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

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Abstract

Background and Aims: Binge eating disorder (BED) is correlated with substance use. This study aimed to estimate the lifetime prevalence of alcohol use disorder (AUD) among individuals with non-compensatory binge eating and determine whether their lifetime 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 lifetime prevalence of AUD was conducted. The PRISMA protocol was followed. The protocol was registered on PROSPERO.

Setting: Studies originating in Canada, Sweden, the United Kingdom, and the United States.

Participants: 18 studies meeting the inclusion criteria were found, representing 69,233 individuals.

Measurements: Lifetime prevalence of AUD among individuals with binge eating disorder and their lifetime relative risk of AUD compared with individuals without this disorder.

Findings: The pooled lifetime prevalence of AUD in individuals with binge eating disorder was 19.9% (95% CI 13.7-27.9). The risk of lifetime AUD incidence among individuals with binge eating disorder was over 1.5 times higher than controls (RR 1.59, 95% CI 1.41-1.79). Lifetime AUD prevalence was higher in community samples than in clinical samples (27.45% vs. 14.45%, $p = 0.041$) and in studies with a lower proportion of women ($\beta = -2.27$, $p = 0.044$).

Conclusions: Lifetime alcohol use disorder appears to be more prevalent with binge eating disorder than among those without.

Keywords: Binge eating disorder, alcohol use disorder, alcohol abuse, alcohol dependence, meta-analysis, systematic review

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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 lifetime 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 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 “nonpurging 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 heterogenous 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

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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 lifetime 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 lifetime prevalence of AUD in studies

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investigating BED and related disorders. The primary aim of this systematic review and meta-analysis was to assess the lifetime prevalence of AUD among individuals who binge eat. The secondary aim of this study was to determine if the lifetime 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 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 (MK, AJ). Three electronic databases (PubMed, Embase, and Web of Science) were searched for articles published from January 1, 1966 until June 1, 2019 for adequate and efficient coverage(32). Additionally, 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 geographic 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 1.19.4.

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Study selection

Two reviewers (KB, MK) independently screened titles and abstracts. Criteria were broad 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 on 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, 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 lifetime 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 judgement); (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 (KB, AJ) according to a predefined coding protocol (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,

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each was recorded as a separate group. If necessary, one reviewer (KB) contacted the corresponding author to ask for additional data.

Two reviewers (KB, AJ) 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(Fig. S1)(33). It includes seven items grouped in 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 lifetime 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 sub-analysis 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 likely 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 the logit transformation, which handles small samples and extreme proportions more precisely than the direct proportions method(39).

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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 tau-squared 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-squared 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-squared of 30% to 60% indicates moderate heterogeneity, 50% to 90% indicates substantial heterogeneity, and I-squared 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) vs. 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-squared 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 funnel 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.

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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(21,29,52–59,44–51). Ten of those studies included a comparison group(21,48–50,52–55,57,59). Two studies(21,47) 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).

Lifetime prevalence of AUD among binge eating individuals

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

The overall pooled lifetime prevalence of AUD was 19.9% (95% CI 13.7-27.9, *p*-value = <0.0001). There was considerable heterogeneity present (I-squared = 96.6%; Q-test *p*-value = <0.0001).

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

A total of 11 samples, including data from 67,652 participants, reported lifetime 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 lifetime AUD among individuals engaging in binge eating was over 1.5 times higher in comparison to non-bingeing controls (RR 1.59, 95% CI 1.41-1.79). The heterogeneity was not statistically significant (I-squared = 26.4%; Q-test *p*-value = 0.19).

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Sensitivity analysis, moderator analysis, and sources of heterogeneity

Prevalence

There was a significant amount of heterogeneity among the included studies reporting prevalence: the I-squared statistic was 96.6% (95% CI 93.5-98.2); the Q-test *p*-value was <0.0001 and τ-squared 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% vs. 14.45%, *p*-value = 0.0412; Fig. S2). The amount of heterogeneity explained by this difference was 27.21% (the R-squared 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; 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-squared statistic was 26.4% (95% CI=0.00-74.9), the Q-test *p*-value was 0.19, and τ-squared 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 vs 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).

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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%.

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 (Table S3.). Four studies failed to reach this threshold and were judged to be at high risk of bias(48,50,52,54). 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, 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 (Fig. 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.

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Discussion

This study investigated the lifetime prevalence of AUD among individuals who binge eat and their relative risk of lifetime AUD compared to non-bingeing controls. There are two main findings. First, the overall lifetime prevalence of AUD among individuals who binge eat is 19.9% or just under one in five patients (Fig. 2). Second, individuals who binge eat are over 1.5 times more likely to be diagnosed with AUD in their lifetime 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 it was 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 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

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accounted for half of 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 a number of barriers to AUD treatment as well, 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 a higher socioeconomic status, a trait linked to 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 vs 30,372 in DSM-IV, and 36,027 in DSM-5) and resulting 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

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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 analytic approach using an a priori protocol, which was pre-registered with PROSPERO and conducted in accordance with PRISMA and MOOSE guidelines. Pre-registration helps 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 across 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. Second, all included studies came from developed countries, mainly from the US, so their pooled findings may not be applicable to other populations. Third, because the included studies spanned across 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. On the other hand, 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).

Fourth, 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. 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 likely moderators; however, these factors could

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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 less than 100 participants and half of all included studies reported data on less than 500 participants. Moreover, only ten 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 lifetime prevalence of AUD among individuals who binge eat. Findings indicate that lifetime 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 lifetime 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 on 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.

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Contributors

KB, MK, AJ, EMT, KK, AW, and MW contributed to conception and design of the study. KB, MK and AJ contributed to the acquisition, analysis and interpretation of data. KB, MK, AJ, and EMT wrote and edited the manuscript. KK, AW, and MW supervised the work and participated in revising the manuscript. All authors reviewed the manuscript and give approval of the final version.

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References

1. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders [Internet]. 5th ed. American Psychiatric Association; 2013. Available from: https://psychiatryonline.org/doi/book/10.1176/appi.books.9780890425596
2. Hilbert A. Binge-Eating Disorder. Psychiatr Clin North Am [Internet]. 2019 Mar [cited 2019 Jul 30];42(1):33–43. Available from: https://doi.org/10.1016/j.psc.2018.10.011
3. Guerdjikova AI, Mori N, Casuto LS, McElroy SL. Binge Eating Disorder. Psychiatr Clin North Am [Internet]. 2017 Jun [cited 2019 Aug 1];40(2):255–66. Available from: http://www.ncbi.nlm.nih.gov/pubmed/28477651
4. Stunkard AJ. Eating patterns and obesity. Psychiatr Q [Internet]. 1959 Jun;33(2):284–95. Available from: http://link.springer.com/10.1007/BF01575455
5. Spitzer RL, Devlin MJ, Walsh BT, Hasin D, Wing R, Marcus MD, et al. Binge eating disorder: To be or not to be in DSM- IV. Int J Eat Disord. 1991;10(6):627–9.
6. Spitzer RL, McCann UD, Agras WS. Nonpurging bulimia nervosa and binge eating disorder [17]. Am J Psychiatry [Internet]. 1991;148(8):1097–8. Available from: http://www.embase.com/search/results?subaction=viewrecord&from=export&id=L21260940
7. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders. 4th ed. 1994.
8. Wilfley DE, Citrome L, Herman BK. Characteristics of binge eating disorder in relation to diagnostic criteria. Neuropsychiatr Dis Treat. 2016;12:2213–23.
9. Striegel-Moore RH, Dohm FA, Solomon EE, Fairburn CG, Pike KM, Wilfley DE. Subthreshold binge eating disorder. Int J Eat Disord [Internet]. 2000 Apr;27(3):270–8. Available from: http://doi.wiley.com/10.1002/%28SICI%291098-108X%28200004%2927%3A3%3C270%3A%3AAID-EAT3%3E3.0.CO%3B2-1
10. Devlin MJ, Goldfein JA, Dobrow I. What is this thing called BED? Current status of binge eating disorder nosology. Int J Eat Disord. 2003;34(SUPPL.):2–18.

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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 - Stud Anorexia, Bulim Obes [Internet]. 2018 Dec 27;23(6):861–9. Available from: http://link.springer.com/10.1007/s40519-018-0603-1
12. Kober H, Boswell RG. Potential psychological & neural mechanisms in binge eating disorder: Implications for treatment. Vol. 60, Clinical Psychology Review. Elsevier Inc.; 2018. p. 32–44.
13. Koob GF, Le Moal M. Neurobiological mechanisms for opponent motivational processes in addiction. Philos Trans R Soc B Biol Sci. 2008;363(1507):3113–23.
14. Berridge KC, Robinson TE. Liking, wanting, and the incentive-sensitization theory of addiction. Am Psychol [Internet]. 2016 Nov;71(8):670–9. Available from: http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&dopt=Citation&list_uids=17375140%0Ahttp://www.ncbi.nlm.nih.gov/pubmed/16167185%0Ahttps://www.ncbi.nlm.nih.gov/pmc/articles/PMC1405013/pdf/amjph00203-0102.pdf%0Ahttps://doi.org/10.1080/08
15. Kober H. Emotion regulation in substance use disorders. Handbook of emotion regulation, 2nd ed. New York, NY, US: Guilford Press; 2014. p. 428–46.
16. Koob GF. 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 Dec [cited 2019 Jul 31];39(12):2471–9. Available from: http://www.ncbi.nlm.nih.gov/pubmed/26613633
18. Killeen T, Brewerton TD, Campbell A, Cohen LR, Hien DA. 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(6):547–52.
19. Wolfe WL, Maisto SA. The relationship between eating disorders and substance use:

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Author Manuscript

Moving beyond co-prevalence research. Clin Psychol Rev. 2000;20(5):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 [cited 2019 Jul 31];10(4):133–40. Available from: http://www.ncbi.nlm.nih.gov/pubmed/17533558

21. Javaras KN, Pope HG, Lalonde JK, Roberts JL, Nillni YI, Laird NM, et al. Co-occurrence of binge eating disorder with psychiatric and medical disorders. J Clin Psychiatry [Internet]. 2008 Feb;69(2):266–73. Available from: http://www.embase.com/search/results?subaction=viewrecord&from=export&id=L351469270

22. Kessler RM, Hutson PH, Herman BK, Potenza MN. The neurobiological basis of binge-eating disorder. Neurosci Biobehav Rev [Internet]. 2016 Apr;63:223–38. Available from: http://www.embase.com/search/results?subaction=viewrecord&from=export&id=L608598867

23. Fritz M, Klawonn AM, Zahr NM. Neuroimaging in alcohol use disorder: From mouse to man. J Neurosci Res. 2019;(March):1–19.

24. Lee JE, Namkoong K, Jung YC. Impaired prefrontal cognitive control over interference by food images in binge-eating disorder and bulimia nervosa. Neurosci Lett [Internet]. 2017;651:95–101. Available from: http://dx.doi.org/10.1016/j.neulet.2017.04.054

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(1).

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

27. Bahji A, Mazhar MN, Hudson CC, Nadkarni P, MacNeil BA, Hawken E. Prevalence of substance use disorder comorbidity among individuals with eating disorders: A systematic review and meta-analysis. Psychiatry Res. 2019 Mar;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

This article is protected by copyright. All rights reserved.

Author Manuscript

Jul;17(4):243–59. Available from: http://www.embase.com/search/results?subaction=viewrecord&from=export&id=L355695295

29. Becker DF, Grilo CM. 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 Aug;79(2):159–64. Available from: http://www.embase.com/search/results?subaction=viewrecord&from=export&id=L602431103

30. Stroup DF, Berlin JA, Morton SC, Olkin I, Williamson GD, Rennie D, et al. Meta-analysis of observational studies in epidemiology: A proposal for reporting. J Am Med Assoc [Internet]. 2000 Apr 19;283(15):2008–12. Available from: http://jama.jamanetwork.com/article.aspx?doi=10.1001/jama.283.15.2008

31. Moher D, Liberati A, Tetzlaff J, Altman DG. Preferred Reporting Items for Systematic Reviews and Meta-Analyses: The PRISMA Statement. PLoS Med [Internet]. 2009 Jul 21;6(7):e1000097. Available from: https://dx.plos.org/10.1371/journal.pmed.1000097

32. Bramer WM, Rethlefsen ML, Kleijnen J, Franco OH. Optimal database combinations for literature searches in systematic reviews: A prospective exploratory study. Syst Rev. 2017;6(1):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 Sep 22;25(9):603–5. Available from: http://link.springer.com/10.1007/s10654-010-9491-z

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 Sep;76(5):773–80. Available from: http://www.jsad.com/doi/10.15288/jsad.2015.76.773

35. Viechtbauer W. Conducting Meta-Analyses in R with the metafor Package. J Stat Softw [Internet]. 2010;36(3). Available from: http://www.jstatsoft.org/v36/i03/

This article is protected by copyright. All rights reserved.

Author Manuscript

36. R Core Team. R: A Language and Environment for Statistical Computing [Internet]. Vienna, Austria: R Foundation for Statistical Computing; 2019. Available from: https://www.r-project.org/
37. Nyaga VN, Arbyn M, Aerts M. Metaprop: a Stata command to perform meta-analysis of binomial data. Arch Public Heal [Internet]. 2014 Dec 10;72(1):39. Available from: http://archpublichealth.biomedcentral.com/articles/10.1186/2049-3258-72-39
38. Borenstein M, Hedges L V., Higgins JPT, Rothstein HR. A basic introduction to fixed-effect and random-effects models for meta-analysis. Res Synth Methods [Internet]. 2010 Apr;1(2):97–111. Available from: http://doi.wiley.com/10.1002/jrsm.12
39. Barendregt JJ, Doi SA, Lee YY, Norman RE, Vos T. Meta-analysis of prevalence. J Epidemiol Community Health [Internet]. 2013 Nov;67(11):974–8. Available from: http://jech.bmj.com/lookup/doi/10.1136/jech-2013-203104
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 Aug 9;21(4):409–26. Available from: http://journals.sagepub.com/doi/10.1177/0962280210392008
41. Higgins JPT, Thompson SG, Deeks JJ, Altman DG. Measuring inconsistency in meta-analyses. BMJ [Internet]. 2003 Sep 6;327(7414):557–60. Available from: http://www.bmj.com/cgi/doi/10.1136/bmj.327.7414.557
42. Higgins J, Green S, editors. Cochrane Handbook for Systematic Reviews of Interventions. Version 5. The Cochrane Collaboration; 2011.
43. Egger M, Smith GD, Schneider M, Minder C. Bias in meta-analysis detected by a simple, graphical test. BMJ [Internet]. 1997 Sep 13;315(7109):629–34. Available from: http://www.bmj.com/cgi/doi/10.1136/bmj.315.7109.629
44. Dohm FA, Striegel-Moore RH, Wilfley DE, Pike KM, Hook J, Fairburn CG. 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 Dec;32(4):389–400. Available from:

This article is protected by copyright. All rights reserved.

Author Manuscript

http://www.embase.com/search/results?subaction=viewrecord&from=export&id=L352 53494

45. Grilo CM, White MA, Barnes RD, Masheb RM. 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 Apr;54(3):209–16. Available from: http://www.embase.com/search/results?subaction=viewrecord&from=export&id=L521 88027

46. Grilo CM, White MA, Masheb RM. DSM-IV psychiatric disorder comorbidity and its correlates in binge eating disorder. Int J Eat Disord [Internet]. 2009 Apr;42(3):228–34. Available from: http://www.embase.com/search/results?subaction=viewrecord&from=export&id=L354 607900

47. Hudson JI, Hiripi E, Pope HG, Kessler RC. The Prevalence and Correlates of Eating Disorders in the National Comorbidity Survey Replication. Biol Psychiatry [Internet]. 2007 Feb;61(3):348–58. Available from: http://www.embase.com/search/results?subaction=viewrecord&from=export&id=L461 08682

48. Johnson JG, Spitzer RL, Williams JB. Health problems, impairment and illnesses associated with bulimia nervosa and binge eating disorder among primary care and obstetric gynaecology patients. Psychol Med [Internet]. 2001 Nov;31(8):1455–66. Available from: http://www.ncbi.nlm.nih.gov/pubmed/11722160

49. Lee Y, Carmona NE, Shekotikhina M, Subramaniapillai M, Mansur RB, Cha DS, et al. Is binge eating a cognitive disorder? Results from the International Mood Disorders Collaborative Project. Ann Clin Psychiatry [Internet]. 2018 Feb;30(1):25–31. Available from: http://www.ncbi.nlm.nih.gov/pubmed/29373615

50. Mitchell JE, King WC, Pories W, Wolfe B, Flum DR, Spaniolas K, et al. Binge eating disorder and medical comorbidities in bariatric surgery candidates. Int J Eat Disord

This article is protected by copyright. All rights reserved.

Author Manuscript

[Internet]. 2015;48(5):471–6. Available from: http://www.embase.com/search/results?subaction=viewrecord&from=export&id=L604950099

51. Pike KM, Dohm FA, Striegel-Moore RH, Wilfley DE, Fairburn CG. A comparison of black and white women with binge eating disorder. Am J Psychiatry [Internet]. 2001;158(9):1455–60. Available from: http://www.embase.com/search/results?subaction=viewrecord&from=export&id=L32847245

52. Robertson DN, Palmer RL. 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 Nov;22(3):323–7. Available from: http://www.embase.com/search/results?subaction=viewrecord&from=export&id=L27371990

53. Root TL, Pisetsky EM, Thornton L, Lichtenstein P, Pedersen NL, Bulik CM. Patterns of co-morbidity of eating disorders and substance use in Swedish females. Psychol Med [Internet]. 2010 Jan;40(1):105–15. Available from: http://www.embase.com/search/results?subaction=viewrecord&from=export&id=L355882623

54. 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(3):185–90. Available from: http://www.embase.com/search/results?subaction=viewrecord&from=export&id=L24154019

55. Telch CF, 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(5):768–76. Available from: http://www.embase.com/search/results?subaction=viewrecord&from=export&id=L28478344

This article is protected by copyright. All rights reserved.

Author Manuscript

56. Udo T, Grilo CM. 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 Jan;52(1):42–50. Available from: http://www.embase.com/search/results?subaction=viewrecord&from=export&id=L626169875

57. Udo T, White MA, Barnes RD, Ivezaj V, Morgan P, Masheb RM, et al. Psychosocial and metabolic function by smoking status in individuals with binge eating disorder and obesity. Addict Behav [Internet]. 2016 Feb;53:46–52. Available from: http://www.embase.com/search/results?subaction=viewrecord&from=export&id=L606306175

58. 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 Dec;230(2):294–9. Available from: http://www.embase.com/search/results?subaction=viewrecord&from=export&id=L606678135

59. Welch E, Jangmo A, Thornton LM, Norring C, von Hausswolff-Juhlin Y, Herman BK, et al. Treatment-seeking patients with binge-eating disorder in the Swedish national registers: Clinical course and psychiatric comorbidity. BMC Psychiatry [Internet]. 2016 May;16(1):163. Available from: http://www.embase.com/search/results?subaction=viewrecord&from=export&id=L610472190

60. Lau J, Ioannidis JPA, Terrin N, Schmid CH, Olkin I. The case of the misleading funnel plot. BMJ [Internet]. 2006 Sep 16;333(7568):597–600. Available from: http://www.bmj.com/lookup/doi/10.1136/bmj.333.7568.597

61. Burrows T, Skinner J, McKenna R, Rollo M. Food addiction, binge eating disorder, and obesity: Is there a relationship? Behav Sci (Basel). 2017 Sep;7(3):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.

This article is protected by copyright. All rights reserved.

Author Manuscript

Compr Psychiatry [Internet]. 2017 [cited 2019 Jul 31];74:118–24. Available from: http://www.ncbi.nlm.nih.gov/pubmed/28160693

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: Eating Disorders, Addictions and Substance Use Disorders [Internet]. Berlin, Heidelberg: Springer Berlin Heidelberg; 2014 [cited 2019 Oct 25]. p. 379–404. Available from: http://link.springer.com/10.1007/978-3-642-45378-6_17

64. Schag K, Schönleber J, Teufel M, Zipfel S, Giel KE. Food-related impulsivity in obesity and Binge Eating Disorder - a systematic review. Obes Rev [Internet]. 2013 Jun;14(6):477–95. Available from: http://doi.wiley.com/10.1111/obr.12017

65. Kwako LE, Schwandt ML, Ramchandani VA, Diazgranados N, Koob GF, Volkow ND, et al. Neurofunctional domains derived from deep behavioral phenotyping in alcohol use disorder. Am J Psychiatry. 2019;176(9):744–53.

66. Nolen-Hoeksema S, Hilt L. Possible Contributors to the Gender Differences in Alcohol Use and Problems. J Gen Psychol [Internet]. 2006 Oct;133(4):357–74. Available from: http://www.tandfonline.com/doi/abs/10.3200/GENP.133.4.357-374

67. Hughes TL, Wilsnack SC, Kantor LW. The Influence of Gender and Sexual Orientation on Alcohol Use and Alcohol-Related Problems: Toward a Global Perspective. Alcohol Res [Internet]. 2016;38(1):121–32. Available from: http://www.ncbi.nlm.nih.gov/pubmed/27159819

68. Crow SJ, Peterson CB, Levine AS, Thuras P, Mitchell JE. A survey of binge eating and obesity treatment practices among primary care providers. Int J Eat Disord [Internet]. 2004 Apr;35(3):348–53. Available from: http://doi.wiley.com/10.1002/eat.10266

69. Sheehan D V., Herman BK. The Psychological and Medical Factors Associated With Untreated Binge Eating Disorder. Prim Care Companion CNS Disord [Internet]. 2015 Apr 23; Available from: http://www.psychiatrist.com/PCC/article/Pages/2015/v17n02/14r01732.aspx

This article is protected by copyright. All rights reserved.

Author Manuscript

70. Herman BK, 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 Sep 13;126(5):52–63. Available from: http://www.tandfonline.com/doi/full/10.3810/pgm.2014.09.2800

71. Grant BF. Barriers to alcoholism treatment: reasons for not seeking treatment in a general population sample. J Stud Alcohol [Internet]. 1997 Jul;58(4):365–71. Available from: http://www.jsad.com/doi/10.15288/jsa.1997.58.365

72. Katikireddi SV, Whitley E, Lewsey J, Gray L, Leyland AH. Socioeconomic status as an effect modifier of alcohol consumption and harm: analysis of linked cohort data. Lancet Public Heal [Internet]. 2017;2(6):e267–76. Available from: http://dx.doi.org/10.1016/S2468-2667(17)30078-6

73. Woodward M. Formulae for Sample Size, Power and Minimum Detectable Relative Risk in Medical Studies. Stat. 1992;41(2):185.

74. Rounsaville BJ, 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 Mar;88(3):337–48. Available from: http://doi.wiley.com/10.1111/j.1360-0443.1993.tb00821.x

75. Quintana DS. From pre-registration to publication: a non-technical primer for conducting a meta-analysis to synthesize correlational data. Front Psychol [Internet]. 2015 Oct 8;6. Available from: http://journal.frontiersin.org/Article/10.3389/fpsyg.2015.01549/abstract

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Figure Legend

Figure 1. Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) flow diagram describing study selection.

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

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.

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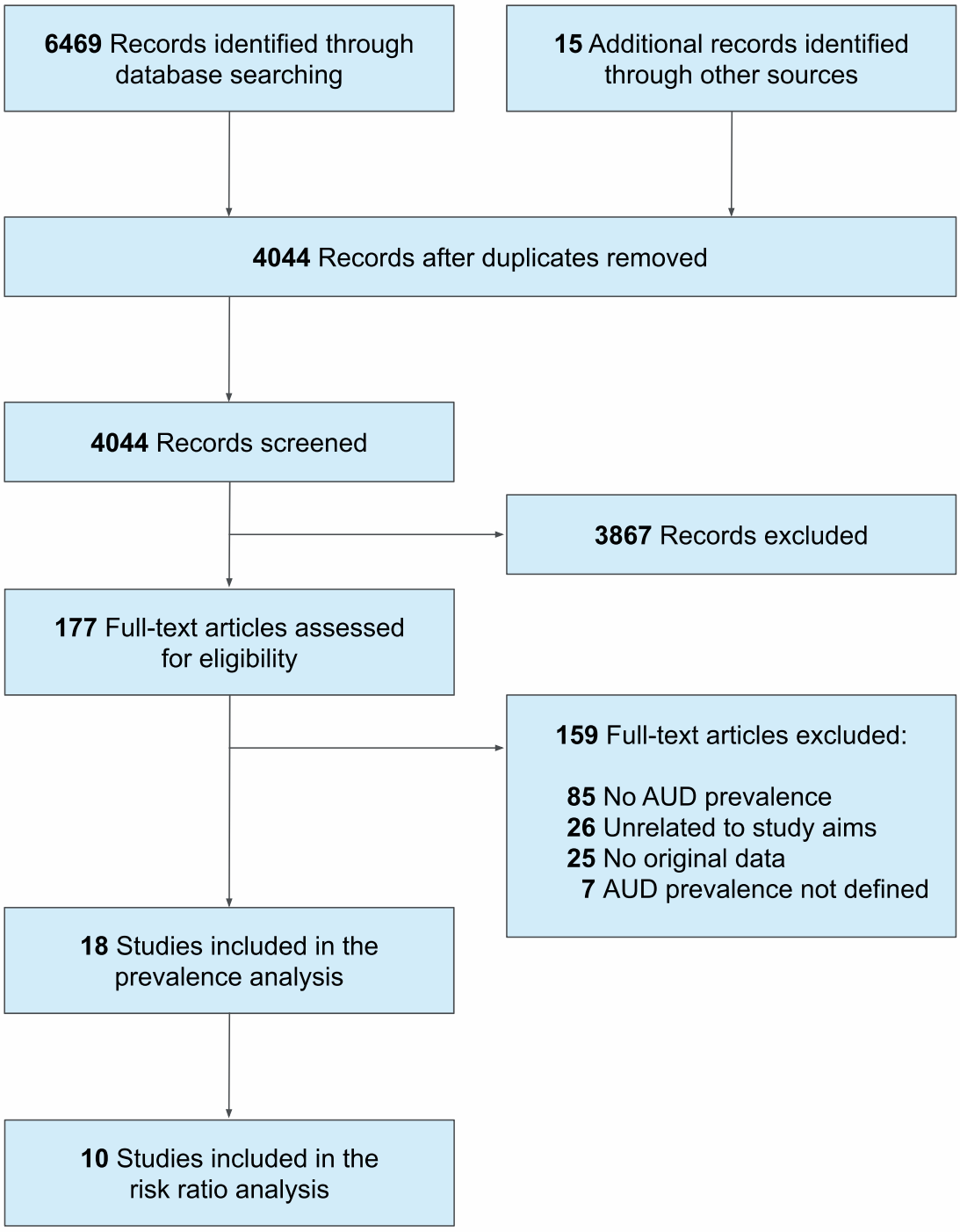
Figure 1. PRISMA flowchart



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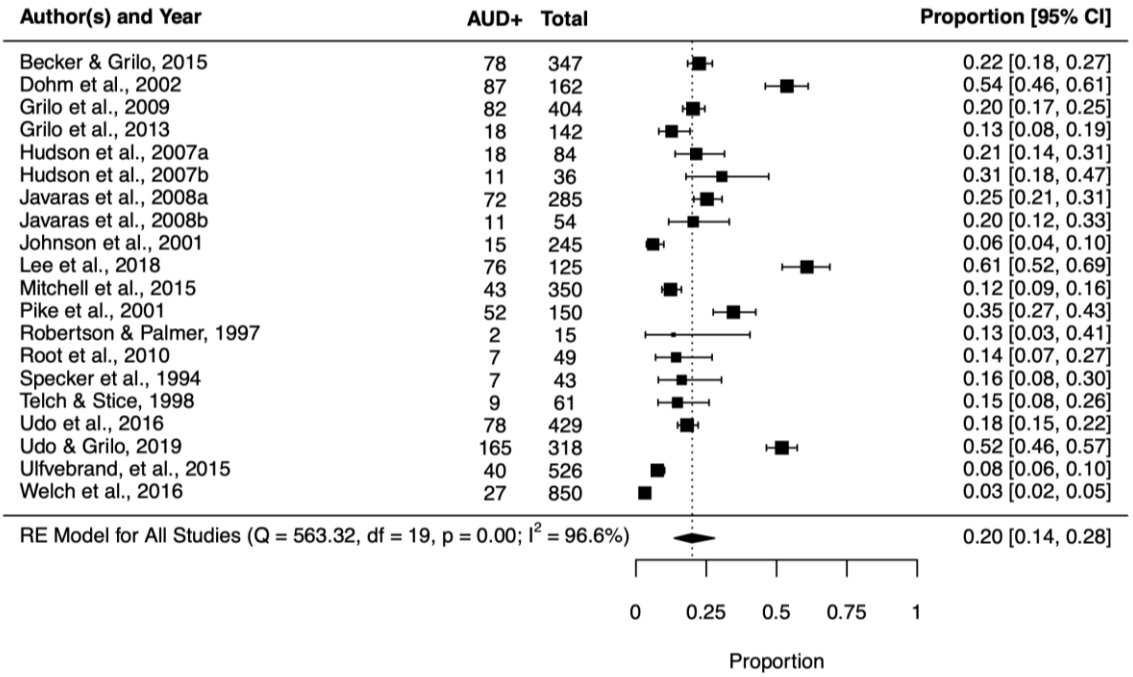
1 Figure 2. AUD prevalence



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1 Figure 3. AUD relative risk

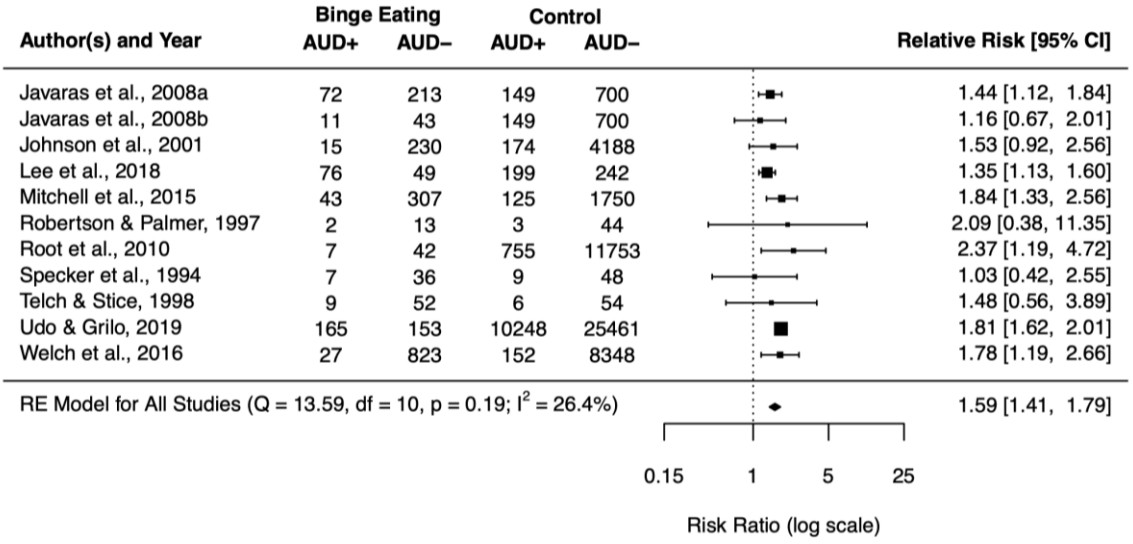

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Table Legend

Table 1. Characteristics of included studies. CLIN – Clinical; COMM – Community; NA – not applicable; NR – not reported.

Table 2. Subgroup analysis of categorical moderators for samples describing prevalence. CI – confidence interval; NA – not applicable; Own criteria – AUD criteria used by study authors that did not use the DSM (e.g. Alcohol Use Disorders Identification Test).

Table 3. Subgroup analysis of categorical moderators for samples describing relative risk. CI – confidence interval; NA – not applicable; Own criteria – AUD criteria used by study authors that did not use the DSM (e.g. Alcohol Use Disorders Identification Test).

Table 4. Meta-regression of continuous moderators. CI – confidence interval; NA – not applicable.

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1 Table 1. Description of included studies

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First author and year	Country	Number of participants who binge eat	Number of controls	Type of sample	Percent of women	Binge eating criteria	AUD criteria	Basis of AUD diagnosis	Percent of AUD in BE group	Percent of AUD in controls	Percent of non-white participants	Mean age of BE group in years	Mean age of controls in years
Becker & Grilo, 2015	US	347	NA	CLIN	85%	DSM-IV	DSM-IV	Clinical interview	22.48%	NA	19.02%	44.7	NA
Dohm et al., 2002	US	162	NA	COMM	100%	DSM-IV	DSM-IV	Clinical interview	53.70%	NA	43.83%	NR	NA
Grilo et al., 2009	US	404	NA	CLIN	77%	DSM-IV	DSM-IV	Clinical interview	20.30%	NA	18.81%	44.9	NA
Grilo et al., 2013	US	142	NA	CLIN	74%	DSM-IV	DSM-IV	Clinical interview	12.68%	NA	57.04%	43.6	NA
Hudson et al., 2007a	US	84	NA	COMM	73%	DSM-IV	DSM-IV	Clinical interview	21.43%	NA	NR	NR	NA
Hudson et al., 2007b	US	36	NA	COMM	31%	Own criteria	DSM-IV	Clinical interview	30.56%	NA	NR	NR	NA
Javaras et al., 2008a	US	285	849	COMM	68%	DSM-IV	DSM-IV	Clinical interview	25.26%	17.55%	NR	46.4	48.0
Javaras et al., 2008b	US	54	849	COMM	67%	Own criteria	DSM-IV	Clinical interview	20.37%	17.55%	NR	46.2	48.0

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Johnson et al., 2001	US	245	4362	CLIN	100%	DSM-IV	DSM-IV	Self-report	6.12%	3.99%	NR	NR	NR
Lee et al., 2018	US, 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	US	350	1875	CLIN	77%	DSM-5	Own criteria	Self-report	12.29%	6.67%	13.03%	NR	NR
Pike et al., 2001	US	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	12508	COMM	100%	DSM-IV	DSM-IV	Clinical interview	14.29%	6.04%	NR	31.9	33.7
Specker et al., 1994	US	43	57	CLIN	100%	DSM-IV	DSM-III	Clinical interview	16.28%	15.79%	NR	NR	NR
Telch & Stice, 1998	US	61	60	COMM	100%	DSM-IV	DSM-III	Clinical interview	14.75%	10.00%	26.45%	43.5	5.0
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	US	318	35709	COMM	57%	DSM-5	DSM-5	Clinical interview	51.89%	28.70%	47.1%	NR	NR

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Ulfvebrand, 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

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1 Table 2. Prevalence subgroup analysis

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Moderator	Prevalence [%]	95% CI [%]	I-squared statistic	Test of moderators *p*-value	R-squared 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%		

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1 Table 3. Relative risk subgroup analysis

Moderator	Relative risk	95% CI	I-squared statistic	Test of moderators *p*-value	R-squared 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%		

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1 Table 4. Meta-regression

Prevalence				
Moderator	**Slope (β)**	**95% CI**	**Test of moderators *p*-value**	**R-squared 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-squared 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%

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