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THE VICIOUS CYCLE OF POLYCYSTIC OVARY SYNDROME AND BINGE EATING DISORDER: A REVIEW OF METABOLIC, PSYCHOLOGICAL, AND IATROGENIC PATHWAYS

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ABSTRACT

Background: Polycystic Ovary Syndrome (PCOS) is a prevalent endocrine and metabolic disorder traditionally managed through a weight-centric paradigm, which frequently overlooks the significant psychiatric comorbidity of Binge Eating Disorder (BED). This narrative review aims to systematically map the interacting metabolic, psychological, and iatrogenic pathways driving BED in women with PCOS.
Methods: A comprehensive literature search was conducted across electronic databases, primarily PubMed, for peer-reviewed articles published up to May 2026. The review focused on studies examining the intersection of PCOS and eating disorders, emphasizing metabolic and neuroendocrine mechanisms, psychosocial impact, and recent clinical guidelines.
Findings: The biological susceptibility to BED in PCOS is driven by severe insulin resistance, reactive hypoglycaemia, reduced cholecystokinin (CCK) secretion linked to hyperandrogenism, and disrupted mesolimbic reward pathways. This physiological predisposition is heavily exacerbated by psychosocial distress stemming from visible hyperandrogenic symptoms, diagnostic delays, and pervasive societal and provider-induced weight stigma. Furthermore, standard clinical recommendations enforcing strict caloric restrictions often act as an iatrogenic trigger for rebound binge eating due to the profound metabolic inflexibility in these patients.
Conclusions: Dismantling the vicious PCOS-BED cycle requires an urgent shift away from punitive, weight-centric models. In alignment with the 2023 International Evidence-based Guidelines, multidisciplinary clinical management should incorporate mandatory eating disorder screening, trauma-informed cognitive-behavioural therapy (CBT), intuitive eating principles, and the evaluation of novel pharmacological agents, such as GLP-1 receptor agonists, to target the underlying pathophysiological mechanisms of hyperphagia.

KEYWORDS

Polycystic Ovary Syndrome, Binge Eating Disorder, Weight Stigma, Insulin Resistance, Intuitive Eating, GLP-1 Receptor Agonists

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1. Introduction

Polycystic Ovary Syndrome (PCOS) is universally recognized as the most prevalent metabolic endocrinopathy affecting women of reproductive age, with global prevalence rates ranging from 6% to 20% depending on the diagnostic criteria applied (Bozdag et al., 2016; Deswal et al., 2020). Characterized by a heterogeneous presentation of hyperandrogenism, ovulatory dysfunction, and polycystic ovarian morphology (PCOM), the clinical management of PCOS has traditionally focused on its reproductive and metabolic sequelae, namely infertility, type 2 diabetes, and cardiovascular risk (Escobar-Morreale, 2018). However, this traditional, weight-centric paradigm frequently overlooks a profound psychiatric comorbidity that severely compromises both treatment efficacy and patient quality of life - eating disorders, specifically Binge Eating Disorder (BED).

Recent epidemiological and clinical studies suggest that women with PCOS are at increased risk of developing disordered eating behaviours. A large meta-analysis including nearly 30,000 women diagnosed using the Rotterdam criteria reported approximately threefold higher odds of eating disorders compared with controls (OR 2.88). Notably, the strongest associations were observed for binge eating and bulimic-spectrum disorders rather than restrictive eating patterns (Cooney et al., 2024). These findings indicate that binge eating in PCOS is likely linked not only to psychological factors but also to underlying metabolic and hormonal disturbances associated with the syndrome.

The aetiology of BED in PCOS is complex and appears to be driven by interacting metabolic and psychological factors. Metabolically, profound insulin resistance and compensatory hyperinsulinemia predispose patients to reactive hypoglycaemia and disrupted satiety signalling (via dysregulated ghrelin and leptin), thereby driving a strong biological imperative to consume high-calorie, carbohydrate-dense foods (Stefanaki et al., 2024). Psychologically, the distress associated with hyperandrogenic symptoms (hirsutism, acne) and chronic weight stigma leads to severe body dissatisfaction and emotional dysregulation, establishing binge eating as a primary coping mechanism (Davitadze et al., 2023). Standard clinical management often emphasizes immediate caloric restriction. However, in patients with impaired metabolic flexibility, this approach may inadvertently encourage restrictive eating patterns that often precede binge episodes, thereby perpetuating disordered behaviours.

While the clinical overlap between these conditions is increasingly recognized in recent literature, multidisciplinary treatment approaches have not yet been widely adopted in standard practice. This review aims to systematically map the reciprocal metabolic, psychological, and iatrogenic pathways driving Binge Eating Disorder in women with PCOS, and to underscore the urgent need for weight-inclusive, trauma-informed clinical management strategies.

2. Methodology

To map the complex mechanisms interlinking PCOS and BED, a comprehensive narrative review of the literature was conducted. A systematic search strategy was applied to minimize selection bias and ensure a robust evaluation of current evidence.

2.1. Search Strategy

Electronic databases, primarily PubMed, were systematically searched for peer-reviewed articles. The search encompassed literature published from inception up to May 2026, reflecting the most recent advancements in the field. The search strategy combined Medical Subject Headings (MeSH) and free-text keywords with Boolean operators. The primary search string included: ("Polycystic Ovary Syndrome" OR "PCOS") AND ("Binge Eating Disorder" OR "BED" OR "eating disorders" OR "hyperphagia") AND ("insulin resistance" OR "appetite dysregulation" OR "cholecystokinin" OR "weight stigma" OR "body image" OR "iatrogenic").

2.2. Inclusion and Exclusion Criteria

To ensure the relevance and quality of the reviewed literature, specific inclusion and exclusion criteria were established.

- **Inclusion criteria:** (1) Peer-reviewed original research articles, systematic reviews, and meta-analyses; (2) studies specifically examining the intersection of PCOS and eating disorders, particularly BED; (3) articles detailing metabolic, neuroendocrine, or psychological mechanisms of hyperphagia in PCOS; (4) articles published in English. Special emphasis was placed on recent systematic reviews and the foundational methodology of the 2023 International Evidence-based Guideline for the Assessment and Management of Polycystic Ovary Syndrome.

• **Exclusion criteria:** (1) non-English publications; (2) conference abstracts, case reports, and non-peer-reviewed opinion pieces; (3) studies focusing primarily on restrictive eating disorders (e.g., Anorexia Nervosa) without relevance to the BED/PCOS cycle; (4) animal models, unless specifically required to explain fundamental neuroendocrine pathways (e.g., GLP-1 or CCK signalling).

2.3. Data Selection and Extraction

Titles and abstracts of the retrieved articles were initially screened for relevance to the study objectives. Following this, full-text versions of potentially eligible articles were reviewed to confirm their inclusion based on the established criteria. Priority was given to large-scale epidemiological studies, clinical trials regarding novel pharmacotherapies (e.g., GLP-1 RAs), and studies exploring the psychological impact of weight stigma and diagnostic trauma in women with PCOS.

3. Literature review

3.1. Metabolic and Neuroendocrine Pathophysiology

The biological susceptibility to binge eating in PCOS is primarily linked to multi-systemic hormonal dysregulation that affects the gut-brain axis and alters central satiety signalling. This is often conceptualized as the first phase of a two-hit hypothesis (Stefanaki et al., 2024).

3.1.1. Insulin Resistance, Reactive Hypoglycaemia, and Brain IR

Peripheral insulin resistance (IR) and compensatory hyperinsulinemia form the metabolic core of PCOS. Following carbohydrate ingestion, an exaggerated insulin response often leads to rapid glucose clearance, triggering reactive postprandial hypoglycaemia. This sudden drop in serum glucose is thought to initiate a physiological survival response, which patients experience as intense and uncontrollable cravings for simple carbohydrates.

Furthermore, hyperinsulinemia stimulates ovarian theca cells to overproduce androgens while simultaneously suppressing hepatic synthesis of sex hormone-binding globulin (SHBG). This resulting hyperandrogenism promotes abdominal adiposity, which actively inhibits insulin-stimulated glucose uptake in peripheral tissues, creating a feedback loop that progressively worsens the patient's baseline metabolic profile (Stefanaki et al., 2024).

The central nervous system undergoes pathological changes. Brain insulin resistance appears to diminish dopamine processing within the mesolimbic reward system. Patients may require larger quantities of highly palatable, energy-dense foods to achieve a normal neurochemical reward response, which clinically manifests as a drive toward binge eating behaviours (Stefanaki et al., 2024).

3.1.2. Disruption of Enteric Satiety Signals and HPA Axis Dysregulation

Normal appetite is concluded by enteric peptides like cholecystokinin (CCK), which travels via the vagus nerve to the nucleus of the solitary tract to signal fullness. In women with PCOS, elevated testosterone levels are directly associated with significantly reduced postprandial secretion of CCK (Lindén Hirschberg et al., 2004). The absence of this essential physiological satiety signal impairs the patient's perception of fullness, thereby predisposing them to consume abnormally large quantities of food and increasing the likelihood of objective binge eating episodes.

Women with PCOS frequently exhibit a hyperactive hypothalamic-pituitary-adrenal (HPA) axis due to the chronic physiological and psychological stress of the syndrome (Sarkisian et al., 2023). Elevated cortisol levels inhibit oogenesis and ovarian follicle development, while the chronic hypercortisolaemic state simultaneously suppresses dopamine release in the reward system (Kolnikaj et al., 2022). Recent neuroendocrine data suggest that stress-induced cortisol dysregulation may predispose individuals to binge eating, which serves as a compensatory mechanism to derive temporary psychological relief and reward.

3.2. Psychological Mediators and the Impact of Sociocultural Stigma

Although metabolic dysregulation establishes a physiological predisposition to hyperphagia, the secondary psychosocial distress associated with PCOS often acts as the precipitating factor that drives the development of clinical binge eating disorder (BED).

3.2.1. Hyperandrogenism, Body Dissatisfaction, and Diagnostic Trauma

The clinical manifestations of hyperandrogenism, including hirsutism, severe acne, and androgenetic alopecia, frequently co-occur with weight gain, significantly impairing the patient's self-esteem. Because these visible symptoms contrast with prevailing cultural aesthetic norms, they tend to exacerbate appearance-related distress and contribute to body image dissatisfaction (Geller et al., 2025).

This psychological distress is often compounded by delays and difficulties encountered during the diagnostic process. In a study by Gibson-Helm et al. (2017) involving over 1,300 women with PCOS, nearly half of the participants (47.1%) required consultations with three or more healthcare professionals, and one-third experienced a diagnostic delay of over two years. Furthermore, only 35.2% of respondents reported satisfaction with their diagnostic experience or the information provided. Such diagnostic barriers frequently lead to patient dissatisfaction and a perceived lack of support within the healthcare system, potentially exacerbating emotional distress and reinforcing maladaptive coping mechanisms (Gibson-Helm et al., 2017).

3.2.2. Weight Stigma

Societal weight stigma and prevailing cultural pressures surrounding weight loss significantly compound psychological distress. A recent Canadian cohort study revealed that 75% of individuals living with PCOS report experiencing weight stigma (Virk et al., 2025). Patients who experienced weight stigma were significantly more likely to report clinical depression (70% vs 50%) and eating disorders (37% vs 19%) compared to those who did not (Virk et al., 2025). It is worth noting, however, that these findings may be particularly reflective of North American clinical settings, and the prevalence of perceived stigma might differ in other cultural contexts

When these patients interact with the healthcare system, they often encounter provider-induced weight bias. A mixed-methods systematic review of 68 studies demonstrated that individuals with PCOS frequently perceive lifestyle management interventions as disproportionately focused on weight reduction and fertility, often giving less consideration to their personal motivations and long-term goals (McGowan et al., 2025). This perceived bias can exacerbate internalized stigma, thereby creating substantial psychological barriers to healthcare utilization for both the underlying endocrine disorder and the co-occurring eating pathology.

3.3. Iatrogenic Effects of Aggressive Caloric Restriction

Clinical recommendations for overweight patients with PCOS frequently enforce strict caloric deficits for weight management. However, this generalized approach can serve as an iatrogenic trigger for disordered eating patterns, especially taking into account the fact that these patients suffer from profound metabolic inflexibility (Kim et al., 2018). In a highly hyperinsulinemic state, rigid dietary restriction is compounded by elevated insulin levels, which effectively inhibit efficient lipolysis of stored adipose tissue. Although the central nervous system registers a severe energy deficit, peripheral fat stores cannot be efficiently mobilized to bridge this deficit (Jeanes & Reeves, 2017). In response to this perceived energy deprivation, the hypothalamus initiates a counter-regulatory signalling cascade. This homeostatic drive to restore energy balance overrides cognitive dietary restraint, leading to a rebound binge eating episode (Stefanaki et al., 2024).

The clinical and psychological limitations of this weight-centric paradigm are further reflected in the high attrition rates observed in lifestyle interventions. A systematic review and meta-analysis by Moran et al. (2019) demonstrated a pooled dropout rate of 47.1% in weight loss trials for premenopausal women with PCOS. Notably, baseline depressive symptoms were independently associated with a higher likelihood of treatment discontinuation. These findings suggest that prescribing rigid dietary restrictions to a psychologically vulnerable population may negatively impact treatment adherence and potentially exacerbate the psychological distress, including guilt and shame, that maintains binge eating behaviours (Moran et al., 2019).

3.4. Emerging Therapies and Updates from the 2023 International Guidelines

In response to the limitations associated with strictly weight-centric approaches, international clinical guidelines have increasingly integrated comprehensive pharmacological and behavioural strategies into the standard of care.

3.4.1. Mandatory Eating Disorder Screening and Psychotherapy

The 2023 International Evidence-based Guideline for the Assessment and Management of Polycystic Ovary Syndrome introduces significant updates to the standard clinical approach. These guidelines recommend that disordered eating behaviours be evaluated in all patients diagnosed with PCOS, irrespective of body mass index (BMI), particularly prior to the initiation of weight management or lifestyle interventions (Teede et al., 2023). Identifying specific traits that predispose individuals with PCOS to binge eating disorder (BED) is essential, as these patients often achieve favourable outcomes with targeted psychotherapies. In particular, Cognitive-Behavioural Therapy (CBT) has been shown to improve emotional regulation and reduce appearance- and weight-related distress (Jiskoot et al., 2022).

3.4.2. The Efficacy of Intuitive Eating

To address gut-brain axis dysregulation, clinical nutrition paradigms in PCOS are increasingly incorporating the principles of intuitive eating. This approach emphasizes responsiveness to internal hunger and satiety cues rather than adherence to rigid, externally imposed dietary rules. Evidence demonstrates that higher baseline intuitive eating scores, as well as subsequent increases in these scores over time, are associated with a 74% reduction in the odds of engaging in binge eating behaviours among women diagnosed with PCOS (Hazzard et al., 2021). While these longitudinal results are promising, the implementation of intuitive eating in PCOS requires careful guidance, as severe insulin resistance may initially distort a patient's internal hunger and satiety cues.

3.4.3. The Emerging Role of Novel Pharmacotherapy (GLP-1 RAs and Inositol)

From a pharmacological perspective, addressing the underlying pathophysiological mechanisms of hunger is essential. Glucagon-like peptide-1 receptor agonists (GLP-1 RAs), such as semaglutide and liraglutide, represent an important therapeutic development in this context. GLP-1 RAs target both metabolic and neurobehavioral pathways by reducing hyperandrogenaemia and modulating mesolimbic reward circuits in the brain. By regulating dopamine signalling within these reward networks, these agents can effectively reduce the frequency and severity of objective binge eating episodes (Baranowska-Bik, 2022; Tongta et al., 2025).

Additionally, alternative agents like inositol are gaining interest. While current PCOS guidelines consider inositol experimental specifically for fertility therapies, earlier targeted psychiatric trials have demonstrated that high-dose inositol can be highly therapeutic for patients with bulimia nervosa and binge eating, working in parallel to pathways targeted by selective serotonin reuptake inhibitors (SSRIs) (Concerto et al., 2023). This highlights the need for broader pharmacological tools that address both metabolic and psychiatric pathways simultaneously.

4. Conclusions

Current evidence suggests that the relationship between PCOS and Binge Eating Disorder is influenced by both biological and psychological factors, rather than a mere coincidental comorbidity. The vicious cycle is initiated by severe metabolic derangements, such as insulin resistance, brain reward dysfunction, hyperandrogenism, and blunted CCK signalling, that drive an overwhelming physiological urge to binge eat. This underlying biological vulnerability is further exacerbated by the psychological distress stemming from visible hyperandrogenism, pervasive weight stigma, and potential iatrogenic harm from rigid caloric restrictions. The 2023 International Evidence-based Guidelines mandate a long-overdue shift away from punitive weight-centric models. Only through a multidisciplinary approach—incorporating intuitive eating, evaluating novel pharmacological agents (like GLP-1 RAs and inositol), and prioritizing trauma-informed cognitive behavioural care—can healthcare providers effectively dismantle the PCOS-BED cycle. Future prospective longitudinal studies are urgently needed to fully map the chronological onset of BED in newly diagnosed PCOS patients. Additionally, while the off-label use of GLP-1 RAs shows promise, large-scale randomized controlled trials specifically targeting the PCOS-BED phenotype are required to establish long-term safety and efficacy.

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