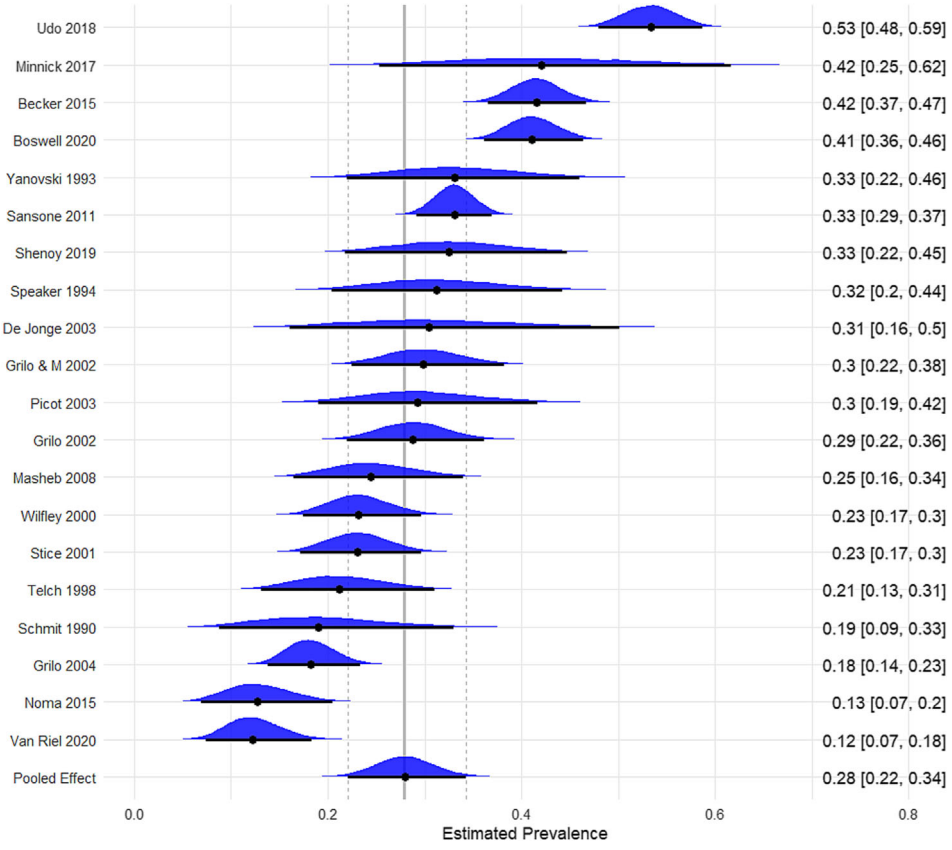

FIGURE 2 Forest plot for the main Bayesian meta-analysis.

heterogeneity. In the same way, the 95% CrI for the parameter of effect (μ) do not contain values close to zero, which reveals that the magnitude of the effect is not small. Furthermore, the Rhat value is 1 for all models presented, which indicates that the algorithm found the optimal solution and the model has converged, as mentioned before. After removing the study identified as an outlier,[29] the results are also very similar to those obtained in the main meta-analysis and other analyses computed as part of the sensitivity analysis (Table 3).

Meta-analysis of the prevalence of PD in BED patients using a frequentist approach reported an effect size of 0.28 (95% CI: 0.23, 0.34), suggesting a similar effect size to the one found in the main Bayesian meta-analysis (see the frequentist meta-analysis forest

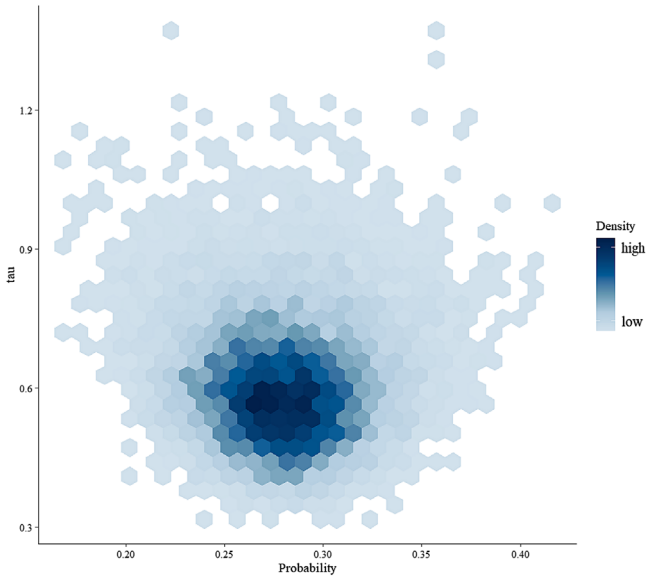

FIGURE 3 Joint posterior predictive density plot.

TABLE 4 Comparison between Bayesian meta-analysis and frequentist approaches.

	Bayesian	Frequentist
τ	0.61 (0.40, 0.90)	0.57[a] (I^2 = 89.5% [85.2%; 92.5%])
μ	0.28 (0.22, 0.34)	0.28 (0.23, 0.34)

Abbreviations: μ, estimate of effect; τ, estimate of heterogeneity.
[a]No confidence intervals for the heterogeneity parameter are provided by the *meta* R package when using the GLMM method. However, using the inverse variance method, we obtained tau = 0.56 [0.40; 0.91].

plot in Figure S3). High levels of between-study heterogeneity were also detected in the frequentist meta-analysis ($\tau^2 = 0.329$; I2 = 89.5% [85.2%; 92.5%]; H = 3.09 [2.60; 3.66]). Table 4 summarizes the results of the main Bayesian meta-analysis and the frequentist meta-analysis.

3.5 | Bayesian meta-regression

Three Bayesian meta-regression models were computed to investigate potential factors underpinning variance and heterogeneity in our main meta-analysis (Table 5). One full-model with all covariates, plus two additional models with the variables study setting and diagnostic criteria being stepwise removed, as they showed the largest range of credible intervals. As shown in Table S5, the 95% CrI for all covariates did cross zero, which suggests that the alternative hypothesis of an effect cannot be accepted, that is, the covariates might not have an effect as factors of variance and heterogeneity in our meta-analysis at 95% probability. In terms of model diagnostics for our meta-regression, model convergence was confirmed, with all Rhat values equal to 1. Table 6 presents the model comparison, using the WAIC criteria, which suggests that meta-regression models with less covariates do not bring better model fitness, as the WAIC values are very similar.

3.6 | Risk of bias across studies (publication bias)

No asymmetry was detected in the funnel plot reporting the effect estimates (yi) versus the standard errors (σi) of our main Bayesian meta-analysis (see Funnel Plot in Data S1).

4 | DISCUSSION

To the best of our knowledge, this is the first Bayesian meta-analysis estimating the prevalence of PD in people with a diagnosis of BED, and the first meta-analysis on the topic in which studies adopting the most recent diagnostic criteria for BED and PD (DSM-5) were included. Our meta-analysis suggests that PD are highly comorbid in BED patients, particularly for obsessive-compulsive, avoidant, and borderline personality disorders. The current meta-analysis estimates a pooled prevalence of any PD in BED patients of about 28%, which is similar to the prevalence found in a meta-analysis published in 2014 on the same topic (29%),[11] and considerably higher than the prevalence of PD found in general population (7.8%; 95% CI: 6.1%–9.5%).[52] However, the findings from the current meta-analysis come with a higher level of evidence as they result from a larger pool of studies in comparison with the meta-analysis conducted in 2014 (20 studies (N = 2945) vs. 9 studies (N = 838)), with our sensitivity analysis suggesting similar pooled prevalence rates, which strengthens the level of evidence on the co-occurrence of PD in BED patients. Additionally, the current meta-analysis includes an updated examination of BED and PD's according to studies adopting more recent diagnostic systems, particularly DSM-IV and DSM-5, whereas in the 2014 meta-analysis, most articles examined had adopted older versions of the DSM. Furthermore, the adoption of Bayesian and generalized linear mixed models (frequentist) to compute effect sizes in our meta-analysis allow us to draw more reliable conclusions on the prevalence of PD's in BED patients, in comparison with other statistical methods.[18–20]

Knowing that BED is a risk factor for obesity and overweight,[4,7–9] the comorbidity BED-PD is of particular concern as it has potential to deteriorate patients' long term physical health and to compromise treatment outcomes.[1–4,53] Obesity itself can be challenging to treat, as it involves a multidisciplinary approach to promote a considerable change in patients' lifestyle and behavior. The possible co-occurrence of BED with obesity, with BED potentially associated with PD, draws attention to a complex clinical presentation that deserves attention by clinicians working in obesity settings, as these patients might only present, at the first glance, obesity as the main symptom or complaint.

In obesity patients, the prevalence of PD's is still unclear, but large-scale studies have highlighted the elevated prevalence of psychiatric disorders in obesity patients, including BED, PD, and mood

TABLE 5 Summary of estimates for the Bayesian meta-regression.

Variable	Parameter	Estimate[a]	SE	Lower 95% CrI	Upper 95% CrI	Rhat[b]
	τ Group-level effects	0.643	0.147	0.413	0.989	1
Mean age	μ Population-level effects	0.021	0.022	−0.022	0.065	1
Percentage of female patients	μ Population-level effects	0.003	0.012	−0.022	0.028	1
Study setting (clinical vs. non-clinical)	μ Population-level effects	−0.330	0.351	−1.023	0.372	1
Diagnostic criteria for PDs (DSM-III versus DSM-IV/DSM-5)	μ Population-level effects	0.363	0.362	−0.358	1.070	1

Abbreviations: DSM, Diagnostic and Statistical Manual of Mental Disorders; PDs, personality disorders; SE, standard error; μ, estimate of effect; τ, estimate of heterogeneity.
[a]μ Population-level effects: values in log-odds scale;
[b]Rhat parameter for model convergence.

TABLE 6 Meta-regression model comparison.

Variable	Model 1	Model 2	Model 3
Mean age	X	X	X
Percentage of female patients	X	X	X
Diagnostic criteria for PDs (DSM-III vs DSM-IV/DSM-5)	X	X	
Study setting (clinical vs. non-clinical)	X		
WAIC	127.9	128	128

Abbreviations: DSM, Diagnostic and Statistical Manual of Mental Disorders; WAIC, Watanabe-Akaike information criteria.

disorders.[53–56] A systematic review examining the association between obesity and PD suggested a higher risk for obesity in individuals with PD, with clinical studies highlighting a particular comorbidity between PD, such as borderline and avoidant, and BED.[57] A 17-year large cohort study conducted in Austria found patients with obesity to be 1.5 times more likely to have PD than patients without obesity (OR: 1.56, 95% CI: 1.49–1.64).[56]

The reasons for the potential high comorbidity of BED and PD have not yet been well-understood due to the lack of robust cohort studies addressing the topic, as seen in our meta-analysis. The same lack of evidence is observed for the relationship between obesity and mental health problems, including BED and PD.[57] Intriguingly, BED and PD share several common clinical and psychopathological features,[1,2,58–60] such as an impairment in emotional regulation, impulse control, self-esteem, and social and interpersonal functioning, together with pathological personality traits (obsessive, neuroticism, impulsivity, avoidance), and compulsive behaviors. These common clinical features raise the hypothesis of both conditions sharing common etiological factors. The neurobiological literature has also found specific alterations in the dopaminergic system that are independently associated with impulsivity–compulsivity and reward-related processes, both in BED and in patients with borderline personality disorder.[61,62] Future studies will clarify the etiology of the association between BED and PDs, which might also contribute to the understanding of the relationship between obesity and psychiatric disorders such as BED and PD.

High levels of between-study heterogeneity were found in our meta-analysis. The meta-regression analysis, however, did not find any potential factors of between-study heterogeneity. In the meta-regression model, the 95% CrI for the percentage of female patients slightly crossed zero, which might suggest a tendency (although not statistically significant) for studies with a greater proportion of females (>80%) to show a lower prevalence of PD. This should be clarified in future large-scale studies (including meta-analyses). Unexpectedly, the diagnostic criteria (DSM version) were not found to have any statistical relevance in relation to the pooled effect size (prevalence of PD in BED patients), which was also confirmed by the sensitivity analysis we conducted. The meta-regression, therefore, suggests that the high between-study heterogeneity detected in our meta-analysis might be explained by other random factors that were not possible to be accounted for in our regression model.

The main strengths of the current meta-analysis include sounder evidence of the prevalence of PD in BED patients in comparison with previous findings,[11] in spite of changes in the diagnostic system (DSM). The lack of epidemiologically sound cohort studies is one of the main limitations of the current meta-analysis, as the majority of studies included were observational cross-sectional, which limits the levels of evidence suggested by our analysis. The reliability of main results might have also been affected by other factors entailing between-study heterogeneity, such as sample sizes, percentage of females, different diagnostic criteria adopted for BED and PD (since DSM-III), and study setting, although the meta-regression analysis conducted did not find these factors relevant to explain variability in the effect sizes. Finally, future studies analyzing more dynamic constructs of pathological personality in BED patients, instead of using the categorical diagnostic system, and in line with the recent developments in the DSM-5-TR and ICD-11, should provide a more detailed information on the pathological mechanisms underlying the comorbidity of BED and PD, which is paramount to improve treatment for BED.

In conclusion, our meta-analysis draws attention to the potential complexity of BED cases entailing other enduring comorbid mental health problems such as PD, and therefore contribute to making BED an even more challenging condition to treat. With obesity being a common comorbid problem to BED, the co-occurrence of PD in BED patients has potential to make these patients more difficult to treat and, therefore, with poorer treatment outcomes for mental health and weight control, which should be taken into account in clinical routine for obesity patients. Future large-scale longitudinal studies will bring more evidence to the topic and shed light on the factors underpinning this comorbidity, which is of paramount importance for clinicians and researchers. Finally, with this meta-analysis, we hope the Bayesian estimation to have lent some of its beauty and elegance to the current uncertainty (knowledge) in fields of mental health.

AUTHOR CONTRIBUTIONS

Hugo Senra played a lead role in the conceptualization, data curation, methodology, and data analysis of the current meta-analysis. Hugo Senra designed the study with input from Catarina Gouveia Gaglianone, Susan McPherson, and Human Unterrainer. Catarina Gouveia Gaglianone and Hugo Senra set up the database, with help from Human Unterrainer and Susan McPherson. Hugo Senra and Catarina Gouveia Gaglianone screened the literature search with help from Human Unterrainer. Catarina Gouveia Gaglianone and Hugo Senra extracted the data from selected studies. Human Unterrainer, Catarina Gouveia Gaglianone, and Hugo Senra conducted the studies' quality check and risk of bias assessment. Hugo Senra did the statistical analysis. Data were interpreted by Hugo Senra with input received from Susan McPherson, Human Unterrainer, and Catarina Gouveia Gaglianone. All findings were discussed by all authors. Hugo Senra wrote the draft and the final version of the manuscript with input received from Susan McPherson, Human Unterrainer, and Catarina Gouveia Gaglianone. All authors critically reviewed the report for important intellectual content and approved the final submitted version. All authors had full access to all the data in the study and accept responsibility to submit for publication.

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CONFLICT OF INTEREST STATEMENT

The authors have no interests to declare.

DATA AVAILABILITY STATEMENT

Please contact the corresponding author if you would like to see any data that are not included in the article or in the Supporting Information.

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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