Prevalence of alcohol use disorder among individuals who binge eat: a systematic review and meta-analysis

Krzysztof Bogusz[1] , Maciej Kopera[2] , Andrzej Jakubczyk[2] , Elisa M. Trucco[3,4,5] ,
Katarzyna Kucharska[6] , Anna Walenda[6] & Marcin Wojnar[2,7]

Nowowiejski Hospital, Warsaw, Poland,[1] Department of Psychiatry, Medical University of Warsaw, Warsaw, Poland,[2] Department of Psychology, Florida International University, Miami, Florida, USA,[3] Center for Children and Families, Florida International University, Miami, Florida, USA,[4] Department of Psychiatry, University of Michigan, Ann Arbor, MI, USA,[5] Institute of Psychology, Cardinal Stefan Wyszynski University, Warsaw, Poland[6] and Department of Psychiatry, Addiction Center, University of Michigan, Ann Arbor, MI, USA[7]

ABSTRACT

Background and Aims   Binge eating disorder (BED) is correlated with substance use. This study aimed to estimate the life-time prevalence of alcohol use disorder (AUD) among individuals with non-compensatory binge eating and determine whether their life-time prevalence of AUD is higher than in non-bingeing controls. **Design**   A systematic search of databases (PubMed, Embase and Web of Science) for studies of adults diagnosed with BED or a related behavior that also reported the life-time prevalence of AUD was conducted. The Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) protocol was followed. The protocol was registered on the International Prospective Register of Systematic Reviews (PROSPERO). **Setting**   Studies originating in Canada, Sweden, the United Kingdom and the United States. **Participants**   Eighteen studies meeting the inclusion criteria were found, representing 69 233 individuals. **Measurements**   Life-time prevalence of AUD among individuals with binge eating disorder and their life-time relative risk of AUD compared with individuals without this disorder. **Results**   The pooled life-time prevalence of AUD in individuals with binge eating disorder was 19.9% [95% confidence interval (CI) = 13.7–27.9]. The risk of life-time AUD incidence among individuals with binge eating disorder was more than 1.5 times higher than controls (relative risk = 1.59, 95% CI = 1.41–1.79). Life-time AUD prevalence was higher in community samples than in clinical samples (27.45 versus 14.45%, $P = 0.041$) and in studies with a lower proportion of women ($\beta = -2.2773$, $P = 0.044$). **Conclusions**   Life-time alcohol use disorder appears to be more prevalent with binge eating disorder than among those without.

Keywords   Alcohol abuse, alcohol dependence, alcohol use disorder, binge eating disorder, meta-analysis, systematic review.

INTRODUCTION

Binge eating disorder (BED) was introduced into the 5th edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM) in 2013 [1]. It is characterized by recurrent (≥1 per week for 3 months), periodic and uncontrolled episodes of consuming large quantities of food, which are accompanied by psychological and social distress. In contrast with bulimia nervosa, the episodes are not followed by inappropriate compensatory behaviors. According to epidemiological studies, it is the most common eating disorder in the world [2], with a life-time prevalence of 2.6% in the United States and a 3 : 2 female to male ratio [3].

Binge eating (uncontrolled episodes of consuming large quantities of food) was recognized as a clinical condition as early as 1959 [4]. Since then, clinical evidence has suggested the existence of individuals with marked distress over binge eating that could not be diagnosed with bulimia nervosa because they did not engage in compensatory behaviors [5]. Such individuals were referred to as 'obese binge eaters' or 'non-purging bulimia nervosa patients' [5,6]. BED first appeared as a diagnostic entity in 1994 in an appendix to the 4th edition of the DSM; it was a provisional diagnosis that required further research [7]. Even after its establishment as a distinct diagnosis, BED remained a heterogeneous and complex disorder [8]. Moreover, in addition to BED, the DSM-5 now recognizes a

lower-threshold form of BED [1]; research suggests that subthreshold BED does not differ significantly from full-syndrome BED regarding outcomes such as body weight, eating disorder symptoms and associated psychiatric symptoms [9]. Thus, BED may be regarded as existing on a spectrum of non-compensatory binge eating [10].

Emotional dysregulation has been noted as a predictor of binge eating [11] and is regarded as an etiological factor of BED [12]. In addition to established theories of addiction, such as the opponent process theory [13] or incentive sensitization theory [14], research also supports the role of emotion dysregulation in the development of alcohol use disorder (AUD) [15,16]. Inefficient utilization of emotion regulation strategies may increase arousal, negative affect and craving, thus fueling a vicious cycle of dependence [17]. Both binge eating and AUD may represent a maladaptive way of coping with intolerable affective states [18], and the development of one disorder may be linked with the development of the other [19]. Studies confirm higher rates of life-time AUD among individuals with BED [20] and binge-eating behavior not meeting the DSM criteria [21] compared with non-bingeing controls.

Aside from deficits in emotion regulation, neuroimaging studies conducted on individuals with BED showed impairment in impulse-control-related areas (e.g. ventromedial–prefrontal, inferior-frontal and insular cortex) [22]; similar changes were found among individuals with AUD [23]. Lee and colleagues [24] observed that individuals with BED showed stronger activation of the ventral striatum in response to food pictures than healthy controls in a cue–reactivity paradigm. These changes might indicate specific changes in reward response and difficulties with decision-making and motivation [25]. The same mechanisms contribute to the development of AUD [26].

There have been a few meta-analyses conducted on substance use among patients with various eating disorders [20,27,28]. One previous meta-analysis investigated the co-occurrence of binge eating and AUD [20]; however, the association was not the main focus of the article, as it reported only one effect size. Prior meta-analyses have several methodological limitations, such as not conforming to reporting guidelines, not pre-registering their protocols or including only a small number of studies with data on participants who binge eat. Thus, the extent of comorbidity between binge eating and AUD remains unclear, as there is a lack of systematic empirical support on this topic. Patients with co-occurring AUD and binge-eating behavior also pose unique challenges for diagnosis and treatment, including differential diagnosis and greater symptom severity [29]. Broader awareness regarding the link between AUD and binge-eating behavior could be informative of the importance of assessing past or current alcohol use and related psychopathology, as well as treating co-occurring psychopathology.

In order to address the current gap in the literature, a systematic review and meta-analysis was performed on data pertaining to the life-time prevalence of AUD in studies investigating BED and related disorders. The primary aim of this systematic review and meta-analysis was to assess the life-time prevalence of AUD among individuals who binge eat. The secondary aim of this study was to determine if the life-time prevalence of AUD among individuals who binge eat is higher than in non-bingeing controls.

MATERIALS AND METHODS

This systematic review and meta-analysis was registered with International Prospective Register of Systematic Reviews (PROSPERO) (CRD42019140622) and conducted using an a priori protocol. It was carried out in accordance with the guidelines for Meta-analysis of Observational Studies in Epidemiology (MOOSE) [30] and the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) [31].

Search strategy

The search strategy was developed by two researchers with experience in eating disorders and substance use (M.K., A.J.). Three electronic databases (PubMed, Embase and Web of Science) were searched for articles published from 1 January 1966 to 1 June 2019 for adequate and efficient coverage [32]. Additionally, as per prior recommendations [32], the first 200 relevant references from Google Scholar were screened using a shortened search strategy. There were no restrictions on language or geographical location. The search was limited to studies performed in humans. The following search terms were used: 'binge eating', 'binge eating disorder', 'alcohol', 'alcoholism', 'alcohol use', 'alcohol use disorder', 'alcohol consumption', 'alcohol abuse', 'alcohol drinking', 'substance abuse', 'substance use', 'correlate', 'co-occurrence' and 'association'. Medical subject headings and 'explode' commands were used. The complete search strategy is provided in Supporting information, Table S1. Reference lists of all relevant articles were screened to identify any studies missed in the initial search. References were managed using Mendeley Desktop version 1.19.4.

Study selection

Two reviewers (K.B., M.K.) independently screened titles and abstracts. Criteria were wide-ranging, to include all potentially relevant studies. Studies had to report on associated psychopathology in adult patients with disordered eating for full text review. Articles that focused upon binge-eating comorbidity in a subgroup of patients with another specific mental disorder (e.g. major depressive disorder, bipolar disorder) were excluded, as their inclusion

could bias the outcome. References that consisted only of abstracts, case reports or case-series were also excluded. Titles and abstracts in languages other than English, such as German, French or Spanish, were either translated or available in English online.

Next, the full text of any study selected by either reviewer was obtained. Articles were eligible if they: (1) consisted of original research; (2) were epidemiological, case-controlled or longitudinal studies; (3) included individuals diagnosed with BED using DSM-IV or DSM-5 criteria; (4) included individuals diagnosed with subthreshold BED or binge-eating behavior (BEB) using other defined criteria (e.g. Composite International Diagnostic Interview, Questionnaire on Eating and Weight Patterns–revised) or those meeting partial DSM criteria; and (5) reported the life-time prevalence of AUD among those individuals. Articles were excluded if they: (1) were performed using an underage (<18 years old) sample; (2) used a sample that was chosen using selective sampling (i.e. subjective criteria or personal judgment); (3) measured BED comorbidity only in a subgroup of individuals with a specific mental disorder; or (4) did not include a description of the criteria used for establishing an AUD diagnosis.

Data extraction and quality assessment

Data extraction was performed by two reviewers (K.B., A.J.), according to a predefined coding protocol (Supporting information, Table S2). Disagreement was resolved by discussion. The following data were recorded in a Google Sheets spreadsheet: bibliographic data, design details, sample characteristics and measures of outcomes. If a study reported more than one outcome, each was recorded as a separate group. If necessary, one reviewer (K.B.) contacted the corresponding author to ask for additional data.

Two reviewers (K.B., A.J.) independently assessed the methodological quality of the articles using a modified version of the Newcastle–Ottawa Scale, a scale used to evaluate the quality of non-randomized studies (Supporting information, Fig. S1) [33]. It includes seven items grouped into three categories: selection, comparability and outcome. The scale is scored from zero to eight stars. Studies were identified as having an overall low risk of bias (≥6 stars) or a high risk of bias (<6 stars).

Data synthesis and analysis

The primary outcome measure was the prevalence of life-time AUD among individuals engaging in binge eating. The secondary outcome measure was the relative risk (RR) of AUD among patients engaging in binge eating compared to a non-bingeing control group. To avoid excluding individuals diagnosed prior to the publication of the DSM diagnostic criteria for BED, we decided to include studies on individuals who engaged in all non-compensatory binge

eating, with a later subanalysis of individuals diagnosed according to DSM-IV and DSM-5. For studies using DSM-III and DSM-IV diagnostic criteria, the number of individuals with alcohol abuse and alcohol dependence was summed to represent the number of individuals with AUD. In this way, older diagnoses are comparable with the DSM-5 AUD diagnosis with substantial to almost perfect agreement [34].

To perform the meta-analyses, we used the 'meta' and 'metafor' packages [35] within the R software environment, version 3.6.0 [36]. Meta-analyses of prevalence produce the weighted average proportion, which is an average of the results of multiple studies weighted by the inverse of their sampling variances [37]. As design parameters and sample characteristics would probably vary, a random-effects model was chosen [38]. The proportions among included studies could be less than 0.2 or greater than 0.8; therefore, we used logit transformation, which handles small samples and extreme proportions more precisely than the direct proportions method [39].

We used the DerSimonian–Laird method to estimate between-study variance, as it is better equipped to handle non-normally distributed study effects than the restricted maximum likelihood [40]. The between-study variance was measured via the τ^2 statistic and the presence of heterogeneity was identified by using the Q-test. Heterogeneity can be described as genuine differences underlying the results of the studies; meta-analyses are less generalizable with increased heterogeneity among included studies [41]. Heterogeneity was quantified via the I^2 statistic, which estimates the amount of the observed heterogeneity that constitutes the true variation between studies rather than chance. The Cochrane Handbook proposes a classification where I^2 of 30–60% indicates moderate heterogeneity, 50–90% indicates substantial heterogeneity and I^2 greater than 75% indicates considerable heterogeneity [42].

We performed subgroup analyses investigating the difference in the outcome measures between studies conducted in community settings (individuals from a given area regardless of treatment status) versus clinical settings (individuals treated for BED at a hospital or clinic); studies assessing the AUD diagnosis using different DSM versions; and between studies with low and high risk of bias. To help explain residual heterogeneity and to assess the potential effect of factors on the outcome, we ran meta-regression analyses for the proportion of females and publication year. The R^2 statistic was assessed, regarding the amount of true heterogeneity that could be explained by tested moderators.

We evaluated the sensitivity of our analyses by comparing fitted models with and without samples that we assumed to be influential outliers and by excluding samples without a confirmed BED diagnosis. Influential outliers

were identified using the 'influence' function. Publication bias and small study effects were assessed with the 'funnel' function, which funnels plots for the visual detection of asymmetries. In addition, the Egger test for the detection of asymmetry in the funnel plot was performed; we considered analyses to be statistically significant if the *P*-value was <0.10 [43]. For other outcomes, the *P*-value <0.05 was considered statistically significant.

RESULTS

Literature search

We identified 6469 entries through database searches. After removing duplicates, we screened a total of 4044 unique records and excluded 3867 that were determined to not be relevant (Fig. 1). Next, the full text of 177 articles was reviewed. Of these, 18 studies describing 69 233 individuals met the inclusion criteria [21,29,44–59]. Ten of those studies included a comparison group [21,44–47,49,51,56–58]. Two studies [21,55] included two samples each, which were recorded as separate groups for the meta-analyses. The most relevant characteristics of

included studies are summarized in Table 1 (e.g. country of origin, DSM version, sample type).

Life-time prevalence of AUD among binge-eating individuals

A total of 20 samples including data from 69 233 participants reported life-time prevalence of AUD among individuals who binge eat. Their findings are summarized in Fig. 1.

The overall pooled life-time prevalence of AUD was 19.9% [95% confidence interval (CI) = 13.7–27.9, *P*-value < 0.0001]. There was considerable heterogeneity present (I^2 = 96.6%; *Q*-test *P*-value < 0.0001).

Relative risk of life-time AUD between binge-eating individuals and non-bingeing controls

A total of 11 samples, including data from 67 652 participants, reported life-time prevalence of AUD among individuals who binge eat compared with a non-bingeing control group. The results are presented in Fig. 2.

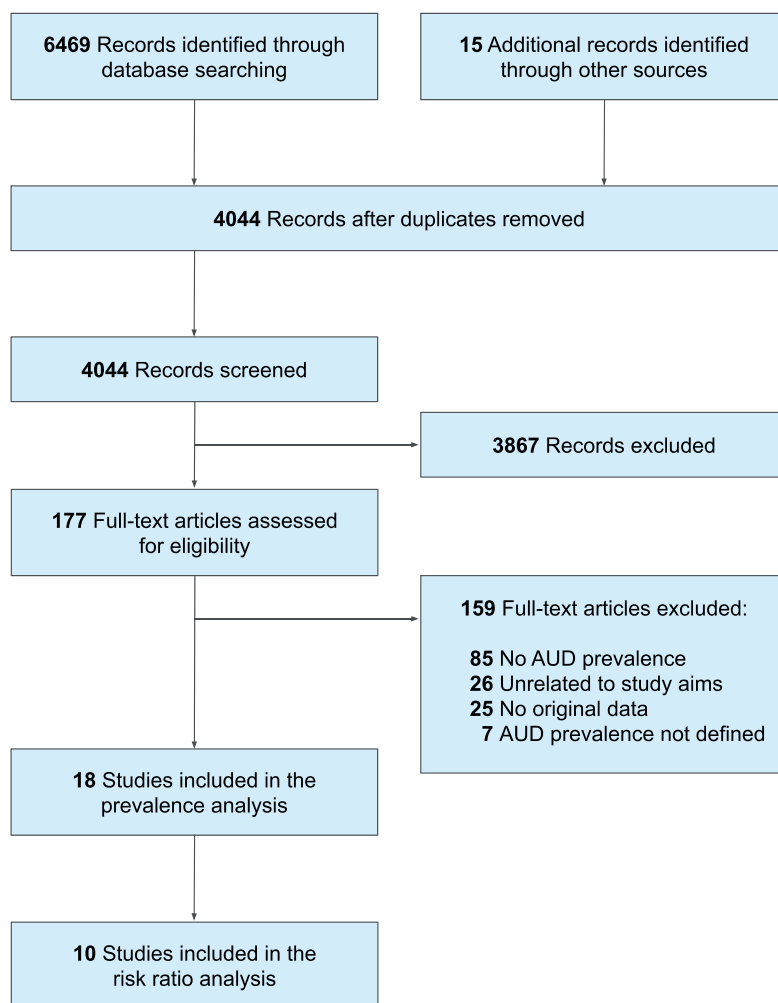Results indicate that the incidence of life-time AUD among individuals engaging in binge eating was more than

Figure I Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) flow diagram describing study selection [Colour figure can be viewed at wileyonlinelibrary.com]

Table 1 Characteristics of included studies.

First author and year	Country	Number of participants who binge eat	Number of controls	Type of sample	Percentage of women	Binge eating criteria	AUD criteria	Basis of AUD diagnosis	Percentage of AUD in BE group	Percentage of AUD in controls	Percentage of non-white participants	Mean age of BE group in years	Mean age of controls in years
Becker & Grilo 2015	USA	347	NA	CLIN	85%	DSM-IV	DSM-IV	Clinical interview	22.48%	NA	19.02%	44.7	NA
Dohm et al. 2002	USA	162	NA	COMM	100%	DSM-IV	DSM-IV	Clinical interview	53.70%	NA	43.83%	NR	NA
Grilo et al. 2009	USA	404	NA	CLIN	77%	DSM-IV	DSM-IV	Clinical interview	20.30%	NA	18.81%	44.9	NA
Grilo et al. 2013	USA	142	NA	CLIN	74%	DSM-IV	DSM-IV	Clinical interview	12.68%	NA	57.04%	43.6	NA
Hudson et al. 2007a	USA	84	NA	COMM	73%	DSM-IV	DSM-IV	Clinical interview	21.43%	NA	NR	NR	NA
Hudson et al. 2007b	USA	36	NA	COMM	31%	Own criteria	DSM-IV	Clinical interview	30.56%	NA	NR	NR	NA
Javaras et al. 2008a	USA	285	849	COMM	68%	DSM-IV	DSM-IV	Clinical interview	25.26%	17.55%	NR	46.4	48.0
Javaras et al. 2008b	USA	54	849	COMM	67%	Own criteria	DSM-IV	Clinical interview	20.37%	17.55%	NR	46.2	48.0
Johnson et al. 2001	USA	245	4362	CLIN	100%	DSM-IV	DSM-IV	Self-report	6.12%	3.99%	NR	NR	NR
Lee et al. 2018	USA, CA	125	441	CLIN	55%	DSM-5	DSM-IV	Clinical interview	60.80%	45.12%	14.84%	35.7	39.2
Mitchell et al. 2015	USA	350	1875	CLIN	77%	DSM-5	Own criteria	Self-report	12.29%	6.67%	13.03%	NR	NR
Pike et al. 2001	USA	150	150	COMM	100%	DSM-IV	DSM-IV	Clinical interview	34.67%	NA	34.67%	31.3	NR
Robertson & Palmer 1997	UK	15	47	COMM	100%	Own criteria	DSM-III	Clinical interview	13.33%	6.38%	NR	NR	NR
Root et al. 2010	SE	49	12 508	COMM	100%	DSM-IV	DSM-IV	Clinical interview	14.29%	6.04%	NR	31.9	33.7
Specker et al. 1994	USA	43	57	CLIN	100%	DSM-IV	DSM-III	Clinical interview	16.28%	15.79%	NR	NR	NR
Telch & Stice 1998	USA	61	60	COMM	100%	DSM-IV	DSM-III	Clinical interview	14.75%	10.00%	26.45%	43.5	5.0

(*Continues*)

Table 1 (Continued)

First author and year	Country	Number of participants who binge eat	Number of controls	Type of sample	Percentage of women	Binge eating criteria	AUD criteria	Basis of AUD diagnosis	Percentage of AUD in BE group	Percentage of AUD in controls	Percentage of non-white participants	Mean age of BE group in years	Mean age of controls in years
Udo et al. 2016	CA	429	NA	CLIN	72%	DSM-5	DSM-IV	Clinical interview	18.18%	NA	72.49%	46.2	NA
Udo & Grilo 2019	USA	318	35 709	COMM	57%	DSM-5	DSM-5	Clinical interview	51.89%	28.70%	47.1%	NR	NR
Ullvebrand et al. 2015	SE	526	NA	CLIN	95%	DSM-IV	DSM-IV	Clinical interview	7.60%	NA	NR	NR	NR
Welch et al. 2016	SE	850	8500	CLIN	95%	DSM-IV	DSM-IV	Clinical interview	3.18%	1.79%	NR	NR	NR

CLIN = clinical; COMM = community; NA = not applicable; NR = not reported; BE = binge eating; CA = Canada; SE = Sweden; AUD = alcohol use disorder.

1.5 times higher in comparison to non-bingeing controls [relative risk (RR) = 1.59, 95% CI = 1.41–1.79]. The heterogeneity was not statistically significant (I^2 = 26.4%; Q-test P-value = 0.19).

Sensitivity analysis, moderator analysis and sources of heterogeneity

Prevalence

There was a significant amount of heterogeneity among the included studies reporting prevalence: the I^2 statistic was 96.6% (95% CI = 93.5–98.2); the Q-test P-value was <0.0001 and τ^2 was equal to 0.970 (95% CI = 0.483–1.891). Sensitivity analysis did not reveal any significant outliers, and excluding samples without a BED diagnosis did not influence the result (19.6%, 95% CI = 13.0–28.5).

The results of subgroup analyses are detailed in Table 2. Among subgroups tested, only one significant effect was found; namely, findings indicate that AUD prevalence is higher among studies performed in a community setting compared to a clinical setting (27.45 versus 14.45%, P-value = 0.0412; Supporting information, Fig. S2). The amount of heterogeneity explained by this difference was 27.21% (the R^2 statistic).

The slope (β), 95% CIs and P-values for meta-regression models investigating the proportion of women and publication year are detailed in Table 4. A significant effect was found whereby the prevalence of AUD was lower in studies with a larger proportion of women (β = −2.2773, P-value = 0.0441; Supporting information, Fig. S4). This moderator explained 14.56% of the heterogeneity. Publication year did not significantly influence the results.

Relative risk

Statistically significant heterogeneity was not detected among studies reporting the relative risk of AUD between binge-eating individuals and non-bingeing controls; the I^2 statistic was 26.4% (95% CI = 0.00–74.9), the Q-test P-value was 0.19 and τ^2 was equal to 0.009 (95% CI = 0.00–0.072). Sensitivity analyses revealed two influential outliers. However, removing them from the analysis did not affect the final outcome (RR = 1.59, 95% CI = 1.41–1.79 versus RR = 1.57, 95% CI = 1.35–1.83). Moreover, excluding samples without a BED diagnosis did not influence the result (1.61, 95% CI = 1.42–1.82).

The results of subgroup analyses are detailed in Table 3. Among subgroups, only one difference was found; namely, the rate of AUD among individuals who engaged in binge eating did not differ significantly from non-bingeing controls in studies that used the DSM-III diagnostic criteria. The amount of heterogeneity explained by this difference was 100%.
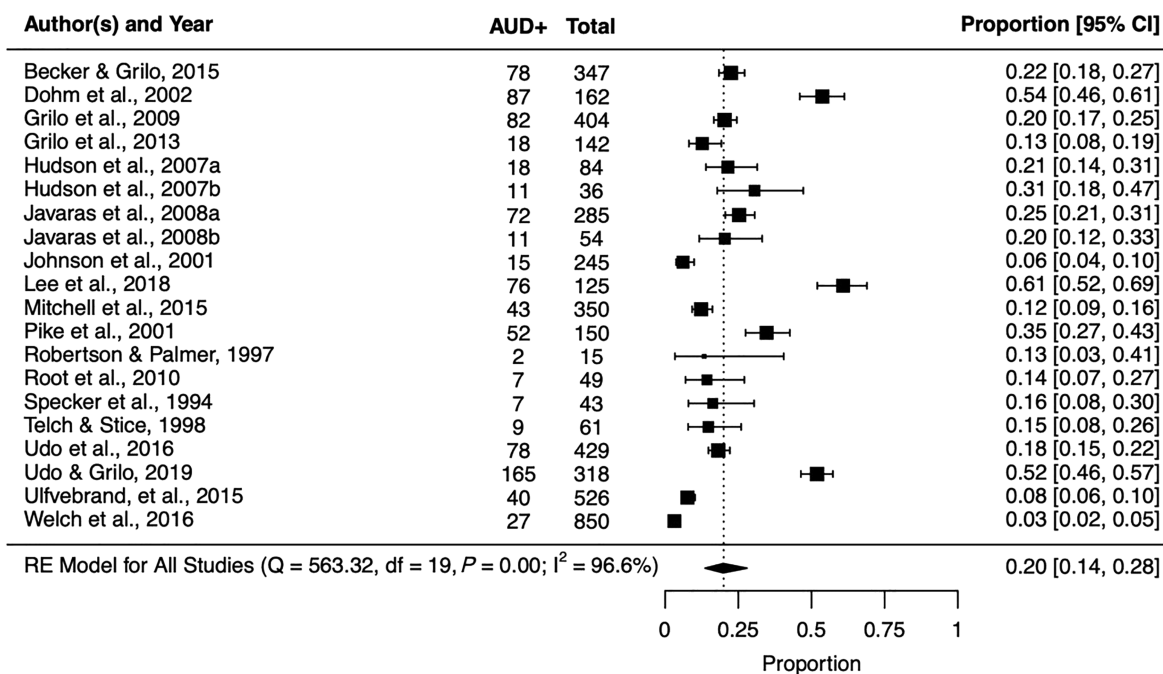
Author(s) and Year	AUD+	Total		Proportion [95% CI]
Becker & Grilo, 2015	78	347		0.22 [0.18, 0.27]
Dohm et al., 2002	87	162		0.54 [0.46, 0.61]
Grilo et al., 2009	82	404		0.20 [0.17, 0.25]
Grilo et al., 2013	18	142		0.13 [0.08, 0.19]
Hudson et al., 2007a	18	84		0.21 [0.14, 0.31]
Hudson et al., 2007b	11	36		0.31 [0.18, 0.47]
Javaras et al., 2008a	72	285		0.25 [0.21, 0.31]
Javaras et al., 2008b	11	54		0.20 [0.12, 0.33]
Johnson et al., 2001	15	245		0.06 [0.04, 0.10]
Lee et al., 2018	76	125		0.61 [0.52, 0.69]
Mitchell et al., 2015	43	350		0.12 [0.09, 0.16]
Pike et al., 2001	52	150		0.35 [0.27, 0.43]
Robertson & Palmer, 1997	2	15		0.13 [0.03, 0.41]
Root et al., 2010	7	49		0.14 [0.07, 0.27]
Specker et al., 1994	7	43		0.16 [0.08, 0.30]
Telch & Stice, 1998	9	61		0.15 [0.08, 0.26]
Udo et al., 2016	78	429		0.18 [0.15, 0.22]
Udo & Grilo, 2019	165	318		0.52 [0.46, 0.57]
Ulfvebrand, et al., 2015	40	526		0.08 [0.06, 0.10]
Welch et al., 2016	27	850		0.03 [0.02, 0.05]

RE Model for All Studies (Q = 563.32, df = 19, P = 0.00; I^2 = 96.6%)   0.20 [0.14, 0.28]

0   0.25   0.5   0.75   1
Proportion

Figure 2 Forest plot of the random-effects meta-analysis of alcohol use disorder (AUD) prevalence in binge eating individuals

Table 2 Subgroup analysis of categorical moderators for samples describing prevalence.

Moderator	Prevalence (%)	95% CI (%)	I^2 statistic	Test of moderators P-value	R^2 statistic
Sample characteristics				0.0412	27.21%
Community setting	27.45	17.8, 39.81	91%		
Clinical setting	14.45	8.98, 22.42	97%		
AUD criteria				0.3763	9.30%
Own criteria	12.29	2.12, 47.5	NA		
DSM-III	14.9	4.97, 36.96	0%		
DSM-IV	19.77	13.13, 28.67	97%		
DSM-5	51.89	14.48, 87.29	NA		
Study quality				0.1487	5.46%
Low risk of bias	22.39	15.11, 31.86	97%		
High risk of bias	11.13	4.30, 25.86	61%		

CI = confidence interval; NA = not applicable; own criteria = alcohol use disorder (AUD) criteria used by study authors who did not use the DSM (e.g. Alcohol Use Disorders Identification Test).

The slope (β), 95% CIs and *P*-values for meta-regression models are detailed in Table 4. The two meta-regressions indicated that neither gender nor publication year were statistically significant.

Assessment of quality

Among all studies, 16 achieved scores of at least 6 stars, indicating low risk of bias (Supporting information, Table S3). Four studies failed to reach this threshold and were judged to be at high risk of bias [44,46,56,58]. They were the only studies with a high risk of bias among those reporting relative risk.

Excluding studies with a high risk of bias from the analysis did not significantly influence the results; namely, the result for prevalence was 22.38% (95% CI = 14.95–32.11) and 1.57 (95% CI = 1.34–1.83) for relative risk.

The item with the largest amount of bias was the assessment of participants that dropped out of the study. Only six studies included any information regarding participants who dropped out of the study early.

Publication bias

The number of studies included was sufficient to perform publication bias testing [60]. Among studies reporting prevalence, as well as in studies reporting relative risk,

Table 3 Subgroup analysis of categorical moderators for samples describing relative risk.

Moderator	Relative risk	95% CI	I^2 statistic	Test of moderators P-value	R^2 statistic
Sample characteristics				0.2499	55.83%
Community setting	1.68	1.46, 1.93	13.3%		
Clinical setting	1.49	1.28, 1.73	6.0%		
AUD criteria				0.0331	100.00%
Own criteria	1.84	1.33, 2.56	0%		
DSM-III	1.31	0.71, 2.42	0%		
DSM-IV	1.43	1.27, 1.62	0%		
DSM-5	1.81	1.62, 2.01	0%		
Study quality				0.7668	0.00%
Low risk of bias	1.68	1.46, 1.93	13.3%		
High risk of bias	1.49	1.41, 1.73	6.0%		

CI = confidence interval; NA = not applicable; own criteria = alcohol use disorder (AUD) criteria used by study authors who did not use the DSM (e.g. Alcohol Use Disorders Identification Test).

Table 4 Meta-regression of continuous moderators.

Prevalence

Moderator	Slope (β)	95% CI	Test of moderators P-value	R^2 statistic
Proportion of women	−2.2773	−4.4940, −0.0607	0.0441	14.56%
Publication year	0.0075	−0.0570, 0.0719	0.8207	0.00%

Relative risk

Moderator	Slope (β)	95% CI	Test of moderators P-value	R^2 statistic
Proportion of women	0.2075	−0.5892, 1.0042	0.6098	0.00%
Publication year	0.0097	−0.0088, 0.0281	0.3039	20.68%

CI = confidence interval; NA = not applicable.

there did not appear to be publication bias upon visual inspection of the funnel plot. There was no evidence of small study effects in either group as indicated by the Egger regression test (Supporting information, Figs S5, S6); tests for funnel plot asymmetry were not significant (P-values = 0.335 and 0.806, respectively). Based on these results, the risk of publication bias in this study was determined to be low.

DISCUSSION

This study investigated the life-time prevalence of AUD among individuals who binge eat and their relative risk of life-time AUD compared to non-bingeing controls. There are two main findings. First, the overall life-time prevalence of AUD among individuals who binge eat is 19.9% or just under one in five patients (Fig. 2). Secondly, individuals who binge eat are more than 1.5 times more likely to be diagnosed with AUD in their life-time when compared to non-bingeing controls (Fig. 3). These results are consistent

with previous reports and extend prior findings. For example, a previous meta-analysis by Gadalla and colleagues [20] used a technique where the effect size in each study was compared to the variability observed in that study; it reported the outcome as a standardized mean difference (SMD). Although not its main focus, it included five studies on individuals with BED and found that this disorder was moderately positively associated with AUD (SMD = 0.39).

There are a number of explanations for this outcome. Both food and alcohol activate the reward systems [61], which may reflect a common neurobiological mechanism underlying both AUD and BED. In general, both food and alcohol may be used by individuals seeking relief and/or craving reward, which are thought to be important mechanisms involved in excessive substance consumption [26]. Individuals who act impulsively when experiencing negative emotional states were shown to be more likely to develop addictive eating patterns [62] and at greater risk for other addictive behaviors, including AUD [63]. Additionally, increased negative urgency and
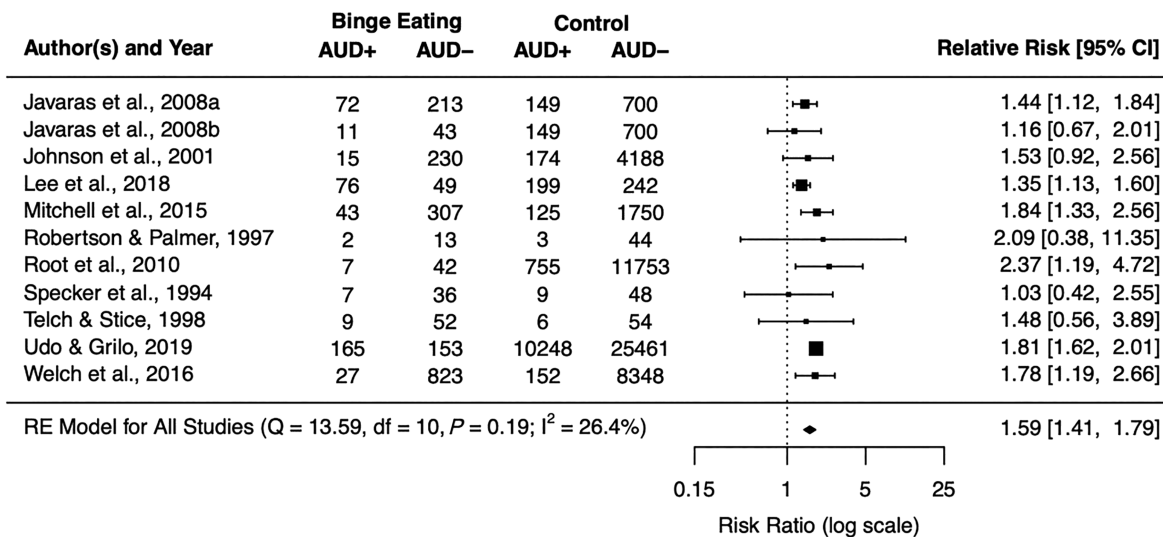
Author(s) and Year	Binge Eating		Control			Relative Risk [95% CI]
	AUD+	AUD−	AUD+	AUD−		
Javaras et al., 2008a	72	213	149	700		1.44 [1.12, 1.84]
Javaras et al., 2008b	11	43	149	700		1.16 [0.67, 2.01]
Johnson et al., 2001	15	230	174	4188		1.53 [0.92, 2.56]
Lee et al., 2018	76	49	199	242		1.35 [1.13, 1.60]
Mitchell et al., 2015	43	307	125	1750		1.84 [1.33, 2.56]
Robertson & Palmer, 1997	2	13	3	44		2.09 [0.38, 11.35]
Root et al., 2010	7	42	755	11753		2.37 [1.19, 4.72]
Specker et al., 1994	7	36	9	48		1.03 [0.42, 2.55]
Telch & Stice, 1998	9	52	6	54		1.48 [0.56, 3.89]
Udo & Grilo, 2019	165	153	10248	25461		1.81 [1.62, 2.01]
Welch et al., 2016	27	823	152	8348		1.78 [1.19, 2.66]
RE Model for All Studies (Q = 13.59, df = 10, P = 0.19; I^2 = 26.4%)						1.59 [1.41, 1.79]

<div align="center">0.15   1   5   25</div>

<div align="center">Risk Ratio (log scale)</div>

Figure 3 Forest plot of the random-effects meta-analysis of relative risk of alcohol use disorder (AUD) incidence in binge eating individuals compared to non-bingeing controls

impulsivity have been demonstrated in both BED [64] and AUD [65].

Sources of variation

Differences in effects of continuous moderators and between subgroups were present among studies reporting prevalence rates. A diagnosis of AUD was more common in studies with a higher proportion of men; in studies which only included women, the prevalence of AUD was nearly two times lower in comparison to studies where men accounted for half the sample. This is consistent with reported biological sex ratios of alcohol abuse and alcohol dependence in the general population, where these disorders are twice as common in men than in women [66,67]. AUD prevalence was also two times higher in community samples as opposed to samples from a clinical setting. This difference may exist for a number of reasons. Despite being the most common eating disorder, BED is still underdiagnosed, and many patients may go untreated [68,69]; half of those who were being treated learned about their disorder on their own [70]. Thus, the treated population may represent the group most motivated to seek help. There are also a number of barriers to AUD treatment, especially among people with a serious comorbidity [71]; the difference between the general population and those in treatment may represent a selection bias. Additionally, in a study comparing individuals diagnosed with BED and those meeting the DSM-5 BED diagnostic criteria, but who were previously undiagnosed [70], it was found that the diagnosed group had higher socio-economic status, a trait linked to a reduced risk of alcohol-attributable harms [72].

In studies reporting relative risk of AUD, the rate of AUD among individuals who engaged in binge eating did not differ significantly from a comparison group in studies using DSM-III diagnostic criteria. Even though the three studies using DSM-III criteria had an earlier year of publication, publication year was not found to influence either prevalence or relative risk of diagnosis. This result may simply stem from a small number of participants included in this subgroup (283 versus 30372 in DSM-IV and 36 027 in DSM-5) and resulting in insufficient statistical power [73], or it may represent discrepancies between versions of the DSM [74].

Not all individuals who binge eat meet full DSM-IV or DSM-5 criteria for BED. For example, they may present with binge-eating behavior (i.e. consume amounts of food larger than what most people would eat in a similar period), but without a sense of lack of control, or engage in binge eating less often than once a week. Studies on individuals who did not meet DSM criteria for BED, but engaged in non-compensatory binge-eating behavior, were included. There were three samples included in this analysis which did not meet DSM criteria; excluding them did not significantly influence either prevalence or relative risk results.

Strengths and limitations

This study's strengths include a strict and comprehensive analytical approach using an a priori protocol, which was pre-registered with PROSPERO and conducted in accordance with PRISMA and MOOSE guidelines. Pre-registration helps to minimize bias by outlining analyses a priori and compels the researchers to formulate a

study rationale for a specific research question [75]. We did not exclude studies based on their geographic location or language to make the results more generalizable. Data extraction and quality assessment were performed using predefined protocols between two independent researchers to further reduce possible bias resulting from arbitrary decision-making. The overall quality of included studies was high and there was no evidence of publication bias.

This study has several limitations. First, despite intentionally employing broad search criteria and comprehensive methods, some eligible studies may not have been identified. Secondly, all included studies came from developed countries, mainly from the United States, so their pooled findings may not be applicable to other populations. Thirdly, because the included studies spanned 25 years, there are marked differences in how AUD was classified in different versions of the DSM. This may result in variability among studies using different criteria. Conversely, publication year was not found to be a significant moderator; studies have also found that there is substantial to almost perfect agreement between DSM-5 classifications of AUDs and those based on the DSM-IV and DSM-III [34].

Fourthly, among studies reporting prevalence rates, there was considerable between-study heterogeneity, suggesting either significant differences in study design, study population or the presence of moderating factors. The reasons behind this variability were explored in a subgroup analysis and meta-regression. There were no outliers that could singularly influence the amount of heterogeneity. Additional moderator analyses revealed that setting and proportion of women were probable moderators; however, these factors could only explain less than half of this heterogeneity. Despite our efforts to only include similar studies by setting eligibility and exclusion criteria, there were considerable discrepancies both in their design and the populations examined.

Lastly, the results of this study are limited by the quality of included studies and their methodology. Three samples included fewer than 100 participants and half of all included studies reported data on fewer than 500 participants. Moreover, only 10 studies specified the race and/or ethnicity of study participants. Therefore, it is unclear whether these findings are generalizable to diverse populations. In conjunction with a relatively low prevalence of disorders analyzed here, this may indicate that some studies were statistically underpowered.

CONCLUSIONS

To our knowledge, this is the first systematic review and meta-analysis to investigate the life-time prevalence of AUD among individuals who binge eat. Findings indicate that life-time AUD is commonly comorbid with binge eating, as one in five individuals who binge eat also meet AUD criteria. When compared with non-bingeing controls, individuals who binge eat are 1.5 times more likely to have a life-time diagnosis of AUD. AUD's prevalence is higher among men than women and in community samples compared to clinical samples. The relative risk in the incidence of AUD did not significantly differ between individuals who binge eat and controls in studies using DSM-III to derive AUD criteria.

In general, our findings indicate that specialists should consider assessing for past or current presence of alcohol use and associated psychopathology among clients who present with binge eating. Future research is warranted that employs similar analyses with studies that include larger sample sizes, represent demographically diverse individuals, focus upon the new DSM-5 criteria, investigate the impact of setting and explore the link between BED and AUD among males. Longitudinal studies investigating whether BED influences the development of AUD or vice versa are also needed.

Author contributions

Krzysztof Bogusz: Conceptualization; data curation; formal analysis; investigation; methodology; project administration; resources; software; visualization. **Maciej Kopera:** Conceptualization; data curation; formal analysis; investigation; methodology; project administration; resources; supervision. **Andrzej Jakubczyk:** Conceptualization; data curation; formal analysis; investigation; methodology; project administration; resources; supervision. **Elisa Trucco:** Conceptualization; methodology; supervision. **Katarzyna Kucharska:** Conceptualization; methodology. **Anna Walenda:** Conceptualization; methodology. **Marcin Wojnar:** Conceptualization; methodology; project administration; supervision.

References

1. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders, 5th edn.* American Psychiatric Association; 2013. Available at: https://psychiatryonline.org/doi/book/10.1176/appi.books.9780890425596 (accessed 24 June 2020).

2. Hilbert A. Binge-eating disorder. *Psychiatr Clin North Am* [internet] 2019; **42**: 33–43. Available at: https://doi.org/10.1016/j.psc.2018.10.011 (accessed 24 June 2020).

3. Guerdjikova A. I., Mori N., Casuto L. S., McElroy S. L. Binge eating disorder. *Psychiatr Clin North Am* [internet] 2017; **40**: 255–66. Available at: http://www.ncbi.nlm.nih.gov/pubmed/28477651 (accessed 24 June 2020).

4. Stunkard A. J. Eating patterns and obesity. *Psychiatr Q [internet].* 1959; **33**: 284–95. Available at: http://link.springer.com/10.1007/BF01575455 (accessed 24 June 2020).

5. Spitzer R. L., Devlin M. J., Walsh B. T., Hasin D., Wing R., Marcus M. D., *et al.* Binge eating disorder: to be or not to be in DSM-IV. *Int J Eat Disord* 1991; **10**: 627–9.

6. Spitzer R. L., McCann U. D., Agras W. S. Nonpurging bulimia nervosa and binge eating disorder. *Am J Psychiatry [internet]* 1991; **148**: 1097–8. Available at: http://www.embase.com/search/results?subaction=viewrecord&from=export&id=L21 260940 (accessed 24 June 2020).

7. American Psychiatric Association (APA) *Diagnostic and Statistical Manual of Mental Disorders, 4th edn. Philadelphia, PA: APA; 1994.*

8. Wilfley D. E., Citrome L., Herman B. K. Characteristics of binge eating disorder in relation to diagnostic criteria. *Neuropsychiatr Dis Treat* 2016; **12**: 2213–23.

9. Striegel-Moore R. H., Dohm F. A., Solomon E. E., Fairburn C. G., Pike K. M., Wilfley D. E. Subthreshold binge eating disorder. *Int J Eat Disord [internet].* 2000; **27**: 270–8. Available at: https://doi.org/10.1002/(sici)1098-108x(200004)27:3%3C270::aid-eat3%3E3.0.co;2-1 (accessed 24 June 2020).

10. Devlin M. J., Goldfein J. A., Dobrow I. What is this thing called BED? Current status of binge eating disorder nosology. *Int J Eat Disord* 2003; **34**: 2–18.

11. Laghi F., Bianchi D., Pompili S., Lonigro A., Baiocco R. Metacognition, emotional functioning and binge eating in adolescence: the moderation role of need to control thoughts. *Eat Weight Disord* [internet] 2018; **23**: 861–9. Available at: http://link.springer.com/10.1007/s40519-018-0603-1 (accessed 24 June 2020).

12. Kober H., Boswell R. G. Potential psychological and neural mechanisms in binge eating disorder: implications for treatment. *Clin Psychol Rev* 2018; **60**: 32–44.

13. Koob G. F., Le Moal M. Neurobiological mechanisms for opponent motivational processes in addiction. *Philos Trans R Soc B Biol Sci* 2008; **363**: 3113–23.

14. Berridge K. C., Robinson T. E. Liking, wanting, and the incentive-sensitization theory of addiction.. *American Psychologist.* 2016; **71**: 670–9. https://doi.org/10.1037/amp0000059

15. Kober H. *Emotion Regulation in Substance Use Disorders. Handbook of Emotion Regulation, 2nd edn. New York, NY: Guilford Press; 2014, pp. 428–46.*

16. Koob G. F. The dark side of emotion: the addiction perspective. *Eur J Pharmacol* 2015; **753**: 73–87.

17. Petit G., Luminet O., Maurage F., Tecco J., Lechantre S., Ferauge M., *et al.* Emotion regulation in alcohol dependence. *Alcohol Clin Exp Res* [internet] 2015; **39**: 2471–9 Available at: http://www.ncbi.nlm.nih.gov/pubmed/26613633 (accessed 24 June 2020).

18. Killeen T., Brewerton T. D., Campbell A., Cohen L. R., Hien D. A. Exploring the relationship between eating disorder symptoms and substance use severity in women with comorbid PTSD and substance use disorders. *Am J Drug Alcohol Abuse* 2015; **41**: 547–52.

19. Wolfe W. L., Maisto S. A. The relationship between eating disorders and substance use: moving beyond co-prevalence research. *Clin Psychol Rev* 2000; **20**: 617–31.

20. Gadalla T., Piran N. Co-occurrence of eating disorders and alcohol use disorders in women: a meta analysis. *Arch Womens Ment Health* [internet] 2007; **10**: 133–40. Available at: http://www.ncbi.nlm.nih.gov/pubmed/17533558 (accessed 24 June 2020).

21. Javaras K. N., Pope H. G., Lalonde J. K., Roberts J. L., Nillni Y. I., Laird N. M., *et al.* Co-occurrence of binge eating disorder with psychiatric and medical disorders. *J Clin Psychiatry [internet]* 2008; **69**: 266–73. Available at: http://www.embase.com/search/results?subaction=viewrecord&from=export&id=L35 1469270 (accessed 24 June 2020).

22. Kessler R. M., Hutson P. H., Herman B. K., Potenza M. N. The neurobiological basis of binge-eating disorder. *Neurosci Biobehav Rev [internet]* 2016; **63**: 223–38. Available at: http://www.embase.com/search/results?subaction=viewrecord&from=export&id=L608598867 (accessed 24 June 2020).

23. Fritz M., Klawonn A. M., Zahr N. M. Neuroimaging in alcohol use disorder: from mouse to man. *J Neurosci Res* 2019; https://doi.org/10.1002/jnr.24423 (accessed 24 June 2020).

24. Lee J. E., Namkoong K., Jung Y. C. Impaired prefrontal cognitive control over interference by food images in binge-eating disorder and bulimia nervosa. *Neurosci Lett* [internet 2017; **651**: 95–101. Available at: https://doi.org/10.1016/j.neulet.2017.04.054 (accessed 24 June 2020).

25. Donnelly B., Touyz S., Hay P., Burton A., Russell J., Caterson I. Neuroimaging in bulimia nervosa and binge eating disorder: a systematic review. *J Eat Disord* 2018; **6**; https://doi.org/10.1186/s40337-018-0187-1 (accessed 24 June 2020).

26. Sinha R. Role of addiction and stress neurobiology on food intake and obesity. *Biol Psychol* 2018; **131**: 5–13.

27. Bahji A., Mazhar M. N., Hudson C. C., Nadkarni P., MacNeil B. A., Hawken E. Prevalence of substance use disorder comorbidity among individuals with eating disorders: a systematic review and meta-analysis. *Psychiatry Res* 2019; **273**: 58–66.

28. Calero-Elvira A., Krug I., Davis K., López C., Fernández-Aranda F., Treasure J. Meta-analysis on drugs in people with eating disorders. *Eur Eat Disord Rev [internet]* 2009; **17**: 243–59. Available at: http://www.embase.com/search/results?subaction=viewrecord&from=export&id=L355695295 (accessed 24 June 2020).

29. Becker D. F., Grilo C. M. Comorbidity of mood and substance use disorders in patients with binge-eating disorder: associations with personality disorder and eating disorder pathology. *J Psychosom Res [internet]* 2015; **79**: 159–64. Available at: http://www.embase.com/search/results?subaction=viewrecord&from=export&id=L602431103 (accessed 24 June 2020).

30. Stroup D. F., Berlin J. A., Morton S. C., Olkin I., Williamson G. D., Rennie D. Meta-analysis of observational studies in epidemiology: a proposal for reporting. *JAMA [internet]* 2000; **283**: 2008–12. Available at: http://jama.jamanetwork.com/article.aspx?doi=10.1001/jama.283.15.2008 (accessed 24 June 2020).

31. Moher D., Liberati A., Tetzlaff J., Altman D. G. Preferred reporting items for systematic reviews and meta-analyses: the PRISMA statement. *PLOS Med* [internet] 2009; **6**: e1000097. Available at: https://dx.plos.org/10.1371/journal.pmed.1000097 (accessed 24 June 2020).

32. Bramer W. M., Rethlefsen M. L., Kleijnen J., Franco O. H. Optimal database combinations for literature searches in systematic reviews: a prospective exploratory study. *Syst Rev* 2017; **6**: 1–12.

33. Stang A. Critical evaluation of the Newcastle–Ottawa scale for the assessment of the quality of nonrandomized studies in meta-analyses. *Eur J Epidemiol* [internet] 2010; **25**: 603–5. Available at: http://link.springer.com/10.1007/s10654-010-9491-z (accessed 24 June 2020).

34. Lundin A., Hallgren M., Forsman M., Forsell Y. Comparison of DSM-5 classifications of alcohol use disorders with those of DSM-IV, DSM-III-R, and ICD-10 in a general population sample in Sweden. *J Stud Alcohol Drugs* [internet] 2015; **76**: 773–80. Available at: http://www.jsad.com/doi/10.15288/jsad.2015.76.773 (accessed 24 June 2020).

35. Viechtbauer W. Conducting meta-analyses in R with the metafor package. *J Stat Softw* [internet 2010]; **36**. Available at: http://www.jstatsoft.org/v36/i03/ (accessed 24 June 2020).

36. R Core Team. *R: A Language and Environment for Statistical Computing* [internet]. Vienna, Austria: R Foundation for Statistical Computing; 2019. Available at: https://www.r-project.org/ (accessed 24 June 2020).

37. Nyaga V. N., Arbyn M., Aerts M. Metaprop: a Stata command to perform meta-analysis of binomial data. *Arch Public Heal* [internet 2014; **72**: 39. Available at: http://archpublichealth.biomedcentral.com/articles/10.1186/2049-3258-72-39 (accessed 24 June 2020).

38. Borenstein M., Hedges L. V., Higgins J. P. T., Rothstein H. R. A basic introduction to fixed-effect and random-effects models for meta-analysis. *Res Synth Methods* [internet 2010; **1**: 97–111. Available at: http://doi.wiley.com/10.1002/jrsm.12 (accessed 24 June 2020).

39. Barendregt J. J., Doi S. A., Lee Y. Y., Norman R. E., Vos T. Meta-analysis of prevalence. *J Epidemiol Community Health* [internet] 2013; **67**: 974–8. Available at: http://jech.bmj.com/lookup/doi/10.1136/jech-2013-203104 (accessed 24 June 2020).

40. Kontopantelis E., Reeves D. Performance of statistical methods for meta-analysis when true study effects are non-normally distributed: a simulation study. *Stat Methods Med Res* [internet] 2012; **21**: 409–26. Available at: http://journals.sagepub.com/doi/10.1177/0962280210392008 (accessed 24 June 2020).

41. Higgins J. P. T., Thompson S. G., Deeks J. J., Altman D. G. Measuring inconsistency in meta-analyses. *BMJ* [internet] 2003; **327**: 557–60. Available at: http://www.bmj.com/cgi/doi/10.1136/bmj.327.7414.557 (accessed 24 June 2020).

42. Higgins J., Green S., Chapter 10: Analysing data and undertaking meta-analyses. editors. *Cochrane Handbook for Systematic Reviews of Interventions, version 5*. London, UK: The Cochrane Collaboration; 2011. https://training.cochrane.org/handbook/current/chapter-10#section-10-10-2 (accessed 24 June 2020).

43. Egger M., Smith G. D., Schneider M., Minder C. Bias in meta-analysis detected by a simple, graphical test. *Br Med J* [internet] 1997; **315**: 629–34. Available at: http://www.bmj.com/cgi/doi/10.1136/bmj.315.7109.629 (accessed 24 June 2020).

44. Robertson D. N., Palmer R. L. The prevalence and correlates of binge eating in a British community sample of women with a history of obesity. *Int J Eat Disord* [internet]. 1997; **22**: 323–7. Available at: http://www.embase.com/search/results?subaction=viewrecord&from=export&id=L27371990 (accessed 24 June 2020).

45. Root T. L., Pisetsky E. M., Thornton L., Lichtenstein P., Pedersen N. L., Bulik C. M. Patterns of co-morbidity of eating disorders and substance use in Swedish females. *Psychol Med* [internet] 2010; **40**: 105–15. Available at: http://www.embase.com/search/results?subaction=viewrecord&from=export&id=L355882623 (accessed 24 June 2020).

46. Specker S., de Zwaan M., Raymond N., Mitchell J. Psychopathology in subgroups of obese women with and without binge eating disorder. *Compr Psychiatry* [internet] 1994; **35**: 185–90. Available at: http://www.embase.com/search/results?subaction=viewrecord&from=export&id=L24154019 (accessed 24 June 2020).

47. Telch C. F., Stice E. Psychiatric comorbidity in women with binge eating disorder: prevalence rates from a non-treatment-seeking sample. *J Consult Clin Psychol* [internet] 1998; **66**: 768–76. Available at: http://www.embase.com/search/results?subaction=viewrecord&from=export&id=L28478344 (accessed 24 June 2020).

48. Udo T., Grilo C. M. Psychiatric and medical correlates of DSM-5 eating disorders in a nationally representative sample of adults in the United States. *Int J Eat Disord* [internet] 2019; **52**: 42–50. Available at: http://www.embase.com/search/results?subaction=viewrecord&from=export&id=L626169875 (accessed 24 June 2020).

49. Udo T., White M. A., Barnes R. D., Ivezaj V., Morgan P., Masheb R. M., *et al.* Psychosocial and metabolic function by smoking status in individuals with binge eating disorder and obesity. *Addict Behav* [internet] 2016; **53**: 46–52. Available at: http://www.embase.com/search/results?subaction=viewrecord&from=export&id=L606306175 (accessed 24 June 2020).

50. Ulfvebrand S., Birgegård A., Norring C., Högdahl L., von Hausswolff-Juhlin Y. Psychiatric comorbidity in women and men with eating disorders results from a large clinical database. *Psychiatry Res* [internet] 2015; **230**: 294–9. Available at: http://www.embase.com/search/results?subaction=viewrecord&from=export&id=L606678135 (accessed 24 June 2020).

51. Welch E., Jangmo A., Thornton L. M., Norring C., von Hausswolff-Juhlin Y., Herman B. K., *et al.* Treatment-seeking patients with binge-eating disorder in the Swedish national registers: clinical course and psychiatric comorbidity. *BMC Psychiatry* [internet 2016; **16**: 163. Available at: http://www.embase.com/search/results?subaction=viewrecord&from=export&id=L610472190 (accessed 24 June 2020).

52. Dohm F. A., Striegel-Moore R. H., Wilfley D. E., Pike K. M., Hook J., Fairburn C. G. Self-harm and substance use in a community sample of black and white women with binge eating disorder or bulimia nervosa. *Int J Eat Disord* [internet] 2002; **32**: 389–400. Available at: http://www.embase.com/search/results?subaction=viewrecord&from=export&id=L35253494 (accessed 24 June 2020).

53. Grilo C. M., White M. A., Barnes R. D., Masheb R. M. Psychiatric disorder co-morbidity and correlates in an ethnically diverse sample of obese patients with binge eating disorder in primary care settings. *Compr Psychiatry* [internet] 2013; **54**: 209–16. Available at: http://www.embase.com/search/

results?subaction=viewrecord&from=export&id=L52188027 (accessed 24 June 2020).

54. Grilo C. M., White M. A., Masheb R. M. DSM-IV psychiatric disorder comorbidity and its correlates in binge eating disorder. *Int J Eat Disord [internet]* 2009; **42**: 228–34. Available at: http://www.embase.com/search/results?subaction=viewrecord&from=export&id=L354607900 (accessed 24 June 2020).

55. Hudson J. I., Hiripi E., Pope H. G., Kessler R. C. The prevalence and correlates of eating disorders in the National Comorbidity Survey Replication. *Biol Psychiatry [internet]* 2007; **61**: 348–58. Available at: http://www.embase.com/search/results?subaction=viewrecord&from=export&id=L46108682 (accessed 24 June 2020).

56. Johnson J. G., Spitzer R. L., Williams J. B. Health problems, impairment and illnesses associated with bulimia nervosa and binge eating disorder among primary care and obstetric gynaecology patients. *Psychol Med [internet]* 2001; **31**: 1455–66. Available at: http://www.ncbi.nlm.nih.gov/pubmed/11722160 (accessed 24 June 2020).

57. Lee Y., Carmona N. E., Shekotikhina M., Subramaniapillai M., Mansur R. B., Cha D. S., *et al.* Is binge eating a cognitive disorder? Results from the international mood disorders collaborative project. *Ann Clin Psychiatry* [internet] 2018; **30**: 25–31. Available at: http://www.ncbi.nlm.nih.gov/pubmed/29373615 (accessed 24 June 2020).

58. Mitchell J. E., King W. C., Pories W., Wolfe B., Flum D. R., Spaniolas K., *et al.* Binge eating disorder and medical comorbidities in bariatric surgery candidates. *Int J Eat Disord [internet]* 2015; **48**: 471–6. Available at: http://www.embase.com/search/results?subaction=viewrecord&from=export&id=L604950099 (accessed 24 June 2020).

59. Pike K. M., Dohm F. A., Striegel-Moore R. H., Wilfley D. E., Fairburn C. G. A comparison of black and white women with binge eating disorder. *Am J Psychiatry [internet]* 2001; **158**: 1455–60. Available at: http://www.embase.com/search/results?subaction=viewrecord&from=export&id=L32847245 (accessed 24 June 2020).

60. Lau J., Ioannidis J. P. A., Terrin N., Schmid C. H., Olkin I. The case of the misleading funnel plot. *Br Med J* [internet] 2006; **333**: 597–600. Available at: http://www.bmj.com/lookup/doi/10.1136/bmj.333.7568.597 (accessed 24 June 2020).

61. Burrows T., Skinner J., McKenna R., Rollo M. Food addiction, binge eating disorder, and obesity: is there a relationship? *Behav Sci* 2017; **7**: 54.

62. Wolz I., Granero R., Fernández-Aranda F. A comprehensive model of food addiction in patients with binge-eating symptomatology: the essential role of negative urgency. *Compr Psychiatry* [internet] 2017; **74**: 118–24. Available at: http://www.ncbi.nlm.nih.gov/pubmed/28160693 (accessed 24 June 2020).

63. Brewerton TD, Brady K. The role of stress, trauma, and PTSD in the etiology and treatment of eating disorders, addictions, and substance use disorders. In: Brewerton TD, Dennis AB, editors. *Eating Disorders, Addictions and Substance Use Disorders [internet]*. Berlin, Heidelberg: Springer; 2014, pp. 379–404. Available at: http://link.springer.com/10.1007/978-3-642-45378-6_17 (accessed 24 June 2020).

64. Schag K., Schönleber J., Teufel M., Zipfel S., Giel K. E. Food-related impulsivity in obesity and binge eating disorder—a systematic review. *Obes Rev [internet]* 2013; **14**: 477–95. Available at: http://doi.wiley.com/10.1111/obr.12017 (accessed 24 June 2020).

65. Kwako L. E., Schwandt M. L., Ramchandani V. A., Diazgranados N., Koob G. F., Volkow N. D., *et al.* Neurofunctional domains derived from deep behavioral phenotyping in alcohol use disorder. *Am J Psychiatry* 2019; **176**: 744–53.

66. Nolen-Hoeksema S., Hilt L. Possible contributors to the gender differences in alcohol use and problems. *J Gen Psychol [internet]* 2006; **133**: 357–74. Available at: http://www.tandfonline.com/doi/abs/10.3200/GENP.133.4.357-374 (accessed 24 June 2020).

67. Hughes T. L., Wilsnack S. C., Kantor L. W. The influence of gender and sexual orientation on alcohol use and alcohol-related problems: toward a global perspective. *Alcohol Res* [internet] 2016; **38**: 121–32. Available at http://www.ncbi.nlm.nih.gov/pubmed/27159819 (accessed 24 June 2020).

68. Crow S. J., Peterson C. B., Levine A. S., Thuras P., Mitchell J. E. A survey of binge eating and obesity treatment practices among primary care providers. *Int J Eat Disord [internet]* 2004; **35**: 348–53. Available at: http://doi.wiley.com/10.1002/eat.10266 (accessed 24 June 2020).

69. Sheehan D. V., Herman B. K. The psychological and medical factors associated with untreated binge eating disorder. *Prim Care Companion CNS Disord* [internet 2015; http://www.psychiatrist.com/PCC/article/Pages/2015/v17n02/14r01732.aspx (accessed 24 June 2020).

70. Herman B. K., Safikhani S., Hengerer D., Atkins N., Kim A., Cassidy D., *et al.* The patient experience with DSM-5-defined binge eating disorder: characteristics, barriers to treatment, and implications for primary care physicians. *Postgrad Med [internet]* 2014; **126**: 52–63. Available at: http://www.tandfonline.com/doi/full/10.3810/pgm.2014.09.2800 (accessed 24 June 2020).

71. Grant B. F. Barriers to alcoholism treatment: reasons for not seeking treatment in a general population sample. *J Stud Alcohol [internet]* 1997; **58**: 365–71. Available at: http://www.jsad.com/doi/10.15288/jsa.1997.58.365 (accessed 24 June 2020).

72. Katikireddi S. V., Whitley E., Lewsey J., Gray L., Leyland A. H. Socioeconomic status as an effect modifier of alcohol consumption and harm: analysis of linked cohort data. *Lancet Public Heal [internet]* 2017; **2**: e267–e276. Available at: https://doi.org/10.1016/S2468-2667(17)30078-6 (accessed 24 June 2020).

73. Woodward M. Formulae for sample size, power and minimum detectable relative risk in medical studies. *Statistician* 1992; **41**: 185.

74. Rounsaville B. J., Bryant K., Babor T., Kranzler H., Kadden R. Cross system agreement for substance use disorders: DSM-III-R, DSM-IV and ICD-10. *Addiction [internet]* 1993; **88**: 337–48. Available at: http://doi.wiley.com/10.1111/j.1360-0443.1993.tb00821.x (accessed 24 June 2020).

75. Quintana D. S. From pre-registration to publication: a non-technical primer for conducting a meta-analysis to synthesize correlational data. *Front Psychol* [internet 2015; **6**. Available at: http://journal.frontiersin.org/Article/10.3389/fpsyg.2015.01549/abstract (accessed 24 June 2020).

Supporting Information

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