

Review

Prevalence and Etiology of Eating Disorders in Polycystic Ovary Syndrome: A Scoping Review



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ABSTRACT

Polycystic ovary syndrome (PCOS) is the most common endocrine-metabolic disorder affecting females across the lifespan. Eating disorders (EDs) are psychiatric conditions that may impact the development of PCOS and comorbidities including obesity, metabolic syndrome, and type 2 diabetes. The aim of this scoping review was to determine the prevalence of EDs and disordered eating, and to review the etiology of EDs in PCOS. The review was conducted using search terms addressing PCOS, EDs, and disordered eating in databases, including PubMed, Scopus, PsycINFO, and CINAHL. Structured interviews, self-administered questionnaires, chart review, or self-reported diagnosis were used to identify EDs in 38 studies included in the review. The prevalence of any ED in those with PCOS ranged from 0% to 62%. Those with PCOS were 3–6-fold more likely to have an ED and higher odds ratios (ORs) of an elevated ED score compared with controls. In those with PCOS, 30% had a higher OR of bulimia nervosa and binge ED was 3-fold higher compared with controls. Studies were limited on anorexia nervosa and other specified feeding or ED (such as night eating syndrome) and these were not reported to be higher in PCOS. To our knowledge, no studies reported on avoidant/restrictive food intake disorder, rumination disorder, or pica in PCOS. Studies showed strong associations between overweight, body dissatisfaction, and disordered eating in PCOS. The etiologic development of EDs in PCOS remains unclear; however, psychological, metabolic, hypothalamic, and genetic factors are implicated. The prevalence of any ED in PCOS varied because of the use of different diagnostic and screening tools. Screening of all individuals with PCOS for EDs is recommended and high-quality studies on the prevalence, pathogenesis of specific EDs, relationship to comorbidities, and effective interventions to treat ED in those with PCOS are needed.

Keywords: polycystic ovary syndrome, hyperandrogenism, insulin resistance, eating disorder, binge eating disorder, anorexia nervosa, bulimia nervosa

Statement of Significance

Individuals with polycystic ovary syndrome (PCOS) have a high risk of eating disorders (EDs), in particular binge ED and bulimia nervosa. Further studies are required to explore the range of EDs in PCOS, particularly atypical anorexia nervosa. Early identification and management of an ED may prevent significant negative impacts on quality of life and health in those with PCOS.

Abbreviations: AGRP, agouti-related peptide; AN, anorexia nervosa; ARFID, avoidant/restrictive food intake disorder; BED, binge eating disorder; BITE, Bulimic Investigatory Test, Edinburgh; BN, bulimia nervosa; CART, cocaine amphetamine-regulated transcript; CCK, cholecystokinin; CI, confidence interval; DE, disordered eating; DSM, Diagnostic and Statistical Manual of Mental Disorders; EAT, Eating Attitude Test; EAT-26, 26-question Eating Attitude Test; ED, eating disorder; EDE, Eating Disorder Examination; EDE-Q, Eating Disorder Examination Questionnaire; EDNOS, eating disorder not otherwise specified; GLP-1, glucagon-like peptide-1; IR, insulin resistance; LH, luteinizing hormone; NES, night eating syndrome; NPY, neuropeptide Y; OR, odds ratio; OSFED, other specified feeding and eating disorders; PCOS, polycystic ovary syndrome; POMC, pro-opiomelanocortin; RD, rumination disorder; SHBG, sex hormone-binding globulin; TFEQ, Three-Factor Eating Questionnaire.

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Introduction

Polycystic ovary syndrome (PCOS) is the most common endocrine-metabolic disorder affecting 6%–20% of females and impacting health and quality of life across the lifespan [1–6]. The diagnostic criteria for PCOS includes the presence of 2 of the following: biochemical or clinical hyperandrogenism, menstrual-ovulatory dysfunction, and/or polycystic ovary morphology, with the exclusion of other endocrine disorders [7, 8]. PCOS is highly associated with obesity, metabolic syndrome, diabetes, cardiovascular disease, and an increased incidence of infertility and pregnancy complications [8–16]. PCOS is also strongly associated with psychiatric disorders, including depression and anxiety [8,16–20].

Eating disorders (EDs) are psychiatric illnesses that have been reported in PCOS [8,18,21,16,22]. EDs and disordered eating (DE) have been associated with a higher risk of depression, anxiety, and body dissatisfaction in those with PCOS [23–25]. EDs in PCOS may have pathophysiologic causes related to dysregulation of the hypothalamic-neuroregulation of appetite and satiety, associated with metabolic and endocrine factors [21,26, 27]. There remain limited studies on the etiologic causes and the different types of EDs in PCOS [28–30].

EDs are clinically diagnosed using structured interviews according to the Diagnostic and Statistical Manual of Mental Disorders (DSM) by the American Psychiatric Association [31].

These disorders are often characterized by disruptions in behaviors, thoughts, and attitudes toward food, eating, and body weight or shape [31,32]. The DSM-5 defines diagnostic criteria for anorexia nervosa (AN), bulimia nervosa (BN), binge ED (BED), other specified feeding or ED (OSFED), avoidant/restrictive feeding or ED (ARFID), pica, and rumination disorder (RD) (Table 1). DE refers to eating related symptoms with behavioral (e.g. bingeing and food restriction), cognitive (e.g. excessive dietary restraint and poor body image), and emotional (e.g. shame and anxiety) features. Both EDs and DE can have detrimental effects on quality of life and can lead to significant medical and psychosocial consequences [31].

The global prevalence of EDs has increased from 3.5% in 2000–2006 to 4.9% in 2007–2012, and to 7.8% in 2013–2018 [33]. The most common is BED, a disorder characterized by rapid consumption of large amounts of food without purging. It affects 1%–4% of adults [34–38] and 1%–2% of children and adolescents [39]. BN is characterized by rapid consumption of large amounts of food with purging and affects 0.41%–1.5% of adults [33–35,37]. AN is a disorder characterized by fear of gaining weight, behaviors that interfere with maintaining weight, and severe body dysmorphia, and affects 0.2%–3% of adults [31,33,34,37,40,41]. The other recognized EDs are less common, and their prevalence is largely uncharacterized among the general population [31,33,42,43] (Table 2).

TABLE 1

Criteria for feeding and eating disorders from the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5)

Diagnosis	DSM-5 diagnostic criteria
Anorexia Nervosa (AN)	Restriction of energy intake leading to a significantly low body weight, intense fear of gaining weight, disturbance in the way in which one's body weight is experienced, denial of the seriousness of the current low body weight. Two subtypes: restricting type: (no binge eating or purging in the last 3 mo) and binge eating/purging type (binge eating or purging in the last 3 mo)
Bulimia Nervosa (BN)	Recurrent episodes of binge eating (eating a large amount of food in a discrete period of time with sense of lack of control) with compensatory behavior to prevent weight gain (e.g., vomiting, laxatives, diuretics, fasting, and excessive exercise) at least once a week for 3 mo. Self-evaluation is unduly influenced by body weight
Binge Eating Disorder (BED)	Recurrent episodes of binge eating that are associated with 3 (or more) of: eating rapidly, eating until uncomfortably full, eating large amounts when not hungry, eating alone because of being embarrassed, and guilt/disgust after overeating. Occurs at least once a week for 3 mo with absence of compensatory behaviors
Avoidant/Restrictive Food Intake Disorder (ARFID)	A feeding disturbance (e.g., lack of interest in food; sensory avoidance of food; and concern about consequences of eating) as manifested by failure to meet nutritional needs associated with 1 (or more) of: significant weight loss or faltering growth, nutritional deficiency, dependence on nutritional supplements, and interference with psychosocial functioning. Not better explained by lack of available food or by cultural practice. No evidence of body weight disturbance. Not better explained by another medical condition or if so, is severe enough to warrant additional clinical attention
Rumination disorder	Repeated regurgitation of food for a period of ≥ 1 mo. Regurgitated food may be rechewed, reswallowed, or spit out. Not better explained by another medical condition or if so, is severe enough to warrant additional clinical attention
Pica	Persistent eating of non-nutritive, nonfood substances for ≥ 1 mo that is inappropriate to the developmental level of the individual and is not part of a cultural or social practice. If occurring in the context of another condition, it is sufficiently severe to warrant additional clinical attention
Other Specified Feeding or Eating Disorder (OSFED)	Examples of presentations that can be specified using OSFED: <ul style="list-style-type: none"> • Atypical anorexia nervosa: all AN criteria are met, except despite significant weight loss, the individual's weight is within or above the normal range • Bulimia nervosa (of low-frequency and/or limited duration): all of the criteria for BN are met, except that episodes occurs at a lower frequency and/or for < 3 mo • Binge eating disorder (BED) (of low-frequency and/or limited duration): all of the criteria for BED are met, except at a lower frequency and/or for < 3 mo • Purging disorder: recurrent purging behavior to influence weight or shape in the absence of binge eating • Night eating syndrome (NES): recurrent episodes of night eating: eating after awakening from sleep, or excessive eating after the evening meal that causes significant distress/impairment. The behavior is not better explained by environmental influences, social norms, or by another disorder (e.g., BED)

Table adapted from the Diagnostic and Statistical Manual of Mental Disorders, 5th Edition (DSM-5) [31].

TABLE 2

Prevalence of EDs in PCOS and in females in the global general population

Eating disorder	PCOS (%)	General population (%)
Any eating disorder	0–62	3.61–7.8 [33,37]
Binge eating disorder	1.4–42	1.03–3.8 [33,34,37,38]
Bulimia nervosa	0–12	0.41–1.5 [33,34,37]
Anorexia nervosa	0–3.5	0.23–2.8 [33,37,41,43]
Other specified feeding or eating disorder		
Atypical anorexia nervosa	0	0.35 [37]
Low-frequency bulimia nervosa	9.4	0.44 [37]
Low-frequency binge eating disorder	16	0.38 [37]
Purging disorder	1.2	0.23–0.60 [34,37]
Night eating syndrome	12.93	1.6 [42]
Avoidant/restrictive food intake disorder	N/A	Unclear [31]
Rumination disorder	N/A	Unclear [31]
Pica	N/A	Unclear [31]

Abbreviations: AN, anorexia nervosa; ARFID, avoidant/restrictive food intake disorder; BED, binge eating disorder; BN, bulimia nervosa; ED, eating disorder; EDE, Eating Disorder Examination; N/A, not applicable; NES, night eating syndrome; OSFED, other specified feeding or eating disorder; PCOS, polycystic ovary syndrome; RD, rumination disorder.

DE is a descriptive term used to describe a range of irregular eating behaviors such as body dissatisfaction, preoccupation with food, and dieting attempts that are abnormal but not sufficient to warrant a diagnosis using the DSM-5 [31]. DE is a strong predictor for development of an ED, and its prevalence is elevated worldwide [44,45]. Up to 43.8% of Australian people and 74.5% of American adult women have excessive concerns about their body weight/shape that can interfere with quality of life [46,47]. Two in 5 American women have used diet pills to lose weight [47], and 1 in 5 female Canadian adolescents report DE behaviors [48,49].

The coexistence of an ED can complicate first-line recommendations for intervention in PCOS, including diet and lifestyle modifications to target reductions or maintenance of body weight [7,18]. Body weight loss in the overweight-obese individual with PCOS can improve insulin resistance (IR), cardiometabolic risk factors, and menstrual function [7,50–53]. However, a focus on weight loss for an individual with an ED may inadvertently reinforce harmful behaviors and exacerbate the ED [22]. Diet recommendations such as energy restriction may trigger ED symptoms, including restrictive eating, purging, and excessive exercise [54,55]. To add to the challenge, those with PCOS and obesity have been reported to be metabolically resistant to diet-lifestyle interventions to induce weight loss [51,56,57]. Failure to achieve a desired weight goal may then further exacerbate feelings of guilt, shame, and anxiety surrounding food intake and body image, potentially triggering or worsening an ED in those with PCOS [54].

Screening for an ED is recommended in PCOS; however, our understanding of the pathophysiology and prevalence of different types of EDs in PCOS across the lifespan remains limited [20,30,16,58–62]. The aim of this scoping review was to determine the current state of knowledge on the prevalence of different types of EDs and to review the etiology of EDs in PCOS.

Methods

Review protocol

The PRISMA-ScR was used to guide the protocol of this scoping review [63]. Ethics approval and registration was not required for this study.

Eligibility criteria

The eligibility criteria for studies included in this scoping review are shown in Table 3. Eligible studies had to use a primary research study design; accordingly, a variety of study designs were eligible for inclusion (including cross-sectional, cohort, case-control, case studies, case series, randomized control trials, mixed methods, and qualitative studies). Because meta-analyses report new results, they were eligible for inclusion, whereas systematic reviews were not. Studies in any language other than English were excluded for lack of reliable translation methods.

Information sources and search strategy

The databases searched were PubMed, Scopus, PsycINFO, and CINAHL. A search algorithm was developed for PubMed (Table 4), which was adapted for the other databases. Searches were completed up to and including 5 June 2023. Database searches were undertaken by 5 reviewers (SL-B, MM, RM, KN, SS). Search results from each database were exported into Covidence, a systematic review management software. Within Covidence, duplicate studies were removed. To confirm eligibility, the titles and abstracts were screened independently by all 5 reviewers during which any disagreements on eligibility were resolved by

TABLE 3

Eligibility criteria for study inclusion in the scoping review

Inclusion criteria
<ol style="list-style-type: none"> 1. Study design: studies that independently identified the prevalence of an ED in PCOS or measured eating behaviors in PCOS; meta-analyses, RCTs, nonrandomized controlled trials, cross-sectional studies, case-control studies, cohort studies, qualitative studies 2. Participants: human participants of any age with reported or suspected diagnosis of PCOS (by any diagnostic criteria, see Supplemental Table 1) 3. Outcomes: all reported outcomes related to AN, BN, BED, pica, rumination disorder, OSFED, EDNOS, ARFID, any ED, and DE in PCOS (by any method or diagnostic criteria) 4. Year of publication: no limit was applied for the range of publication years
Exclusion criteria
<ol style="list-style-type: none"> 1. Study design: literature reviews, systematic reviews, editorials, commentaries 2. Participants: no reported or suspected diagnosis of PCOS (by any diagnostic criteria) 5. Outcomes: no reported outcomes related to AN, BN, BED, pica, rumination disorder, OSFED, EDNOS, ARFID, any ED, and disordered eating in PCOS (by any method or diagnostic criteria) 3. Language: not written in English 4. Full text: full text unavailable and not received within 4 wk of contact

Abbreviations: AN, anorexia nervosa; ARFID, avoidant/restrictive food intake disorder; BED, binge eating disorder; BN, bulimia nervosa; ED, eating disorder; EDNOS, eating disorder not otherwise specified; OSFED, other specified feeding or eating disorder; PCOS, polycystic ovary syndrome; RCT, randomized controlled trial.

TABLE 4
Search algorithm for PubMed database

Search algorithm for PubMed
((PCOS) OR (PCO) OR (polycystic ovar*)) AND ((eating disorder*) OR (appetite disorder*) OR (feeding disorder*) OR (nutrition disorder*) OR (disordered eating) OR (binge*) OR (bulimi*) OR (anorexi*) OR (feeding disorder) OR (purging) OR (body image) OR (body dissatisfaction) OR (dieting))

discussion. This was followed by full-text review by all 5 reviewers. Where full-text publications were unavailable, corresponding authors were contacted, and a 4-wk response was permitted (no requests for full texts were satisfied). The search strategy was augmented by 1 reviewer hand-searching through reference lists of papers relevant to the topic (including editorials and reviews) for additional studies that met the eligibility criteria.

Study selection

The database searches identified a total of 3409 records and a further 8 were identified from the reference lists of relevant publications. After removing duplicates ($n = 546$), 2863 publications underwent title and abstract screening based on which a further 2790 were excluded. A total of 73 studies were retrieved for full-text eligibility, and 35 were excluded with reasons including absence of PCOS in the study population, primary outcome not related to EDs, inappropriate study design, full text unavailable within 4 wk of contact with the author(s), and study not in English (Figure 1). This left 38 papers that met the eligibility criteria. Of the 38 included studies, 22 are case-control [1, 28,64–83], 9 are cross-sectional [84–92], 3 are cohort [93–95], 2 are trials [96,97], and 2 are meta-analyses [29,62].

Data extraction and synthesis of results

Data from the 38 included studies were extracted by 1 reviewer and verified by 4 other reviewers for calibration. Data relating to author(s), year of publication, geographic location, study population characteristics (age and BMI [kg/m²]), PCOS diagnostic criteria, ED screening/diagnostic tools, ED scores, ED prevalence, and other key findings related to the outcome measures were extracted. Once extracted, data were described and qualitatively synthesized (Table 5).

Results

Study and participant characteristics

Study and participant characteristics are summarized in Table 5. Six of the included studies were conducted in the United States [22,70,81,83,93,95], 6 in the United Kingdom [64,73,78, 87,89,94], 5 in Turkey [66,68,69,74,84], 4 in Australia [28,71, 79,92], 3 in Sweden [65,75,77], 2 in the Netherlands [82,96], and 2 in Poland [88,91]. One each of the remaining studies was conducted in Saudi Arabia [85], France [67], Iran [72], Slovenia [97], Latvia [76], Greece [86], Brazil [90], and Italy [80]. Both the included meta-analyses employed literature from combinations of countries [29,62].

Different criteria were used to diagnose PCOS in studies. A PCOS diagnosis is defined in the international evidence-based guideline for the assessment and management of PCOS (Supplemental Table 1) and the Rotterdam criteria was the most commonly used criteria in 17 studies [22,29,62,69,70,74–77,82, 84–86,88,91,93,95–97]. Eight studies relied upon a self-reported diagnosis or diagnosis found in the participant’s medical chart [28,64,66,68,73,79,81,92]. Several studies relied on other methods, such as solely polycystic ovary morphology on

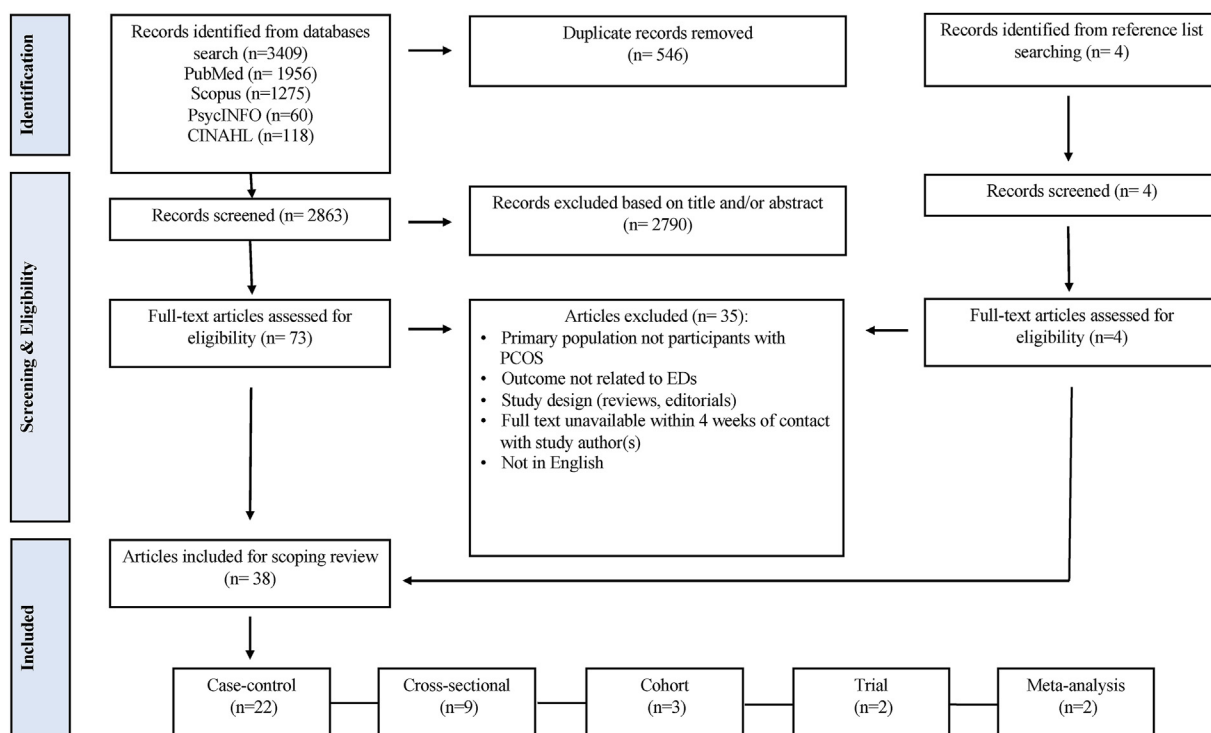


FIGURE 1. Study eligibility, selection, and data extraction. ED, eating disorder; PCOS, polycystic ovary syndrome.

TABLE 5

Summary of studies included in the scoping review

Study	Study characteristics	Participant characteristics Age (y): mean \pm SD BMI (kg/m ²): mean \pm SD	ED screening or diagnostic tool	Selected scores: mean \pm SD		Prevalence (%)	
				PCOS	Control	PCOS	Control
Annagür, 2015 [84]	Design: cross-sectional Location: Turkey Sample size: 88 (PCOS: 88) PCOS diagnosis: Rotterdam	PCOS Age: 22.26 \pm 3.55 BMI: Sample mean <25	SCID-I for the DSM-IV	—	—	Any ED: 9% BED: 6.8% BN: 2.3% AN: 0%	—
Asdaq, 2020 [85]	Design: cross-sectional Location: Saudi Arabia Sample size: 494 (PCOS: 116; control: 378) PCOS diagnosis: Rotterdam	PCOS Age: 65% of sample \geq 30 y, 35% of sample <30 y BMI: 61% of sample “normal”, 39.5% of sample BMI “obese”	Unvalidated binge eating questionnaire	—	—	Risk of BED 2.8 times greater in PCOS compared with controls (OR = 2.856, 95% CI: 1.328, 6.141)	—
Berni, 2018 [64]	Design: case-control Location: United Kingdom Sample size: 33876 (PCOS: 16938; control: 16938) PCOS diagnosis: Documented in medical chart	PCOS Age: 26.9 \pm 7.2 BMI: 29.86 \pm 7.86 Control Age: 27.0 \pm 7.36 BMI: 28.99 \pm 7.01	Documented in medical chart	—	—	Any ED: 1.55%***	Any ED: 1.03%***
Cesta, 2016 [65]	Design: case-control Location: Sweden Sample size: 268235 (PCOS: 24385; control: 243850) PCOS diagnosis: NIH and Rotterdam	PCOS Age: 28.0 \pm 6.8 BMI: N/A Control Age: Matched to PCOS group, otherwise N/A BMI: N/A	Documented in medical chart	—	—	Any ED: 2.45% AN: 0.57% BN: 0.73% Those with PCOS are more likely to have any ED (OR = 1.43, 95% CI: 1.31, 1.56) or BN (OR = 1.35, 95% CI: 1.15, 1.58), but not more likely to have AN (OR = 0.92, 95% CI: 0.78, 1.10)	Any ED: 1.73% AN: 0.62% BN: 0.55%
Coban, 2019 [66]	Design: case-control Location: Turkey Sample size: 59 (PCOS: 28; control: 31) PCOS diagnosis: previously diagnosed	PCOS Age: range from 13–19 y BMI: N/A but higher in PCOS compared with control group Control group Age: range from 13–19 y BMI: N/A but lower in	K-SADS-PL for the DSM-IV	Self-esteem score: 1.19 \pm 0.93	Self-esteem score: 1.52 \pm 0.93	AN: 0% BN: 0%	AN: 0% BN: 0%

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TABLE 5 (continued)

Study	Study characteristics	Participant characteristics Age (y): mean ± SD BMI (kg/m ²): mean ± SD	ED screening or diagnostic tool	Selected scores: mean ± SD		Prevalence (%)	
				PCOS	Control	PCOS	Control
Dumoulin, 1996 [67]	Design: case-control Location: France Sample size: 92 (oligomenorrhea + EDNOS:13; oligomenorrhea + no ED: 61; control: 18) PCOS diagnosis: Polycystic ovaries on US plus elevated LH and testosterone	control compared with PCOS group Oligomenorrhea with EDNOS Age: 23.6 ± 1.6 BMI: 21.4 ± 1.6 Oligomenorrhea with no ED Age: 26.8 ± 0.8 BMI: 21.2 ± 0.3 Control Age: 29.2 ± 1.6 BMI: 21.5 ± 0.6	EAT-26 and psychiatric interview for the DSM-IV	—	—	—	—
Eyupoglu, 2022 [68]	Design: case-control Location: Turkey Sample size: 389 (PCOS: 232; control: 157) PCOS diagnosis: self-reported	PCOS Age: 23 y BMI: 21.9 ± 2.6 Control Age: 23 BMI: 21.5 ± 2.9	TFEQ-18	TFEQ-18 (baseline): 28.5 ± 5.1*	TFEQ-18 (baseline): 27.3 ± 4.9*	—	—
Gökçen, 2020 [69]	Design: case-control Location: Turkey Sample size: 80 (PCOS: 40; control: 40) PCOS diagnosis: Rotterdam	PCOS Age: 25.25 ± 4.79 BMI: 27.23 ± 6.60 Control Age: 24.83 ± 3.17 BMI: 25.37 ± 4.07	EDE-Q, TFEQ-R21	EDE-Q: 2.32 ± 1.45*** TFEQ-R21 EE: 56.26 ± 33.50* CR: 55.13 ± 28.97* UE: 49.91 ± 20.99***	EDE-Q: 1.29 ± 1.07*** TFEQ-R21 EE: 44.58 ± 29.17* CR: 40.56 ± 19.86* UE: 32.21 ± 19.74***	—	—
Greenwood, 2020 [93]	Design: prospective cohort Location: United States Sample size: 5389 (PCOS: 164; control: 5225) PCOS diagnosis: Rotterdam	PCOS Age: 28.9 BMI: 28.1 Control Age: 30.26 BMI: 24.52	EDE-Q	EDE-Q: 2.33 ± 1.47***	EDE-Q: 1.52 ± 1.25***	—	—
Herriot, 2008 [94]	Design: retrospective cohort Location: United Kingdom Sample size: 88 (PCOS:88) PCOS diagnosis: by gynecologist or endocrinologist	PCOS Age: 32.4 ± 9.1 BMI: Normal-weight group mean 21.4; Overweight group mean 32.8	Self-report of previous or current ED	—	—	Any ED: 17%	—
Hollinrake, 2007 [70]	Design: case-control Location: United States Sample size: 206 (PCOS: 103; control: 103)	PCOS Age: 29.8 ± 6.2 BMI: 34.9 ± 8.5 Control	PRIME-MD PHQ for the DSM-IV			BED: 12.6%** BED: 30.5% (with depression) and 3% (no depression)	BED: 1.9%**

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TABLE 5 (continued)

Study	Study characteristics	Participant characteristics Age (y): mean ± SD BMI (kg/m ²): mean ± SD	ED screening or diagnostic tool	Selected scores: mean ± SD		Prevalence (%)	
				PCOS	Control	PCOS	Control
Jahanfar, 1995 [71]	PCOS diagnosis: Rotterdam Design: case-control Location: Australia Sample size: 94 (PCOS: 42; control: 52) PCOS diagnosis: Presence of polycystic ovaries on US	Age: 30.7 ± 8.5 BMI: 25.4 ± 4.7 PCOS Age: 27.55 ± 9.31 BMI: 23.23 ± 5.06 Control Age: 30.42 ± 8.59 BMI: 23.22 ± 3.67	BITE	BITE: 5.35 ± 0.98***	BITE: 3.19 ± 0.83***	—	—
Jahanfar, 2005 [72]	Design: case-control Location: Iran Sample size: 154 (PCOS: 25; control: 129) PCOS diagnosis: elevated testosterone or LH/FSH ratio plus symptoms of hyperandrogenism and chronic anovulation plus polycystic ovaries on US	PCOS Age: 21.77 ± 6.71 BMI: N/A	BITE	BITE: 3.27 ± 5.51	BITE: 2.06 ± 4.48	—	—
Jeanes, 2017 [73]	Design: case-control Location: United Kingdom Sample size: 678 (PCOS: 583; control: 95) PCOS diagnosis: previous diagnosis by healthcare professional	Lean PCOS Age: 31.3 ± 5.6 BMI: 22.5 ± 1.8 Overweight PCOS Age: 31.4 ± 7.6 BMI: 27.3 ± 1.4 Obese PCOS Age: 32.63 ± 7.3 BMI: 39.7 ± 7.3 Control Age: matched to lean PCOS BMI: matched to lean PCOS	BITE, FCQ-T, TFEQ-R18, self-reported	FCQ-T: 114.0 ± 34.9 Binge eating symptom: 10.9 ± 7.8** Binge eating severity: 2.8 ± 2.1	FCQ-T: 105.6 ± 26.6 Binge eating symptom: 7.4 ± 6.0** Binge eating severity: 2.0 ± 2.2	Intake questionnaire: Any ED: 2% (lean), 1% (overweight), 2% (obese) Study results: BED: 13.6% (lean), 23.2% (overweight), 39.2% (obese)	—
Jensterle, 2015 [97]	Design: nonrandomized trial Location: Slovenia Sample size: 36 (PCOS: 36) PCOS diagnosis: NICHD	PCOS Age: 31.2 ± 7.8 BMI: 38.7 ± 0.1	TFEQ-R18	TFEQ-R18 (baseline) UE: 36.8 ± 24.5*** EE: 49.9 ± 33.33*** CR: 52.8 ± 18.3 TFEQ-R18 (post) UE: 19.6.8 ± 18 ± 4*** EE: 28.5 ± 26.9*** CR: 52.5 ± 22.0	—	—	—
Jiskoot, 2022 [96]	Design: randomized controlled trial Location: The Netherlands	Care as usual (control) Age: 28.0 BMI: 30.6 Lifestyle intervention	DEBQ, EDE-Q	EDE-Q (baseline): 1.7–2.1 [0.0–3.1] DEBQ: EE: 2.4–2.9 [1.9–3.5]	—	—	—

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TABLE 5 (continued)

Study	Study characteristics	Participant characteristics Age (y): mean ± SD BMI (kg/m ²): mean ± SD	ED screening or diagnostic tool	Selected scores: mean ± SD		Prevalence (%)	
				PCOS	Control	PCOS	Control
	Sample size: 179 (Care as usual PCOS: 60; lifestyle intervention SMS- PCOS: 61; lifestyle intervention SMS+ PCOS: 58) PCOS diagnosis: Rotterdam	SMS-Age: 30.0 BMI: 33.5 Lifestyle intervention SMS+ Age: 28.0 BMI: 33.6		Restrained eating: 3.0–3.2 [2.7–3.6] External eating: 2.7–2.9 [2.2–3.3] Binge eating episodes (baseline): 41.7–57.1%			
Karacan, 2014 [74]	Design: case-control Location: Turkey Sample size: 94 (PCOS: 42; control: 52) PCOS diagnosis: Rotterdam	PCOS Age: 19.1 ± 2.3 BMI: 22.4 ± 3.8 Control Age: 19.7 ± 2.1 BMI: 21.4 ± 3.8	EAT-26	EAT-26: 46.66 ± 17.03	EAT-26: 48.21 ± 17.55	—	—
Kerchner, 2009 [95]	Design: cohort Location: United States Sample size: 60 (PCOS with depression: 24; PCOS without depression: 36) PCOS diagnosis: Rotterdam	PCOS with depression Age: 32.2 ± 6.3 BMI: 33.9 ± 8.3 PCOS without depression Age: 32.6 ± 6.3 BMI: 33.9 ± 8.3	PRIME-MD PHQ for the DSM-IV	—	—	BED: 25% (with depression) and 22% (without depression)	—
Larsson, 2016 [75]	Design: case-control Location: Sweden Sample size: 102 (PCOS: 72; control: 30) PCOS diagnosis: Rotterdam	PCOS Age: 30.2 ± 4.4 BMI: 28.5 ± 7.2 Control Age: 27.8 ± 3.6 BMI: 24.6 ± 5.0	TFEQ-R21, EAT-40, psychiatric questionnaire for the DSM-IV	TFEQ-R21 CR: 41 ± 23 UE: 42 ± 20 EE: 44 ± 28 EAT-40: 7.8 ± 6.7***	TFEQ-R21: CR: 37 ± 23 UE: 39 ± 15 EE: 37 ± 19 EAT-40: 16.4 ± 10.1***	BN: 0% AN: 0.08%	BN: 0% AN: 0.03%
Lee, 2017 [22]	Design: case-control Location: United States Sample size: 254 (PCOS: 148; control: 106) PCOS diagnosis: Rotterdam	PCOS Age: 28.12 ± 5.13 BMI: 33.85 ± 8.9 Control Age: 31.85 ± 8.09 BMI: 26.82 ± 7.54	EDE-Q, NEQ, DSM-5, DSM-IV	EDE-Q: ~2.4*** NEQ: 16.67 ± 6.18*	EDE-Q: ~1.3*** NEQ: 14.88 ± 5.43*	Any ED: 28.38% AN: 0% BN: 6.08% BED: 17.57% NES: 12.93%	Any ED: 18.87% AN: 0% BN: 5.66% BED: 10.38% NES: 12.38%
Lee, 2019 [29]	Design: meta-analysis Location: global Sample size: 860 (PCOS: 470; control: 390) PCOS diagnosis: Rotterdam or NIH	N/A	EAT-26, EAT-40, MINI, EDE-Q, PRIME-MD PHQ, BITE, NEQ, TFEQ-R21	Odds of an abnormally high ED score (OR = 3.05; 95% CI: 1.33, 6.99; 4 studies) higher in those with PCOS compared with controls	—	Odds of any ED diagnosis (OR = 3.87; 95% CI: 1.43, 10.49; 4 studies) higher in those with PCOS compared with controls	—
Lidaka, 2022 [76]	Design: case-control Location: Latvia Sample size: 129 (PCOS: 63; control: 66)	PCOS Age: 16 BMI: 89.9th percentile Control	BES	BES: 12 Mild to severe binge eating: 37.7%	BES: 12 Mild to severe binge eating: 36%	—	—

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TABLE 5 (continued)

Study	Study characteristics	Participant characteristics Age (y): mean \pm SD BMI (kg/m ²): mean \pm SD	ED screening or diagnostic tool	Selected scores: mean \pm SD		Prevalence (%)	
				PCOS	Control	PCOS	Control
Livadas, 2011 [86]	PCOS diagnosis: 2018 ESHRE guidelines Design: cross-sectional Location: Greece Sample size: 130 (PCOS: 130) PCOS diagnosis: Rotterdam	Age: 17 BMI: 46.9th percentile PCOS Age: 25.4 \pm 6.2 BMI: 26.60 \pm 7.01	Psychiatric interview for DSM (version unspecified), EAT-26	EAT-26: 11.75 \pm 9.6 (normal anxiety); 12.93 \pm 4.2 (moderate anxiety); 17.69 \pm 8.5 (severe anxiety)	—	Any ED: 0%	—
Månsson, 2008 [77]	Design: case-control Location: Sweden Sample size: 98 (PCOS: 49; control: 49) PCOS diagnosis: Rotterdam	PCOS Age: N/A BMI: 29.1 \pm 7.4 Control Age: N/A BMI: 23.5 \pm 3.0	MINI for the DSM-IV	—	—	Any ED: 21%** BN: 12% Women with PCOS have significantly more EDs than age-matched controls (OR = 6.4, 95% CI: 1.3, 31)	Any ED: 4%** BN: 4%
McCluskey, 1991 [78]	Design: case-control Location: United Kingdom Sample size: 261 (PCOS: 152; other endocrinopathy: 109) PCOS diagnosis: Conway et al. 1989 [82]	PCOS Age: 27.5 \pm 6.4 BMI: 24.4 \pm 5.1 Other endocrinopathies Age: 32.3 \pm 8.4 BMI: 25.4 \pm 5.4	BITE for the DSM-III	BITE: 7.8 \pm 6.1***	BITE: 5.2 \pm 4.5***	BN: 6%**	BN: 1%**
Michelmores, 2001 [87]	Design: cross-sectional Location: United Kingdom Sample size: 230 PCOS diagnosis: Polycystic ovaries on US, plus one of: menstrual irregularity, acne, hirsutism, BMI >25 kg/m ² , high testosterone, high LH	PCOS Age: 21.5 BMI: 22.9	EDE for the DSM-IV	EDE: 0.9	EDE: 0.8	AN: 0% BN: 0% BED: 4%	AN: 0% BN: 0% BED: 1%
Mizgier, 2020 [88]	Design: cross-sectional Location: Poland Sample size: 78 (PCOS: 78) PCOS diagnosis: Rotterdam	Normal-weight PCOS Age: 16.29 \pm 1.08 BMI: N/A Overweight/obese PCOS Age: 15.73 \pm 1.66 BMI: N/A	EAT-26	EAT-26: 9.27 \pm 5.67 (normal weight); 13.49 \pm 9.54 (overweight/obese)	—	—	—
Morgan, 2008 [89]	Design: cross-sectional Location: United Kingdom Sample size: 80 (PCOS:	Hirsutism Age: 29 \pm 5 BMI: N/A	EDE, SCID-I for the DSM-IV	—	—	Any ED: 36.3% EDNOS: 22.5% BN: 12.6% AN: 1.3%	—

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TABLE 5 (continued)

Study	Study characteristics	Participant characteristics Age (y): mean ± SD BMI (kg/m ²): mean ± SD	ED screening or diagnostic tool	Selected scores: mean ± SD		Prevalence (%)	
				PCOS	Control	PCOS	Control
	68; CAH: 9; acromegaly: 1; mixed endocrinopathies: 1; idiopathic hirsutism: 1) PCOS Diagnosis: N/A					Note that 85% of the sample had PCOS (whereas 100% had hirsutism) All of those diagnosed with an ED had PCOS. PCOS was the only endocrinopathy associated with ED	
Pirotta, 2019 [28]	Design: case-control Location: Australia Sample size: 899 (PCOS: 501; control: 398) PCOS diagnosis: Self-reported	PCOS Age: 30.5 ± 5.9 BMI: 33.6 ± 9.3 Control Age: 22.8 ± 5.5 BMI: 24.3 ± 6.0	EDE-Q for the DSM-5	EDE-Q: 2.30*	EDE-Q: 2.08*	Any ED: 62% BED: 29% Low-frequency BED: 16% Low-frequency BN: 9.4% BN: 5.2% Unspecified feeding or ED: 1.2% Purging disorder: 1.2% AN: 0%* Atypical AN: 0%*** DE: 21%* BED: 1.4%	Any ED: 56% BED: 23% Low-frequency BED: 12% Low-frequency BN: 8.0% BN: 4.5% Unspecified feeding or ED: 3% Purging disorder: 0.8% AN: 1.3%* Atypical AN: 5.8%*** DE: 15%*
Rassi, 2010 [90]	Design: cross-sectional Location: Brazil Sample size: 72 (PCOS: 68; potential PCOS: 4) PCOS diagnosis: polycystic ovaries on US plus one or more of: menstrual irregularities, acne, hirsutism, and hyperandrogenism	PCOS Age: 26.2 ± 5.05 BMI: N/A	MINI for the DSM-IV	—	—		
Rodino, 2016 [79]	Design: case-control Location: Australia Sample size: Seeking IVF (total): 403 (PCOS: 55; non-PCOS: 348) PCOS diagnosis: Self-reported	PCOS Age: 31.8 ± 4.4 BMI: 27.2 ± 6.6 Control Age: 34.9 ± 4.7 BMI: 24.7 ± 4.7	EDE-Q	EDE-Q Shape concern: 1.7 ± 1.3**	EDE-Q Shape concern: 1.4 ± 1.3**	PCOS status not associated with risk of binge eating	—
Sbaragli, 2007 [80]	Design: case-control Location: Italy Sample size: 302 (infertile couples: 81; pregnant	Infertile Age: 35 ± 5 BMI: N/A Pregnant	SCID-1 for the DSM-IV	—	—	Females with endocrine infertility (i.e., PCOS) BED: 15%*	Females with anatomical infertility: BED: 3%* Females with infertile male partner:

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TABLE 5 (continued)

Study	Study characteristics	Participant characteristics Age (y): mean \pm SD BMI (kg/m ²): mean \pm SD	ED screening or diagnostic tool	Selected scores: mean \pm SD		Prevalence (%)	
				PCOS	Control	PCOS	Control
	couples: 70) PCOS diagnosis: N/A	Age: 31 \pm 4 BMI: N/A					BED: 0%* Females with functional infertility: BED: 25%* Any ED: 0.3%
Sirmans, 2014 [81]	Design: case-control Location: United States Sample size: 6756 (PCOS: 1689; control: 5067) PCOS diagnosis: "Probable PCOS" in medical chart	PCOS Age: 25.24 BMI: N/A Control Age: 25.23 BMI: N/A	Documented in medical chart	—	—	Any ED: 0.4%	
Suchta, 2023 [91]	Design: cross-sectional Location: Poland Sample size: 122 (PCOS: 122) PCOS diagnosis: Rotterdam	PCOS Age: 26 \pm 5.22 BMI: N/A	Psychiatric interview, QBES for the DSM-5	—	—	BED: 42%	—
Tay, 2019 [92]	Design: cross-sectional Location: Australia Sample size: 8467 (PCOS: 875; control: 7592) PCOS diagnosis: self-reported	PCOS Age: 24.8 \pm 1.7 BMI: 29.2 \pm 7.9 Control Age: 24.6 \pm 1.8 BMI: 25.3 \pm 5.8	Self-reported	—	—	Any ED: 11%*** AN: 3.5% BN: 3.4% Other ED: 6.4%***	Any ED: 7.6%*** AN: 3.4% BN: 2.6% Other ED: 3.4%***
Thannickal, 2020 [62]	Design: meta-analysis Sample size: 349,529 PCOS diagnosis: Rotterdam	N/A	Not specified	—	—	Compared with control: AN: OR = 0.92, 95% CI: 0.78, 1.10 (3 studies) BN: OR = 1.37, 95% CI: 1.17, 1.60 (5 studies) BED: OR = 2.95, 95% CI: 1.61, 5.42 (4 studies) Any ED: OR = 3.68, 95% CI: 1.38, 9.81 (4 studies)	—
Wang, 2021 [82]	Design: case-control Location: The Netherlands Sample size: 491 (PCOS: 170; control: 321) PCOS diagnosis: Rotterdam	PCOS Age: 28.0 \pm 4.2 BMI: 36.0 \pm 3.5 Control Age: 30.8 \pm 4.4 BMI: 36 \pm 3.3	DEBQ	DEBQ: EE: 34.6 \pm 11.2 External eating: 27.7 \pm 6.3 Restricted eating: 32.3 \pm 5.9	DEBQ: EE: 34.1 \pm 11.3 External eating: 27.5 \pm 5.8 Restricted eating: 31.7 \pm 6.1	—	—
Weiner, 2004 [83]	Design: case-control Location: United States	PCOS Age: 28.19 \pm 4.84	EDI-2 (selected subscales)			—	—

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TABLE 5 (continued)

Study	Study characteristics	Participant characteristics Age (y): mean ± SD BMI (kg/m ²): mean ± SD	ED screening or diagnostic tool	Selected scores: mean ± SD		Prevalence (%)	
				PCOS	Control	PCOS	Control
	Sample size: 54 (PCOS: 27; control: 27) PCOS diagnosis: oligo/amenorrhea + hyperandrogenemia + infertility, hirsutism, acne, or androgenetic alopecia + exclusion of other endocrinopathy	BMI: 37.7 ± 8.46 Control Age: 30.07 ± 6.48 BMI: 36.89 ± 7.24		No difference in binge eating between groups	No difference in binge eating between groups		

Abbreviations: AN, anorexia nervosa; BED, binge eating disorder; BES, binge eating scale; BITE, Bulimic Investigatory Test, Edinburgh; CR, cognitive restraint; DE, disordered eating; DEBQ, Dutch Eating Behavior Questionnaire; DSM, Diagnostic and Statistical Manual of Mental Disorders; EAT, eating attitudes test; ED, eating disorder; EDI, Eating Disorder Inventory; EDE, Eating Disorder Examination; EDE-Q, Eating Disorder Examination Questionnaire; EE, emotional eating; EDNOS, Eating disorder not otherwise specified; ESHRE, European Society of Human Reproduction and Embryology; FCQ-T, Food Cravings Questionnaire—Trait; FSH, follicle-stimulating hormone; IVF, in vitro fertilization; K-SADS-PL, Schedule for Affective Disorders and Schizophrenia in School Age Children; LH, luteinizing hormone; MINI, Mini-International Neuropsychiatric Interview; NEQ, night eating questionnaire; NICHD, National Institute of Child Health and Human Development; PCOS, polycystic ovary syndrome; PRIME-MD PHQ, PRIME-MD Patient Health Questionnaire; QBES, Questionnaire for Binge Eating Screening; RCT, randomized controlled trial; SCID-I, Structured Clinical Interview 1; SMS, short message service; TFEQ, Three-Factor Eating Questionnaire; UE, uncontrolled eating; US, ultrasound.
P* < 0.05; *P* < 0.01; ****P* < 0.001.

ultrasound [71], polycystic ovaries on ultrasound plus 1 or more other biochemical and clinical features of PCOS [67,78,83,87,90], other clinical and biochemical measures [72], and otherwise unspecified diagnostic criteria [80,89,94] for defining PCOS.

The age of all included participants ranged from 13 y [66,76] to 35.5 ± 5 y [80], whereas BMI ranged from 21.2 ± 0.3 kg/m² [67] to 36.89 ± 7.24 kg/m² [83]. However, 10 studies did not report BMI data for ≥1 study subgroup [29,62,65,66,72,80,88–91] and 4 studies did not report age data for ≥1 study subgroup [29,62,65,77].

Of 25 studies that reported on prevalence of EDs in PCOS, 13 reported on BED, 12 reported on BN, 10 reported on AN, 2 reported on OSFED [including night eating syndrome (NES), low-frequency BN, low-frequency BED, purging disorder, and atypical anorexia], 1 reported on ED not otherwise specified (EDNOS), and 0 reported on ARFID, pica, and RD. Thirteen studies reported on the prevalence of any ED in PCOS (Table 5).

Tools used for screening and diagnosis of EDs and DE

A total of 15 tools were used in 38 studies to explore ED and DE behaviors in those with PCOS (Table 6) [98–115]. To determine the prevalence of individuals with EDs, 12 studies used structured interviews with participants. These included the Structured Clinical Interview 4 [80,84,89], the Schedule for Affective Disorders and Schizophrenia in School Age Children interviews [66], the semistructured Eating Disorder Examination (EDE) [87,89], the Mini-International Neuropsychiatric Interview [77,90], and other psychiatric interviews [67,75,86,91]. Of these, all used the DSM-IV diagnostic criteria for diagnosing an ED, whereas 1 study [91] used the most current DSM-5 criteria, and another [86] did not specify the edition used.

Thirty studies used ≥1 self-administered questionnaire to assess eating behaviors in participants. One study used the original 40-question Eating Attitude Test (EAT-40) [75] and 4 studies used the shorter 26-question test (EAT-26) [67,74,86,88]. Six studies used the Eating Disorder Examination Questionnaire (EDE-Q) [22,28,69,79,93,96], that is a 28-item self-report questionnaire adapted from the EDE interview. None of the studies used the original 51-item Three-Factor Eating Questionnaire (TFEQ), and 4 studies used the condensed TFEQ-R21 [69,75] and TFEQ-R18 [73,97] versions. Four studies explored BN behaviors using the Bulimic Investigatory Test, Edinburgh (BITE) [71–73,78], based on the DSM-III criteria for BN. Two studies screened for EDs through a nonspecific psychiatric disorder questionnaire, the PRIME-MD Patient Health Questionnaire [70,95]. Two studies used the Dutch Eating Behavior Questionnaire [82–96] and another used the second version of the Eating Disorders Inventory [83]. The other tools used were the Food Cravings Questionnaire Trait to assess for lack of control over eating and preoccupations about food [73], the Night Eating Questionnaire to assess for presence of NES [22], the Binge Eating Scale to assess binge-eating severity [76], the unvalidated Questionnaire for Binge Eating Screening to screen for binge eating [91], and other unvalidated binge eating questionnaires [85]. Six studies relied upon ED diagnosis being reported in the patient’s chart or on self-reported diagnosis [64,65,73,81,92,94].

TABLE 6
Tools used in the assessment of eating disorders

Instrument name	Details	Tests for	Psychometric data
Structured interviews Structured Clinical Interview (SCID) [98]	SCID for DSM-5 was developed for making the major diagnoses by DSM-5 criteria. It has now been superseded by the SCID for DSM-5, which has a specific version and module for assessing for the feeding and eating disorders [98]	EDs—all DSM-defined EDs	Currently no psychometric data available for the SCID-5 eating and feeding disorder diagnoses (has been shown to be suitable for the schizophrenia spectrum but less so for the anxiety spectrum) [99]
Eating Disorder Examination (EDE) [100]	Used to identify frequency of DE behaviors over the past 29 d and to provide 4 subscale scores: 1) Restraint, 2) Eating Concern, 3) Shape Concern, and 4) Weight Concern. Community norms are reported and include mean score of 0.932 ± 0.805 for the global EDE [100]	EDs—AN, BN, BED	A systematic review of the EDE supports the reliability and validity of its scores [101]
Mini-International Neuropsychiatric Interview (MINI) [102]	Brief structured diagnostic interview for the major psychiatric disorders by DSM-III, DSM-IV, and DSM-5	EDs—AN, BN	Found to have 90% sensitivity for AN, 99% sensitivity for BN, 100% specificity for AN, and 99% specificity for BN [101]
Schedule for Affective Disorders and Schizophrenia in School Age Children (K-SADS- PL) [103]	Semistructured diagnostic interview designed to assess current and past episodes of psychopathology in those aged 6–18 according to DSM-III and DSM-IV criteria	EDs—all DSM-defined EDs	Suggested to generate reliable and valid child psychiatric diagnoses [103]
Self-administered questionnaires Eating Attitudes Test (EAT-40 and EAT-26) [104,105]	Used to identify ED risk based on behaviors, feelings, and attitudes associated with eating. The EAT-40 (40-item questionnaire) was developed assess for AN. A cutoff score of ≥ 30 is considered a high risk, and 21–30 is a moderate risk. The EAT-26 (26-item questionnaire) was later developed to screen for AN, BN, and BED. A score of >20 is considered a high risk	EDs—AN, BN, BED	For both EAT-40 and EAT-26 internal consistency has been shown to be good (Cronbach's alpha for the total scores found to be 0.83 or higher) [108]. The EAT-26 has 88% sensitivity and 96% specificity for DE [106]
Eating Disorder Examination Questionnaire (EDE-Q)	28-item questionnaire adapted from the EDE interview. Assesses ED behaviors over the past 28 d and uses the same subscales as the EDE Community norms are reported and include a mean score of 1.554 ± 1.213 (Global EDE-Q) [100]	EDs—AN, BN, BED	The EDE-Q has 83% sensitivity and 96% specificity for disordered eating [110]. Some work suggests that the EDE-Q may generate higher scores than the EDE interview for items including binge eating and shape concerns [101]
Bulimic Investigatory Test, Edinburgh (BITE)	33-item questionnaire that assesses symptoms of DSM-III BN. Two subscales: 1) Symptoms and 2) Severity [107]	EDs—BN	Shown to have satisfactory reliability and validity for DSM-III criteria of BN [108]
PRIME-MD Patient Health Questionnaire (PRIME- MD PHQ)	26-item questionnaire that screens for 5 of the most common groups of disorders in primary care (depressive, anxiety, alcohol, somatoform, and EDs) followed by a clinician evaluation [109]	EDs—AN, BN, BED	The PRIME-MD PHQ has diagnostic validity comparable with the original clinician-administered PRIME-MD [109]

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TABLE 6 (continued)

Instrument name	Details	Tests for	Psychometric data
Eating Disorder Inventory (EDI)	Measures behaviors associated with AN and BN. The latest version, the EDI-3, has 91 items and 12 subscales [107]	EDs—AN, BN	Validation studies have shown good internal consistency in EDI-3 subscales and ability to discriminate between cases and controls [110]
Night Eating Questionnaire (NEQ)	14-item questionnaire assessing for NES. Four subscales: 1) Nocturnal Ingestions, 2) Evening Hyperphagia, 3) Morning Anorexia, and 4) Mood/Sleep [107]	ED—NES	Shown to be a valid measure of severity of NES [111]
Three-Factor Eating Questionnaire (TFEQ-R21 and TFEQ-R18)	The full TFEQ is a 51-item self-report measure of eating behavior. It has 3 subscales: 1) Cognitive Restraint of Eating, 2) Hunger, and 3) Disinhibition. The TFEQ-R18 was the first shortened version developed and was later refined to become the TFEQ-R21	DE—Eating behaviors	Cronbach's alpha for the 3 subscales found to be good at 0.85 or higher [116] for the full TFEQ Psychometric properties of the TFEQ-R21 and TFEQ-R18 are both acceptable [112]
Food Cravings Questionnaire—Trait (FCQ-T)	39-item questionnaire designed to measure the frequency and intensity of food cravings. Has 9 subscales that relate to lack of control over eating, preoccupations about food, and food cravings [73]	DE—Eating behaviors	Studies support reliability and validity of the FCQ but findings about the 9 subscales are inconsistent [113]
Dutch Eating Behavior Questionnaire (DEBQ)	33-item questionnaire to assess eating behaviors related to obesity. 3 subscales: 1) Emotional Eating, 2) External Eating, and 3) Restrained Eating [107]	DE – Eating behaviors (in obesity)	Cronbach's alpha for the DEBQ subscales found to range from 0.80 to 0.95 [114]
Binge Eating Scale (BES)	16-item questionnaire assessing binge eating severity by behaviors and cognitive symptoms	DE—binge eating behaviors	BES scores have been shown to differentiate between those with no, moderate, and severe levels of binge eating [115]
Questionnaire for Binge Eating Screening (QBES)	Consists of 4 yes or no questions to screen for binge eating. One or more “yes” responses indicate the presence of binge eating [91]	DE—binge eating behaviors	Psychometrics of this screening tool have not yet been evaluated [91]

Abbreviations: BES, Binge Eating Scale; BITE, Bulimic Investigatory Test, Edinburgh; DE, disordered eating; DEBQ, Dutch Eating Behavior Questionnaire; DSM, Diagnostic and Statistical Manual of Mental Disorders; EAT, Eating Attitudes Test; EDI, Eating Disorder Inventory; EDE, Eating Disorder Examination; EDE-Q, Eating Disorder Examination Questionnaire; FCQ-T, Food Cravings Questionnaire—Trait; K-SADS-PL, Schedule for Affective Disorders and Schizophrenia in School Age Children; MINI, Mini-International Neuropsychiatric Interview; NEQ, Night Eating Questionnaire; NES, night eating syndrome; PRIME-MD PHQ, PRIME-MD Patient Health Questionnaire; QBES, Questionnaire for Binge Eating Screening; SCID, Structured Clinical Interview; TFEQ, Three-Factor Eating Questionnaire.

Prevalence of EDs in PCOS

The prevalence of each ED in PCOS is summarized and compared with the prevalence of EDs in females among the general population in different populations (Table 2).

Prevalence of any ED

The prevalence of any ED in those with PCOS ranged from 0% to 62% as reported in 13 studies [22,28,62,64,65,73,77,81,84,86,89,92,94]. Two large epidemiologic studies found that the prevalence of any ED in those with PCOS was 1.55% [64] and 2.45% [65], and this was significantly higher in age-matched

controls that was 1.03% [64] and 1.73% [65], respectively. A case-control study found the prevalence of any ED was 21% in PCOS compared with 4% in controls [77]. Similarly, a cohort study found the prevalence of any ED to be 11% in PCOS compared with 7.6% in controls [92]. A meta-analysis concluded that those with PCOS were 3–4 times more likely to have any ED [odds ratio (OR): 3.68; 95% confidence interval (CI): 1.38, 9.81] compared with females without PCOS [62]. The highest OR reported in the literature indicated that those with PCOS had 6-fold higher prevalence of EDs compared with age-matched controls (OR: 6.4; 95% CI: 1.3, 31) [77]. In another cohort

study composed of females with endocrinopathies, all those diagnosed with an ED had PCOS, and PCOS was the only endocrine diagnosis associated with the presence of an ED [89].

On the other hand, 3 case-control studies found no significant difference in prevalence of any ED in PCOS compared with controls [22,28,81]. One case-control study found low prevalence of EDs in both PCOS (0.4%) and non-PCOS participants (0.3%) [81]. Two case-control studies found elevated prevalence of EDs in both PCOS (28.38% and 62%, respectively) and non-PCOS participants (18.87% and 56%, respectively) [22,28]. The latter 2 studies concluded that DEs, but not EDs, were more prevalent in PCOS. Four studies reported on ED prevalence in PCOS without comparison with a non-PCOS control group. These studies reported a prevalence of any ED to be 0% in a Greek sample [86], 1.7% in a UK sample [94] and 9% in a group of Turkish university students [84]. One study compared ED prevalence in PCOS based on body weight and reported ED prevalence in lean, overweight, and obese groups as nonsignificant at 2%, 2%, and 1%, respectively [73]. Collectively, studies demonstrate an increased prevalence of EDs in those with PCOS with no apparent impact of body weight.

ED scores

Many studies have reported ED scores without measuring prevalence of an ED in PCOS. Three studies found no difference in ED scores between those with and without PCOS [74,82,97]. Seven studies found higher scores on the TFEQ-18 and TFEQ-21 [68,69], EDE-Q [22,69,93,96], EAT [75], and BITE [73] scales in those with PCOS compared with controls. In one study, 12.16% of those with PCOS had elevated EDE-Q scores compared with 2.83% of controls [22]. Results from a meta-analysis showed that those with PCOS had higher odds of having elevated ED scores when compared with controls (OR: 3.05; 95% CI: 1.33, 6.99) [29].

One study showed higher EDE-Q and TFEQ-21 scores in overweight-obese women with PCOS compared with overweight-obese controls but no difference in scores between the groups in those with BMI <25 kg/m² [69]. Similarly, another study found that those with PCOS and BMI >25 kg/m² had median EDE-Q scores higher than the mean reported in the general population (1.7–2.1 compared with 1.29) [96]. The association between ED score and overweight in PCOS was also supported in another study that found that EAT-26 score ≥ 20 was correlated with a 7-fold (OR: 6.88; 95% CI: 1.35, 34.97) increased OR with overweight or obesity [88]. Another study reported a BMI increase of 1 kg/m² corresponded to 15% increased odds of scoring in highest EDE-Q score tertile in those with PCOS [93]. However, another study in adolescents with PCOS found no significant difference in EAT-26 scores between those with overweight/obesity and PCOS and those without overweight/obesity and PCOS [88]. Together, these findings suggest that there may be an association between overweight and ED in those with PCOS; however, this may depend on the type of ED.

Prevalence of AN

Ten studies reported the prevalence of AN in PCOS and 5 of these found the prevalence of AN to be 0% in PCOS [22,28,66,84,87]. The highest prevalence of AN reported in a PCOS sample was 3.5%, but this result was insignificantly different to the 3.4%

prevalence found in controls [92]. In this case, the authors indicated that AN prevalence may have been unusually high in their large Australian population sample because of reliance on self-reported AN and PCOS diagnoses [92]. Other papers found that AN prevalence in PCOS was 0.08% [75], 0.57% [65], and 1.3% [89]. Both a large epidemiologic study [65] and a meta-analysis [62] concluded that females with PCOS are not any more likely to have AN compared with controls (OR: 0.92, 95% CI: 0.78, 1.10). Overall, the literature suggests that although AN occurs in those with PCOS, it is of low prevalence compared with other EDs.

Prevalence of BN

The prevalence of BN in those with PCOS ranged from 0% to 12% as reported in 13 studies [22,28,62,65,66,71,72,75,77,78,84,87,92]. Early work published in 1991 and 1995 found that people with PCOS scored higher on the BITE questionnaire and were 6 times more likely to have subclinical BN when compared with controls [71,78]. In these studies, subclinical BN was defined as a score of 10–19 on the BITE, where a score above 20 suggests a high risk of having BN. In the context of recent studies and use of other ED assessment tools, subclinical BN would be categorized as OSFED or DE. Interestingly, a decade later, one research team reported no difference in BITE scores in PCOS compared with controls in a cohort study that used PCOS diagnostic criteria and a large sample size [72]. Certain studies have found elevated rates of BN in PCOS at 12% [77], 6.08% [22], 5.2% [28], and 3.4% [92]; however, these were not significantly different from non-PCOS control groups. Three studies found 0% BN prevalence in PCOS [66,75,87]. Both a large epidemiologic study [65] and a meta-analysis [62] have concluded that individuals with PCOS have 30% higher odds of having BN compared with controls (OR: 1.35, 95% CI: 1.15, 1.58 and OR: 1.37, 95% CI: 1.17, 1.60, respectively).

Prevalence of BED

The prevalence of BED in those with PCOS ranges from 1.4% to 42% as reported in 15 studies [22,28,62,70,73,76,79,80,83–85,87,91,95]. One study found elevated rates of BED in PCOS (12.6%) compared with controls (1.9%) [70] and another found a 2.8-fold greater risk of BED in PCOS; however, the latter study did not employ a validated BED questionnaire [85]. A meta-analysis of 4 studies concluded that those with PCOS were more likely to have BED compared with controls (OR: 2.95, 95% CI: 1.61, 5.42) [62]. On the other hand, 6 studies found that BED was not associated with PCOS [22,28,76,79,83,87]. Another 5 studies reported on BED prevalence in PCOS but without comparison with a non-PCOS control group. These studies reported prevalence of BED in PCOS to be 1.4% [90], 6.8% [84], 15% [80], and 42% [91]. One study assessed the relationship between BED and BMI and observed prevalence to be 13.6% in lean, 23.2% in overweight, and 39.2% in obese individuals with PCOS [73], suggesting a significant positive association between body weight and BED risk. BED is also hypothesized to be associated with depression. One study found that BED prevalence was 30.5% in those with both PCOS and depression, whereas it was only 3% in those with PCOS and no depression [70]. However, another study found that risk of BED in those with PCOS and depression was similar to that of those with PCOS and no depression (25% and 22%, respectively) [95].

Prevalence of OSFEDs

Two studies reported on OSFED prevalence in PCOS (which includes NES, low-frequency and/or limited duration BN, low-frequency and/or limited duration BED, purging disorder, and atypical anorexia) [22,28]. NES prevalence was found to be 12.93% in PCOS compared with 12.38% in controls [22]. Although the difference between groups was not significant in this large case–control study ($n = 254$), the prevalence of NES was high compared with a population study that reported the prevalence of NES to be 1.6% [42] (Table 5). In another study, the prevalence of both BN and BED of low-frequency and/or limited duration was found to be similar and not significantly different between those with and without PCOS (9.4% compared with 8%; 16% compared with 12%, respectively) [28]. Purging disorder was found to not be significantly different in those with (1.2%) and without PCOS (1.2%) [28]. Atypical anorexia was reported at 0% in individuals with PCOS, whereas controls had a prevalence of 5.8% [28]. Of note, no studies reported on the prevalence of ARFID, RD, or pica in a PCOS population.

Discussion

EDs are increased in PCOS

EDs have been proposed to be related to the disruption of the hypothalamic–pituitary–ovarian axis through metabolic, genetic, or psychological factors and these can be associated with altered eating and food-seeking behaviors in PCOS [21,65,67]. EDs can profoundly impact quality of life and have been proposed to contribute to the pathology of PCOS related to adiposity, IR, and androgen excess [21,26,116,117]. In the international guidelines for assessment and management of PCOS, it is recommended that ED and DE should be taken into account and subsequent referral of patients for diagnoses and psychological management [8]. In this scoping review we have investigated the prevalence of the full range of medically diagnosed EDs in individuals with PCOS using the DSM and other screening methodologies. We report that individuals with PCOS are 3–6-fold more likely to have an ED and have higher ORs of an elevated ED score compared with controls. PCOS is associated with an increased prevalence of an ED, in particular BED and BN but not AN, NES, or atypical AN. EDs have a low prevalence in the general population; therefore, data on the prevalence of a range of specific ED diagnoses are limited, particularly in those with PCOS; therefore, the prevalence of EDs in PCOS is likely to be underestimated. There are very limited studies on NES, atypical AN, pica, RD, and ARFID in PCOS. Diagnosis also depends on the tool used to assess for an ED and these vary across studies. The relationship between body weight and an ED may depend on the type of ED, and requires further investigation. Body weight does not appear to be associated with the prevalence of an ED; however, ED scores are higher in PCOS individuals with overweight-obesity compared with BMI-matched controls and BED is increased with BMI [73,88,93,96]. The proposed etiology of EDs in PCOS is multifactorial involving genetic, metabolic, endocrine, and psychological factors and remains understudied [20,21,26,29]. Further investigations are required to understand the pathophysiology and risk of specific types of EDs in PCOS. The pathophysiologic mechanisms that underlie EDs in PCOS are discussed below and summarized in Figure 2.

Psychological risk factors for EDs in PCOS

Emerging evidence suggests that body weight dissatisfaction is associated with the risk of EDs in individuals with PCOS [30,66]. Studies have consistently revealed higher prevalence of body dissatisfaction, perception of being overweight, and multiple weight-loss attempts in those with PCOS compared with controls, independent of BMI [30,83,118,119]. The connection between the pursuit of body weight loss and BED risk is well established, with a 10-fold higher prevalence of BED in adults seeking weight loss compared with the general population [120]. The first-line intervention in those with overweight-obesity in PCOS is targeting diet and lifestyle habits to promote healthy eating behaviors and reduce body weight by 5%–10% [7]. Therefore, it is not surprising that those with PCOS who experience body dissatisfaction and/or overweight-obesity may feel psychological pressure to lose weight, and this may promote the development of an ED [30,66]. Energy-restricted diets and restrictive eating patterns are contributors to BED development [96] and several studies have shown that those with PCOS self-report frequently dieting to reduce body weight [30]. However, other studies show that obese individuals with PCOS who report dieting to lose weight have similar binge eating symptom scores compared with obese PCOS women who are not dieting [73]. Another case–control study found that scores for body dissatisfaction, eating attitudes, and self-esteem were not higher in young adults with PCOS compared with non-PCOS controls [74]. However, both groups had very high scores on the EAT-26 (46.66 ± 17.03 and 48.21 ± 17.55 , respectively), where a score of >20 is considered a high risk of an ED [74] (Table 6).

It has been suggested that treating childhood and adolescent obesity as a root cause of PCOS through weight reduction strategies may prevent development of BED and PCOS later in life [121–123]. However, it has been proposed that PCOS may be a result of psychological distress related to dieting and altered food-seeking behaviors during childhood and puberty, contributing to the development of an ED and obesity [27]. Treatment of adolescents with obesity and PCOS using energy-restricted diets rather than promoting healthy eating patterns and health-promoting behaviors may contribute to development of an ED because of increased pressure to lose weight, emotional distress associated with body weight, and body dissatisfaction [30,61,74,124,125].

It has been proposed that menstrual irregularities, hirsutism, overweight, and anxiety about fertility in PCOS are in conflict with accepted societal views of femininity, and these factors contribute to emotional distress and low self-esteem that may lead to altered food-seeking and consumption behaviors to cope [78,126–128]. Self-esteem building with guidance from a psychotherapist or counselor to reframe negative self-talk, cultivate self-compassion, and improve confidence may reduce the risk of ED development, without explicitly focusing on body weight change [92].

Individuals with PCOS are at higher risk for psychological comorbidities, such as anxiety and depressive disorders [16,20]. In several studies, it has been shown that anxiety and depressive disorders are associated with an increased risk of an ED in those with PCOS [22,64,86,93]. One study found that BED prevalence was 30.5% in those with both PCOS and depression, whereas it

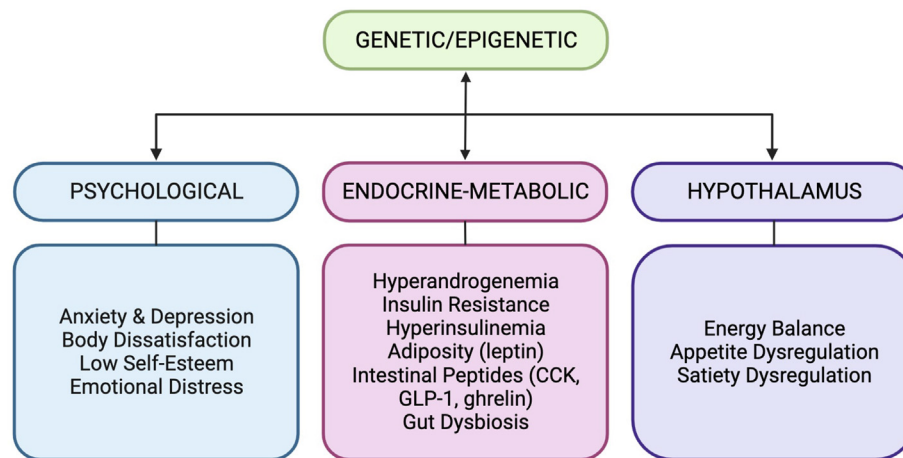


FIGURE 2. Multifactorial etiology of eating disorders in polycystic ovary syndrome. CCK, cholecystokinin; GLP-1, glucagon-like-peptide-1.

was only 3% in those with PCOS and no depression [70]. It has been proposed that those with PCOS may be highly sensitive and hyper-responsive to adrenocorticotropic hormone leading to higher production of cortisol that stimulates adrenal androgen production and related hyperandrogenic symptoms (acne, hirsutism, and hunger) [129,130]. Thus, a vicious cycle may ensue of hyperandrogenism symptoms associated with psychological distress, and further release of adrenal cortisol contributing to an exacerbation of PCOS symptoms [129]. It has been reported that individuals with PCOS can lack intrinsic motivation to undertake lifestyle changes or perform regular physical activity [58]. This lack of motivation may be associated with a vicious cycle of body weight gain, body dissatisfaction, and psychological distress, and these may contribute to barriers to implement healthy diet-lifestyle behaviors [58]. Therefore, psychological comorbidities and EDs may coexist in those with PCOS and screening for these conditions can be part of the global health assessment with appropriate referrals to psychotherapy [8,18].

Metabolic and hormonal factors increase ED risk in PCOS

Hyperandrogenemia

Androgens are considered orexigenic, stimulating appetite, disrupting impulse control, and have been associated with binge eating behaviors in those with PCOS [66,67, 97,125,131,132]. Indeed, the androgen receptor is expressed in the arcuate and ventromedial nuclei involved in energy balance and food intake [133,134]. It has been reported that BN is associated with higher serum levels of androgens independent of PCOS status [65,67, 132,135–137]; however, other studies have not found the same association [138,139]. A placebo-controlled trial found that flutamide, an androgen receptor antagonist, improved symptoms of BN, binge eating behaviors, and food cravings [140]. In another trial, an antiandrogenic oral contraceptive was shown to improve hunger and lower self-induced vomiting in those with BN [135]. The results of these trials suggest that BN may be associated with hyperandrogenism and androgen receptor mediated effects on the feeding center related to BN and BED.

Elevated testosterone in-utero may predispose individuals to PCOS and simultaneous ED development [141,142], whereas others suggest decreased ED risk [143–145]. Serum testosterone levels are reported to be higher in females with BN and lower in

females with AN [132,146]. This may help to explain why AN is not commonly reported in PCOS [28].

Other studies have hypothesized that decreased serum estrogen levels, rather than increased androgen levels, increase the risk of an ED in PCOS [69,147]. Estrogen and the estrogen receptor are considered anorexigenic, promoting satiety that may be protective against emotional eating [69,131,148,149]. Although estrogen levels may not be reduced or may be normal in PCOS, excess luteinizing hormone (LH) may reduce aromatization of androgens to estrogen in ovarian granulosa cells, and this may be associated with emotional eating [131,150,151]. Overall, the literature supports the association of hyperandrogenemia and dysregulation of hypothalamic-neuroregulation of energy balance and food intake and elevated ED scores, and these may contribute to ED risk in those with PCOS [93].

BN has also been associated with PCOS and hyperandrogenemia [131,139]. A case-control study reported that a diagnosis of PCOS occurred in 16.6% of BN cases compared with 1.7% in controls without BN, and those with BN had higher prevalence of menstrual disturbances, hirsutism, high testosterone/sex hormone-binding globulin (SHBG) ratio, and low estrogen levels [131]. Another study reported that 10 of 12 females with BN had polycystic ovary morphology; however, no difference in serum LH, follicle-stimulating hormone, testosterone, or SHBG levels was reported between those with BN and controls [139]. In a 9-year longitudinal follow-up study of 8 females originally treated for BN, all 5 females who had persistent BN also had polycystic ovaries [137]. In contrast, 0% of individuals with BN and 23% of individuals with EDNOS were found to have polycystic ovarian morphology [152]. These findings corroborate with our results that show 30% higher odds of those with PCOS having BN compared with controls [62,65]. There appears to be an association between ED, particularly BN and ovarian follicular morphology and further research to examine this relationship is warranted.

Insulin resistance

IR is common in those affected by PCOS, including lean individuals with PCOS [153]. Hyperinsulinemia can lead to postprandial hypoglycemia, which occurs when blood glucose levels fall <4 mmol/L 2–5 h after a meal. Symptoms of postprandial hypoglycemia can include mood changes, tremor, dizziness, and

hunger. Hypoglycemia can therefore cause individuals with PCOS to crave and seek out carbohydrate-rich foods and beverages [154,155]. Studies have found that $\leq 50\%$ of lean and 66% of obese individuals with PCOS have postprandial hypoglycemia, and this may contribute to mood changes and hunger [156–158].

Binge eating episodes may contribute to excess energy intake, visceral adiposity, and IR [26,159]. BED may contribute to postprandial hyperinsulinemia and contribute to IR via pancreas β -cell dysfunction and peripheral IR [160,161]. IR can result in increased free serum testosterone via decreased hepatic SHBG, and androgens may contribute to changes in eating behavior [66, 67,97,125,131–133,161]. In a study of individuals with PCOS ($n = 164$), fasting serum insulin, 2-h postprandial blood glucose, and homeostatic model assessment of IR were positively correlated with EDE-Q scores [93], whereas other studies found no association between insulin levels in patients with EDs, psychiatric disorders (mood disorders, anxiety disorders, and EDs), and PCOS [84,86]. The high prevalence of IR and reactive hypoglycemia may contribute to increased carbohydrate and food craving scores in PCOS [73,155]. Studies have suggested that primary management of PCOS should target treatment of IR rather than weight loss [59]. One study found that a low glycemic index diet and a hypocaloric diet in overweight individuals with PCOS reduced symptoms of carbohydrate cravings, hunger, and tiredness [94]. Further research is needed to test whether low glycemic index diets and other diet regimes may improve reactive hypoglycemia, carbohydrate cravings, and ED behaviors in PCOS [129,156,162–166].

IR has been linked to increased cortisol and lower central nervous system serotonin production and may contribute to risk of mood and depressive symptoms [70]. In a case–control study in those with PCOS ($n = 12$) and controls ($n = 12$) matched for age, BMI, and eating behavior, it was found that those with PCOS had more adverse mood symptoms [156].

Adiposity

An association between ED and overweight-obesity in PCOS is reported and appears to be associated with the type of ED [69,72, 88,93,96]. The prevalence of EDs in people with obesity and PCOS was 39.2%, compared with 23.2% and 13.6% in those with overweight and normal body weight with PCOS, respectively [73]. Our review results indicate that those with PCOS who are overweight are at higher risk for BED and BN. However, some studies show that EDs in PCOS are independent of BMI or visceral adiposity in normal-weight and overweight individuals. In a case–control study, binge eating symptom scores were higher in lean participants with and without PCOS [73]. Obesity independent of PCOS was shown to be associated with higher risk of binge eating (OR: 7.915, 95% CI: 3.421, 18.312) [79].

Increased adiposity may contribute to the development of EDs via impairment of the hypothalamic-neuroregulation of energy balance and food intake, and this applies to those with PCOS and overweight-obesity as we observed in several studies [67,68,88, 93,96]. The arcuate nuclei contain pro-opiomelanocortin (POMC)/cocaine amphetamine-regulated transcript (CART), neuropeptide Y (NPY)/agouti-related peptide (AGRP), and kisspeptin neurons that coregulate energy balance and the hypothalamic–pituitary–ovarian axis to impact estrogen and testosterone production and reproduction [72,167]. Adipose tissue produces adipokines, including leptin that inhibits

appetite and promotes satiety [72]. In those with higher levels of adiposity, there is often increased leptin and ensuing leptin resistance that can impair activation of anorexigenic POMC/CART signaling to inhibit food-energy intake and lack of inhibition of orexigenic pathways NPY/AGRP that promote food-energy intake, resulting in impaired satiety and this may lead to the development of BED [28,72]. In addition, hyperinsulinemia and IR commonly coexist with visceral adiposity and may further inhibit satiety signals and simultaneously promote ovarian androgen production [28].

In examining the relationship between AN and PCOS, one study found that BMI was higher in those with AN and PCOS (19.5 ± 2.5) compared with AN alone (16.9 ± 0.7) [124]. We only identified one study on atypical AN and the prevalence in PCOS was 0% compared with non-PCOS controls [28]. Diagnostic criteria of atypical AN is a more recent addition to OFSED in the DSM-5; therefore, the prevalence of atypical AN in PCOS may be underestimated [31]. AN is commonly associated with low BMI and weight loss; therefore, awareness and screening for atypical AN may not regularly occur in PCOS. It may be useful to screen for atypical AN in those with PCOS, because in atypical AN, BMI is often within or above the normal range, and this may be the case in those with PCOS [28,124].

PCOS diet, lifestyle, and weight management recommendations need to be sensitive to EDs so as not to perpetuate DE behaviors. One way to potentially achieve this is through a weight-neutral approach and a focus on health eating patterns and lifestyle behaviors [8,28,58]. Weight-neutral approaches that focus on intuitive eating, stress management, physical activity, and healthy eating, independent of body weight have been successful in improving BN in non-PCOS populations [168,169]. Research is needed to evaluate the impact of weight-neutral programs and the impact on modulation of an ED in those affected by PCOS.

Appetite dysregulation

Appetite dysregulation appears to be a contributor to ED risk in PCOS and can be associated with altered leptin and insulin metabolism, as described above. Individuals with obesity and PCOS were found to have higher energy intake and an earlier return of hunger after an ad libitum meal compared with controls with obesity [170]. A randomized controlled trial found that those with PCOS were less satiated and felt hungrier after meals compared to those without PCOS [171].

Endogenous opioids have been linked to hedonic aspects of eating and sensory reward processes, stimulating a desire for high fat and glucose foods through μ -opioid receptors within the amygdala and nucleus accumbens [97]. IR has been found to stimulate central opioid activity in PCOS, potentially contributing to higher risk for binge eating [172]. Ghrelin, a hormone that signals that the stomach is empty to promote appetite and food-seeking behavior, has been found to be associated with IR in those with PCOS [74]. Because of imbalances in ghrelin levels, those with PCOS may experience feelings of hunger leading to food-seeking behaviors and overeating [171]. On the other hand, glucagon-like peptide-1 (GLP-1), a hormone released from the ileum in response to feeding to enhance insulin secretion, also promotes satiety and reduces energy intake [97]. Impaired secretion of GLP-1 has been demonstrated in PCOS [97]. Studies support the role of GLP-1 receptor agonist medications

(such as semaglutide and liraglutide) that improve insulin sensitivity, reduce appetite, and reduce food-seeking behavior in those with PCOS [97,173,174]. Cholecystokinin (CCK) is a satiety-promoting hormone secreted in response to dietary lipid in the duodenum and has been reported to have reduced secretion in PCOS [155]. CCK stimulates gallbladder contraction, inhibits gastric emptying, and signals the hypothalamic arcuate nuclei to reduce food intake. Impaired CCK production has been observed in PCOS and may lead to decreased satiety and potentially contribute to overeating or EDs [155].

Gut microbiota dysbiosis

A growing body of evidence suggests that gut microbiota dysbiosis plays a role in the development of psychiatric disorders, obesity, IR, and PCOS [175–177]. The microbiome phenotype can affect how nutrients are absorbed and can influence the effects of peptides, CCK, GLP-1, and ghrelin [27]. It has been shown that food restriction, a characteristic of EDs, is associated with altered gut microbiota composition [175]. A Japanese study found that females with AN had significantly lower amounts of total gut bacteria and obligate anaerobes including *Clostridium coccoides*, *Clostridium leptum* subgroup, and *Bacteroides fragilis* [178]. Gut microbes can be influenced by hormonal signals that are affected by psychological stress caused by EDs [175]. At this time the mechanisms of how the microbiome is directly linked to ED development in PCOS remain unclear.

Taste perception

Altered taste perception has been proposed to contribute to ED development in PCOS; however, more studies are needed in this area. Those with PCOS were found to have lower sour, salty, and total taste scores compared with controls, and higher free androgen index was associated with lower total taste score [179]. Oral contraceptive treatment was shown to correct hyperandrogenism but did not affect total taste score. No conclusion could be drawn about taste perception and food cravings in those with PCOS and EDs, because participants with EDs were excluded from the trial [179].

Genetics and increased ED risk in PCOS

Most PCOS phenotypes do not show a clear Mendelian pattern of inheritance and the genetic loci linked to PCOS to date account for only 10% of its heritability, which is estimated at 70% [180–182]. Genes that may contribute to the risk of developing PCOS include gene variants of obesity, insulin, steroid hormone regulation, insulin signaling, and ovarian function [183]. Overall, PCOS is a polygenic and multifactorial disorder, and epigenetic modifications resulting from hormonal dysregulation of the maternal uterine environment may also contribute the development of PCOS [180,183].

Genetics may contribute to the risk of developing EDs [184]. Twin and family studies have demonstrated that heritable genetic traits related to hypothalamic-neuroregulation appetite and energy balance may play a role in BN development [71,184]. However, genetics alone may not appear to be sufficient to cause an ED, because environmental and psychological factors play a significant role in the development of EDs [72]. A large epidemiologic study found that the odds of siblings of those with PCOS with unclear ED status (both genetically male and female) having

AN, BN, and any ED were not different (OR: 0.88, 95% CI: 0.68, 1.14; OR: 1.20, 95% CI: 0.91, 1.59; OR: 0.94, 95% CI: 0.81, 1.10, respectively) [65]. However, elevated risk in female siblings was found for other psychiatric disorders, such as depressive, anxiety, and schizophrenia disorders [65]. No shared genetic link between PCOS and psychiatric disorders (depression, anxiety, schizophrenia, and bipolar disorder) has been observed; however, this study did not examine EDs [185]. Therefore, genetic predisposition to psychiatric disorders may contribute but does not appear to be the strongest predictor of possible shared etiology between PCOS and EDs.

Limitations of the review

This scoping review represents a summary of medically diagnosed EDs and those identified using different ED assessment screening tools. The findings on the prevalence of EDs in PCOS compared with controls varied and had large ranges, which may be because of the array of diagnostic and screening tools used. There was no consistent method for the assessment of EDs used in PCOS studies and we did not restrict the review to a specific type of tool to assess EDs and DE (Tables 5 and 6). The methods used to diagnose EDs have evolved as new versions of the DSM have been published by the American Psychiatric Association; therefore, our review included studies that used the DSM-III, DSM-IV, and DSM-5. Different PCOS diagnostic criteria were used in studies and this may have resulted in different prevalence rates of EDs. However, the Rotterdam diagnostic criteria was mostly used by studies. A strength of the study was inclusion of a range of tools and diagnostic criteria to assess the prevalence of an ED and this allowed us to report on the scope of EDs and DE in PCOS.

Summary

Individuals with PCOS are at high risk of EDs, particularly BED and BN. However, the degree of risk for specific subtypes of EDs remains largely unclear. The pathophysiology of EDs in PCOS involves multiple pathways including genetic, metabolic, endocrine and psychological factors [20,21,26,29]. Body weight dissatisfaction, anxiety, and depression have been identified as risk factors for ED development in those with PCOS. Furthermore, those with PCOS may have altered eating and food-seeking behaviors related to inherent hyperandrogenism. Several studies have proposed a bidirectional relationship between hyperandrogenism and hyperinsulinemia in contributing to the development of ED in PCOS, particularly BED and BN, wherein hyperinsulinemia can predispose individuals to binge eating, whereas binge eating behaviors may contribute to hyperinsulinemia, adiposity, and androgen excess [26]. Taste perception, gut microbiome dysbiosis, and genetic dysregulation of the hypothalamic-neuroregulation of energy metabolism have been hypothesized to play a role in EDs in PCOS.

Research implications

High-quality studies on the prevalence of EDs in individuals with PCOS are needed that employ large cohorts and assessment of rare ED subtypes, such as NES, atypical AN, pica, RD, and ARFID. Ideally, future studies should use clinical interviews with a registered psychologist or psychiatrist to clinically diagnose EDs [58]. Alternatively, studies could use multiple validated ED

instruments and avoid reliance on self-reported ED or DE symptoms. There are no studies assessing the development or change in ED diagnosis or prevalence over time. Furthermore, differences in prevalence based on ethnicity, gender, and impact of management strategies including counseling, diet and lifestyle, and impact of body weight discrimination on EDs in PCOS would be of interest.

Clinical implications

EDs impact quality of life and health in individuals with PCOS across the lifespan [186]. It is recommended that clinicians and healthcare providers be alert to EDs and DE, regardless of a patient's body weight, especially in the context of weight management and lifestyle interventions [8]. If an ED is detected, patients should be offered appropriate therapy through a clinical psychiatrist and/or psychologist and/or registered dietitian [8, 18]. The care of a patient with both PCOS and an ED requires specialized and comprehensive care involving a multidisciplinary team of health care providers. Although prioritizing weight loss and/or weight management in those with obesity and PCOS can improve preconception health, menstrual function, insulin sensitivity, and hyperandrogenism, it is essential to acknowledge the serious potential harm of placing emphasis on weight loss in someone with an ED or DE behaviors [8,22, 50–54]. Weight loss regimes encouraged by a clinician without follow-up care or referral to a registered dietitian may be interpreted as promotion of an ideal body weight, reinforcing feelings of despair, worsening body dissatisfaction that may increase the risk of restrictive dietary behaviors and exacerbate an ED [29]. A cautious, compassionate, weight-neutral, and self-esteem building approach may be necessary during ED treatment to prevent prioritizing achieving an arbitrary weight goal at the expense of mental health and overall wellbeing.

Conclusions

This scoping review has shown that individuals with PCOS have a high risk of developing EDs, especially BN and BED. The etiology of ED development in PCOS is not yet fully understood, but it is proposed to include psychological (body dissatisfaction, anxiety, depression), metabolic (hyperandrogenism, IR, adiposity, appetite dysregulation, gut dysbiosis), and genetic predisposition. High-quality studies are needed to rigorously assess the prevalence of EDs in individuals with PCOS and to better understand the relationship between risk factors and ED development. Early identification and management of an ED may prevent significant negative impact on quality of life in those with PCOS.

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Author contributions

The authors' responsibilities were as follows – SL-B, DV: designed the research; SL-B, MM, RM, KN, SS: conducted the research; SL-B: analyzed the data; SL-B, DV: wrote the paper; MG:

reviewed the manuscript; DV: responsible for final content; and all authors: read and approved the final manuscript.

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Conflict of interest

The authors report no conflicts of interest.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.advnut.2024.100193>.

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