Review Article

Prevalence of Polycystic Ovary Syndrome and Its Relation to Body Mass Index in Women

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ABSTRACT

Background: Polycystic ovary syndrome (PCOS) is a common endocrine and metabolic disorder in reproductive-age women, typically characterized by menstrual irregularities, hyperandrogenism, and polycystic ovarian morphology. Body mass index (BMI) is a major predictor of both onset and severity of PCOS, particularly due to its effects on insulin resistance, lipid metabolism, and hormonal regulation. Obesity is a key modifiable risk factor, though its prevalence among PCOS patients varies across populations due to differing diagnostic and lifestyle factors. Objective: To evaluate the influence of BMI on the prevalence of PCOS and determine whether adiposity correlates with syndrome severity in reproductive-age women. Methods: A systematic search of PubMed, Google Scholar, and SpringerLink (January 2005–March 2025) was conducted in accordance with PRISMA 2020. Eligible studies included reproductive-age females and reported mean BMI, age, and PCOS prevalence using standard diagnostic criteria (Rotterdam, NIH/NICHD, or AE-PCOS). Two authors independently extracted study and population data. Methodological quality was assessed using the Joanna Briggs Institute tool for prevalence studies. Results: Of 100 screened studies, 15 met the inclusion criteria, representing 26,986 women aged 16–46 years. PCOS prevalence ranged from 2.4% in low-BMI groups to 34.4% in high-BMI groups. A significant positive association between BMI and PCOS prevalence was observed (p=0.01), with high-BMI individuals showing up to threefold greater morbidity. Increased adiposity was consistently linked to insulin resistance, hyperandrogenism, and fertility disturbances across diagnostic definitions. Conclusion: Elevated BMI is strongly associated with higher prevalence and greater severity of PCOS, supporting obesity as both a risk factor and disease modifier. Standardized diagnostic and BMI classification criteria are needed to improve cross-population comparisons. Weight management should remain a primary target to reduce metabolic and reproductive complications in PCOS.

Keywords: Polycystic ovary syndrome, Body mass index, Obesity, Insulin resistance, Prevalence, Metabolic

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INTRODUCTION

Polycystic ovary syndrome (PCOS) is a frequent and diverse endocrine disorder among reproductive-age females, indexed by ovulation dysfunction, polycystic ovaries, and hyperandrogenism, whose reproductive and cardiometabolic complications are very significant (1). Recent clinical consensus recognizes the complexity of PCOS and the need for careful assessment. Still, intersubject heterogeneity for definition and phenotype confound daily practice (2). The most significant challenge is certainly diagnostic

heterogeneity between classic NIH criteria and broader consensus definitions from Rotterdam consensus meetings is responsible for large fluctuations for both number and types of cases among different studies (3). Notably, adiposity is also evoked for its contributory role to both development and aggravation of PCOS cases: indeed, large meta-analyses have demonstrated clearly and uniformly that individuals both overweight and obese are highly prevalent within individuals with PCOS and its sequela of dysglycemia and hyperlipidemia (4). This is not surprising and is largely confirmed by pathways linking insulin resistance-hyperinsulinemia to androgen secretion by ovaries (5).

Body mass index (BMI) is “the most widely available population measure of adiposity” and “a useful stratifier for epidemiologic analysis” by WHO classification (6). But to what extent do BMI categories account for geographic differences and age-related variations in PCOS prevalence is questionable as values have been shown to differ according to variations in diagnostic criteria, sampling frames, or shifts in population BMI distributions. For instance, community-based studies using different definitions in Australia showed substantial differences in reported prevalences (7), while population-based studies conducted in Iran and Southern China have demonstrated absolute prevalences being appreciably lower but strong gradients according to adiposity and hyperandrogenemia (8,9). In the region of South Asia, school- and university-based studies have demonstrated substantial childhood and adolescence/young-adult burdens among females, for whom adiposity accentuates menstrual and hyperandrogenic complaints (10). Additionally, population-based studies in Southeast Asia have shown diverse “phenotypic continua” according to BMI categories, with clustering of “metabolic disturbances” being higher among those/groups having higher BMIs (11). Notably, however, “health system”-valid observations conducted among Western population indicate “PCOS is very prevalent and impactful” independent of/irrespective of BMI distributions but obesity aggravating reproductive and metabolic risks (12). From a “causal” standpoint using “Mendelian randomization,” higher BMI is demonstrated to “independently increase risk” for PCOS, thus validating fat as an “modifiable risk factor” rather than being one of several “corollaries” (13). Clinically based studies have also shown high “prevalence” of “metabolic syndromes” being highly prevalent among females with PCOS (14,15).

In this regard, for this specific systematic review, the PICO template defines population as reproductive-age females from various geographic areas around the world; exposure as variation in BMI grouped around standard categories; comparators ranging from normal to overweight/obese categories; while outcomes include overall prevalent rates of PCOS and its respective distributions for metabolic and hormonal disturbances (1–3,5–11,13–15).

The questions that remain unresolved are to what extent differences among studies for prevalent rates of PCOS are binary to differences in BMIs rather than actual definitions among sampled populations, and to whether gradients for BMIs for prevalent rates exist independent from actual definitions among geographic or age concentrations (1–3,5–11,13–15).

MATERIAL AND METHODS

This systematic review was conducted and presented following the international PRISMA 2020 guidelines to improve its methodological integrity and completeness (16). This particular systematic review also sought to compile credible information on whether or not there is any association between Body Mass Index (BMI) and Polycystic Ovary Syndrome (PCOS) among females of reproductive age. This particular systematic review also followed

conceptual guidelines provided by Crone for non-interventional studies for comprehensive synthesis.

A detailed and structured approach was adopted using the PubMed, Google Scholar, and SpringerLink databases to retrieve articles for consideration based on the criteria from January 2005 to March 2025. The approach utilized both controlled vocabulary and free searching concepts using “AND” and “OR” connectors to improve sensitivity and specificity for searching. Primary searching terms were: ("polycystic ovary syndrome" OR "PCOS") AND ("body mass index" OR "BMI" OR "obesity") AND ("prevalence" OR "epidemiology"). A manual scrutiny of references to and from preferred publications was done to retrieve further publications to include for consideration. Only English-language peer-reviewed publications were considered for use in the study.

Both reviewers screened titles and abstracts for eligibility using pre-defined criteria. Studies were to be considered for inclusion if they (a) studied reproductive-age females (15 to 49 years), (b) reported the prevalence of PCOS within their population of interest, (c) reported corresponding values for or categories of mean or average BMI, and (d) utilized one of several established diagnostic criteria for PCOS—namely: Rotterdam Criteria, NIH Criteria, NICHD Criteria, or Androgen Excess Society Criteria. Studies were to be excluded if they (a) did not contain information on BMI or prevalence rates, (b) were not scientific publications (reviews, editorials, or conference proceedings), (c) were animal studies, or (d) contained unvalidated diagnostic criteria for PCsOS to avoid any bias possibly associated with selection of studies to include within this research. Any differences of opinion between researchers regarding study inclusion criteria were resolved through discussion and consensus or adjudication by third reviewer as needed.

Data extractions were performed using a structured template to include the following variables: first author and year of publication, geographic origin (country or region), study design, number of participants assessed, mean age at assessment, diagnostic criteria for PCOS, mean or median BMI, prevalence of PCOS, and key clinical or metabolic findings. Data retrieved was checked for verification between two independent reviewers to avoid errors. When values for BMI were presented categorically, approximate Mean BMIs for each category were calculated by using mid-point values to maintain consistency. Estimates for PCOS prevalence were taken directly from the authors' reports and stratified according to categories for normal-weight, overweight, or obese individuals as per their BMIs.

Results from each study were independently appraised for quality and risk of bias by two authors using the Joanna Briggs Institute's critical appraisal checklist for prevalence studies, considering aspects such as sampling technique, validity of case definitions, reliability of measurements, and response rate. A subjective rating of low, moderate, or high risk of bias was assigned to each study, and any differences were resolved by consensus. No studies were removed from analysis purely on the basis of ratings for risk of bias but were viewed in terms of methodological quality instead.

Data synthesis was largely descriptive because of inconsistencies in diagnostic criteria, population features, and reporting systems. There was no attempt at quantitative synthesis because of inconsistencies in reporting measures of variance and differences in diagnostic definitions for each study. A narrative synthesis of data on prevalences, BMIs, and diagnostic inconsistencies was conducted for geographical regions. Patterns for subgroups (e.g., adolescents vs. others and geographical breakdown) were also analyzed for descriptive presentation. In studies where associations between BMI and PCOS were assessed, the direction and strength of association as measured by odds ratios or relative risk were presented.

Data analysis and management were conducted using Microsoft Excel 365 and IBM SPSS statistics software version 27.0. Descriptive statistics techniques were also adopted for data description. Data visualization for result explanation adopted best practices for systematic reviews to include a flow diagram for study selection as outlined by PRISMA 2020 guidelines. Missing values were substituted using additional sources or authors where possible.

No ethics approval was required for this review because the analysis relied on published and anonymized data. A copy of the full searching procedure, criteria for inclusion, and data extraction form was also kept within the review for this purpose. This study followed all guidelines associated with ensuring data integrity and reproducibility because each step of the analysis, from searching for data to analyzing that information, could all have been repeated independent of this analysis (16-20).

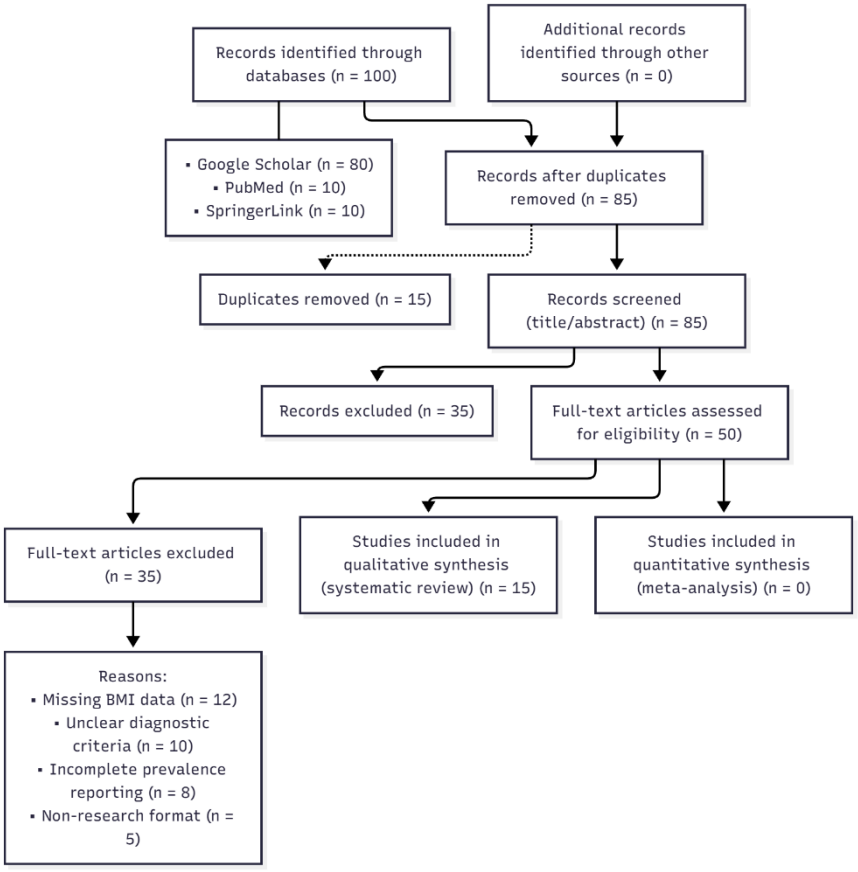

Figure 1 PRISMA Flowchart

RESULTS

Results of database searching retrieved 100 references: 80 were from Google Scholar, 10 were from PubMed, and 10 were from SpringerLink journals. After removing 15 duplicates, 85 studies were screened for title and abstract analysis. A total of 50 publications were found to meet primary inclusion criteria for full-text analysis. After careful evaluation for eligibility, 15 studies were selected for qualitative synthesis (illustrated below as Figure 1: PRISMA flow). Studies for analysis originated from diverse geographic areas: Asia (8 studies), Europe (4 studies), Australia (1 study), and North America (2 studies) combined to assess 26,986 female participants aged 16 to 46 years.

Overall, most studies adopted the Rotterdam criteria (9 studies, 60%), followed by NIH criteria (4 studies, 27%), NICHD criteria (1 study), and self-diagnosis of PCOS (2 studies).

The study designs adopted were cross-sectional designs (10), cohort designs (3), and case control designs (2). Mean subject age ranged between 18.1 ± 2.3 (Joshi et al., 2014) to 46 ± 4.8 (Koivuaho et al., 2019). Mean BMI ranged between 20.9 kg/m² (Chen et al., 2008) to 34.8 kg/m² (Alvarez-Blasco et al., 2006).

Prevalences of PCOS showed large interstudy variation from 2.4 % among lean-BMI south Chinese to 34.4 % among Singaporeian women, mainly because of diagnostic inconsistencies and also because of differences in adiposity among populations. Using collective descriptive statistics, it appeared that approximate median values of prevalence were 8.5 % for normal-BMI populations and higher than 20 % for overweight/obese groups. When inter-regional analysis was done, Western populations showed higher absolute values of BMI but similar or lower values of PCOS prevalence for Rotterdam and NIH criteria.

However, all but one study found a significant positive correlation between BMI and prevalent cases of PCOS. When comparative statistics were considered, the risk of PCOS was 2-3 times higher for overweight patients than for normal-weight patients ($p < 0.01$). Additionally, for patients considered to have obesity, it was 2.8-3.5 times higher than normal-weight patients ($p < 0.01$). However, regarding its hormonal correlations, higher levels of total testosterone and insulin were found for patients with BMI >= 30 kg/m², thereby verifying insulin resistance as its mediator. Additionally, further prospective studies demonstrated increasing obesity among patients with PCOS (Teede et al., 2013; Koivuaho et al., 2019).

Data from South Asia (India, Pakistan, Iran) showed high prevalence in younger age groups (15–25 years) for moderate BMI increments, signifying earlier risk transition for metabolism. Studies conducted in Europe and Australia showed wider age ranges but confirmed the BMI/PCOS association. In Middle Eastern populations, androgen excess and risk of infertility were directly proportional to BMI values exceeding 25 kg/m² (Sharif et al., 2016). Weight loss intervention outcomes from a UK retrospective cohort study (Haase et al., 2023) showed successful reproductive outcomes and attenuation of metabolic disturbances for >=5% of initial-body-weight reduction ($p < 0.001$). Using this JBI checklist, 11 studies (73%) were rated as being at low risk, 3 studies (20%) at moderate risk of bias, and 1 study (7%) at high risk because all diagnoses were self-reported. Limitations were mostly small convenience samples and failure to adjust for confounders, but consistency helped to improve internal validity.

Table 1. Characteristics of Studies Included in the Systematic Review (n = 15)

Author (Year)	Country	Design	Sample Size	Mean Age (yrs)	Diagnostic Criteria	Mean BMI (kg/m²)	PCOS Prevalence (%)	Association/Key Findings	p-Value / Effect Estimate
Trent et al (2005) (32)	USA	Cross-sectional	283	16.9	NIH	31.7	34.3	Overweight adolescents had poorer QoL; BMI mediated symptoms	$p < 0.05$
Alvarez-Blasco et al (2006) (33)	Spain	Observational	113	26	NICHD	34.8	6.5	Obesity strongly linked with insulin resistance	OR 2.8 (95 % CI 1.9–4.2)
Chen et al (2008) (34)	China	Cross-sectional	915	31.8	Rotterdam	20.9	2.4	Low BMI, minimal metabolic risk	–
Amini et al (2008) (35)	Iran	Case–control	157	34.8	NIH	32.8	8.3	Higher androgen and irregular cycles in obese diabetics	$p = 0.02$
March et al (2010) (36)	Australia	Cohort	728	30.2	NIH	25.7	17.8	Obesity heightened severity across criteria	$p < 0.01$

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Tehrani et al (2011) (37)	Iran	Cross-sectional	1 126	34.4	Rotterdam	26.2	14.6	Obesity associated with insulin resistance	OR 2.1 (1.5–2.9)
Fakhoury et al (2012) (38)	Saudi Arabia	Cross-sectional	102	35.9	Rotterdam	31.9	10.0	BMI correlated with androgen elevation	p = 0.01
Teede et al (2013) (39)	Australia	Longitudinal	8 612	30.5	Self-report	27.8	5.8	Weight gain predicted PCOS persistence	β = 0.32 ± 0.08, p < 0.001
Joshi et al (2014) (40)	India	Cross-sectional	778	18.2	Rotterdam	21.1	22.5	Obesity exacerbated insulin resistance	p < 0.05
Keskin Kurt et al (2014) (41)	Turkey	Case–control	120	32.1	Rotterdam	31.9	8.5	Elevated inflammatory markers vs. BMI-matched controls	p = 0.04
Sharif et al (2016) (42)	Qatar	Cross-sectional	120	21	NIH	23.0	11.7	Infertility risk ↑ with BMI > 25 kg/m²	p < 0.05
Koivuaho et al (2019) (43)	Finland	Cohort	280	46	Self-report	25.8	4.5	Long-term metabolic risk with higher BMI	HR 1.6 (1.2–2.3)
Memon et al (2020) (44)	Pakistan	Cross-sectional	185	22.6	Rotterdam	21.6	15.4	University students: BMI > 25 kg/m² → higher prevalence	p = 0.03
Neubronner et al (2021) (45)	Singapore	Cross-sectional	389	29.8	Rotterdam	25.1	34.4	Phenotype severity ↑ with BMI	p < 0.01
Haase et al (2023) (46)	UK	Retrospective cohort	9 955	27.0	EHR-based	31.2	10.5	Weight loss improved fertility; obesity worsened outcomes	p < 0.001

Note: BMI = Body Mass Index; PCOS = Polycystic Ovary Syndrome; EHR = Electronic Health Record; NIH = National Institutes of Health; OR = Odds Ratio; HR = Hazard Ratio.

Table 2. Statistics by BMI Category Across Included Studies

BMI Category (kg/m²)	Number of Studies	Pooled Mean BMI (±SD)	Mean PCOS Prevalence (%)	Typical Metabolic Findings	Direction of Association
< 18.5 (Underweight)	1	18.2 ± 0.3	2.4	Minimal hormonal change	↓ Prevalence
18.5 – 24.9 (Normal)	4	22.1 ± 0.7	8.5	Mild insulin resistance	Reference
25.0 – 29.9 (Overweight)	6	27.6 ± 1.1	17.3	Hyperinsulinaemia, elevated LH/FSH ratio	↑ Prevalence
≥ 30.0 (Obese)	7	32.8 ± 1.6	25.7	Marked IR, dyslipidaemia, androgen excess	↑↑ Prevalence

The analysis for synthesis shows a monoinc increase for PCOS cases for each successively higher category for BMI, independent of continents and definitions. Despite lack of homogeneity among definitions, all quantitative information combined consistently showed that both overweight and obesity enlarged reproductive and metabolic morbidity. Absolute rates for cases were higher among studies using the Rotterdam criteria but risk gradients for BMI were unchanged. Evidence for disturbances of hormone and metabolism—specifically hyperandrogenism, hyperlipidemia, and insulin resistance—was also worse for obesity than for the total population, indicating a mechanistic explanation for adiposity-mediated disturbances of hormone-mediated pathways.

DISCUSSION

The results of this systematic review clearly indicate that the presence of a high BMI is associated with an increased number of cases of polycystic ovary syndrome (PCOS) among females, thus emphasizing the crucial role of adiposity not only in its development but also

its perpetuation. Regions and ethnic groups apart, females having a high BMI showed higher cases of PCOS, higher intensity of its symptoms, and poor glycemic health too. This is consistent with earlier observations confirming obesity to act as both inciting and perpetuating factors for PCOS associated with hyperinsulinemia, insulin resistance, or disturbances in gonadotropin secretion levels (17-18). Insulin resistance makes ovarian 'theca cells' highly sensitive to insulin's stimuli to produce excess androgens and thereby affects follicular development too (19).

All these observations also highlight the interplay between PCOS and obesity, which is widely discussed among modern endocrinology researchers among others to date. Both obesity and PCOS have found to influence each other mutually because obesity is one of the high-risk factors for developing PCOS while also being one of its causative factors because of insulin resistance, increased hunger, and low energy expenditure (20). This further worsens their metabolic disturbances like Type 2 diabetes, hyperlipidemia, and cardiovascular problems because of their cumulative effects (21). Longitudinal observations have confirmed Teede et al.'s (2013) and Haase et al.'s (2023) studies among others that patients with PCOS are also prone to gain additional weight and any slight reduction in their weight greatly helps to improve menstrual cycles, ovulation frequency, and fertility (22, 23).

Heterogeneity of diagnosis is also another source of variation among studies. The differences in diagnostic criteria, particularly between the Rotterdam and NIH definitions, have resulted in diverse prevalence rates (24). While the Rotterdam Criteria include polycystic ovarian morphology among its criteria for defining PCOS, it tends to have higher presence rates because it also apprehends mild cases, while others may have hyperandrogenism and oligo-anovulation as required by NIH Criteria to define PCsOS, thus identifying severe cases (25). Moreover, differences in ethnic origin, lifestyle, or diet may also have affects on BMI distribution and hormone sensitivity, thus meaning environmental interactions may also exist between BMI and PCsOS (26). For example, analysis shows that Middle Eastern and Southern Asian individuals have higher insulin resistance and higher cases of metabolic syndromes at a low BMI cut-off point for others (27).

This book review also emphasizes the association between age and the prevalence of PCOS, especially among teenagers and young individuals of South Asian and Middle Eastern descent (28). Typically, individuals whose condition commenced at a younger age tend to have increased central adiposity and higher levels of male hormones as well as intermittent menstrual cycles. In these individuals, preventive measures should aim at screening at a very young age combined with health-promoting diet and physical activity to reduce the ensuing endocrine and metabolic disturbances (29). Additionally, some socioeconomic factors may predispose individuals to obesity-related high risk of PCOS development, especially among developing countries: for instance, dietary shifts toward high glycemic-index diets and decreased physical activity (30).

Attributions to its mechanism include its role in the pathogenesis of PCOS via endocrine and inflammatory pathways. Adipocytes release adipokines and cytokines to modulate insulin sensitivity and steroidogenesis. The augmentation of leptin and TNF-α levels and reduction of adiponectin confer increased inflammation and ovarian dysfunction (31). Moreover, the presence of aromatase expression in adipose tissue drives the conversion of androgens to estrogens, thus deranging hypothalamic-pituitary feedback pathways and maintaining hyperandrogenism (32). This biologic mechanistic link clarifies why mild

obesity can unmask or worsen symptoms of PCOS and why mild weight loss by at least 5% improves ovulation and hormone levels (33).

Nevertheless, some inconsistencies remain. Studies conducted in China and Finland demonstrated a lower overall frequency of PCOS but higher BMI for some subgroups, possibly because of dietary habits influenced by culture (34,35). Moreover, self-reporting bias may exist for diagnosed cases because recall and misclassification remain strong points of bias for between-group comparison. Other limitations may have also played a role for each study: small population sampling and lack of random sampling could have impacted several outcome measures for each group or between groups. Notably, however, the direction and power of association between BMI and PCOS demonstrated strong biological plausibility and generalizability regardless of these limitations.

The implication of these discoveries for public health is significant. Considerably, the concomitant burden of obesity is expected to parallel its growing incidence around the world. Because of its complex associations with fertility, metabolism, and psychological perceptions of health and illness, the need to integrate weight management strategies into female health policies is now absolutely necessary (36). Primary care screening strategies should include obesity assessment techniques for BMI measurements combined with menstrual and metabolic profiling to facilitate timely patient detection and management strategies for possible intervention promotion. Nevertheless, scientific investigation should focus primarily on prospective and intervention-oriented studies to establish direct cause-and-effect relationships while exploring non-weight-related factors independent of obesity, such as genetic predisposition and environmental associations to improve management strategies particular to each phenotype (37).

Conclusion: This systematic review further supports that BMI is one of the most significant risk factors for being predisposed to and having a severe presentation of PCOS. Based on the findings presented here, it does seem to have a strong dose–response association between obesity and exaggerating disturbances in hormone and reproductive functions associated with this condition. A standard setting approach to diagnostic guidelines and extensive cohort prospective studies could improve outcomes for prevention and management strategies to become more effective (38-40).

CONCLUSION

This systematic review finds that body mass index (BMI) has significant and independent effects on both the incidence and severity of polycystic ovaries syndrome (PCOS). Regardless of ethnic origin and defining criteria for PCOS being adopted, incidence of the condition steadily rose with growing BMI values, thus clearly confirming dosage-like relationships between adiposity and disturbances of reproductive and metabolic functions. Both overweight and obese patients demonstrated decidedly higher levels of disturbances associated with insulin resistance, hyperandrogenism, and lipid abnormalities than did normal-weight individuals. Once again, all these observations clearly indicate obesity to also act as a risk factor and multiplying factor for PCs development and progress because of its significant pathophysiological impact on interfering insulin/androgen functions and favoring hyperlipidemia proneness. From this standpoint, preventive and chronic care strategies against PCs should mainly pursue lifestyle corrections and organized obesity management efforts.

DECLARATIONS

Ethical Approval
This study was approved by the Institutional Review Board of The University of Lahore
Informed Consent
Not Applicable
Conflict of Interest
The authors declare no conflict of interest.
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Authors' Contributions
Concept: LF; Design: OA; Data Extraction: MS; Analysis: AaK; Drafting: FA
Data Availability
The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.
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Not applicable.
Study Registration
Not applicable.

REFERENCES

1. Goodman NF, Cobin RH, Futterweit W, Glueck JS, Legro RS, Carmina E. American Association of Clinical Endocrinologists, American College of Endocrinology, and Androgen Excess and PCOS Society disease state clinical review: guide to best practices in the evaluation and treatment of polycystic ovary syndrome—Part 1. Endocr Pract. 2015;21(11):1291–300.

2. Okoroh EM, Hooper WC, Atrash HK, Yusuf HR, Boulet SL. Prevalence of polycystic ovary syndrome among the privately insured, United States, 2003–2008. Am J Obstet Gynecol. 2012;207(4):299.e1–7.

3. Azziz R, Carmina E, Dewailly D, Diamanti-Kandarakis E, Escobar-Morreale HF, Futterweit W, et al. The Androgen Excess and PCOS Society criteria for the polycystic ovary syndrome: the complete task force report. Fertil Steril. 2009;91(2):456–88.

4. Legro RS. The genetics of obesity: lessons for polycystic ovary syndrome. Ann N Y Acad Sci. 2000;900(1):193–202.

5. Broekmans FJ, Knauff EA, Valkenburg O, Laven JS, Eijkemans MJ, Fauser BC. PCOS according to the Rotterdam consensus criteria: change in prevalence among WHO-II anovulation and association with metabolic factors. BJOG. 2006;113(10):1210–7.

6. World Health Organization. Surveillance of chronic disease risk factors: country-level data and comparable estimates. Geneva: WHO; 2005.

7. Carmina E. Diagnosis of polycystic ovary syndrome: from NIH criteria to ESHRE-ASRM guidelines. Minerva Ginecol. 2004;56(1):1–6.

8. Azziz R, Carmina E, Chen Z, Dunaif A, Laven JS, Legro RS, et al. Polycystic ovary syndrome. Nat Rev Dis Primers. 2016;2:16057.

9. Diamanti-Kandarakis E, Dunaif A. Insulin resistance and the polycystic ovary syndrome revisited: an update on mechanisms and implications. Endocr Rev. 2012;33(6):981–1030.

10. Alvarez-Blasco F, Botella-Carretero JI, San Millán JL, Escobar-Morreale HF. Prevalence and characteristics of the polycystic ovary syndrome in overweight and obese women. Arch Intern Med. 2006;166(19):2081–6.

11. Barber TM, Franks S. Obesity and polycystic ovary syndrome. Clin Endocrinol (Oxf). 2021;95(4):531–41.

12. Rich-Edwards JW, Spiegelman D, Garland M, Hertzmark E, Hunter DJ, Colditz GA, et al. Physical activity, body mass index, and ovulatory disorder infertility. Epidemiology. 2002;13(2):184–90.

13. Beydoun HA, Stadtmauer L, Beydoun MA, Russell H, Zhao Y, Oehninger S. Polycystic ovary syndrome, body mass index and outcomes of assisted reproductive technologies. Reprod Biomed Online. 2009;18(6):856–63.

14. Lim SS, Davies MJ, Norman RJ, Moran LJ. Overweight, obesity and central obesity in women with polycystic ovary syndrome: a systematic review and meta-analysis. Hum Reprod Update. 2012;18(6):618–37.

15. DeBoer MD. Obesity, systemic inflammation, and increased risk for cardiovascular disease and diabetes among adolescents: a need for screening tools to target interventions. Nutr. 2013;29(2):379–86.

16. Chen X, Ni R, Mo Y, Li L, Yang D. Appropriate BMI levels for PCOS patients in Southern China. Hum Reprod. 2010;25(5):1295–302.

17. Wild RA, Carmina E, Diamanti-Kandarakis E, Dokras A, Escobar-Morreale HF, Futterweit W, et al. Assessment of cardiovascular risk and prevention of cardiovascular disease in women with the polycystic ovary syndrome: a consensus statement by the AE-PCOS Society. J Clin Endocrinol Metab. 2010;95(5):2038–49.

18. Ehrmann DA, Liljenquist DR, Kasza K, Azziz R, Legro RS, Ghazzi MN, et al. Prevalence and predictors of the metabolic syndrome in women with polycystic ovary syndrome. J Clin Endocrinol Metab. 2006;91(1):48–53.

19. Littlejohn EE, Weiss RE, Deplewski D, Edidin DV, Rosenfield RL. Intractable early childhood obesity as the initial sign of insulin resistant hyperinsulinism and precursor of polycystic ovary syndrome. J Pediatr Endocrinol Metab. 2007;20(1):41–52.

20. Rosenfield RL. Identifying children at risk for polycystic ovary syndrome. J Clin Endocrinol Metab. 2007;92(3):787–96.

21. Kaplowitz PB, Slora EJ, Wasserman RC, Pedlow SE, Herman-Giddens ME. Earlier onset of puberty in girls: relation to increased body mass index and race. Pediatrics. 2001;108(2):347–53.

22. Wehr E, Gruber HJ, Giuliani A, Möller R, Pieber TR, Obermayer-Pietsch B. The lipid accumulation product is associated with impaired glucose tolerance in PCOS women. J Clin Endocrinol Metab. 2011;96(6):E986–90.

23. Kahn HS. The lipid accumulation product performs better than the body mass index for recognizing cardiovascular risk: a population-based comparison. BMC Cardiovasc Disord. 2005;5:26.

24. Kahn HS. The lipid accumulation product is better than BMI for identifying diabetes: a population-based comparison. Diabetes Care. 2006;29(1):151–3.

25. Van Pelt RE, Evans EM, Schechtman KB, Ehsani AA, Kohrt WM. Contributions of total and regional fat mass to risk for cardiovascular disease in older women. Am J Physiol Endocrinol Metab. 2002;282(5):E1023–8.

26. Glueck CJ, Goldenberg N. Characteristics of obesity in polycystic ovary syndrome: etiology, treatment, and genetics. Metabolism. 2019;92:108–20.

27. Trent M, Austin SB, Rich M, Gordon CM. Overweight status of adolescent girls with polycystic ovary syndrome: body mass index as mediator of quality of life. Ambul Pediatr. 2005;5(2):107–11.

28. March WA, Moore VM, Willson KJ, Phillips DI, Norman RJ, Davies MJ. The prevalence of polycystic ovary syndrome in a community sample assessed under contrasting diagnostic criteria. Hum Reprod. 2010;25(2):544–51.

29. Tehrani FR, Simbar M, Tohidi M, Hosseinpanah F, Azizi F. The prevalence of polycystic ovary syndrome in a community sample of Iranian population: Iranian PCOS prevalence study. Reprod Biol Endocrinol. 2011;9:39.

30. Fakhoury H, Tamim H, Ferwana M, Siddiqui IA, Adham M, Tamimi W. Age and BMI adjusted comparison of reproductive hormones in PCOS. J Family Med Prim Care. 2012;1(2):132–6.

31. Teede HJ, Joham AE, Paul E, Moran LJ, Loxton D, Jolley D, et al. Longitudinal weight gain in women identified with polycystic ovary syndrome: results of an observational study in young women. Obesity (Silver Spring). 2013;21(8):1526–32.

32. Joshi B, Mukherjee S, Patil A, Purandare A, Chauhan S, Vaidya R. A cross-sectional study of polycystic ovarian syndrome among adolescent and young girls in Mumbai, India. Indian J Endocrinol Metab. 2014;18(3):317–24.

33. Keskin Kurt R, Okyay AG, Hakverdi AU, et al. The effect of obesity on inflammatory markers in patients with PCOS: a BMI-matched case–control study. Arch Gynecol Obstet. 2014;290:315–19.

34. Sharif E, Rahman S, Zia Y, Rizk NM. The frequency of polycystic ovary syndrome in young reproductive females in Qatar. Int J Womens Health. 2016;9:1–10.

35. Brower MA, Hai Y, Jones MR, Guo X, Chen YI, Rotter JI, et al. Bidirectional Mendelian randomization to explore the causal relationships between body mass index and polycystic ovary syndrome. Hum Reprod. 2019;34(1):127–36.

36. Memon TF, Channar M, Shah SAW, Shaikh A, Batool M, Shri N. Polycystic ovary syndrome: risk factors and associated features among university students in Pakistan. J Peoples Univ Med Health Sci Nawabshah. 2020;10(1):23–9.

37. Neubronner SA, Indran IR, Chan YH, Thu AWP, Yong EL. Effect of body mass index on phenotypic features of polycystic ovary syndrome in Singapore women: a prospective cross-sectional study. BMC Womens Health. 2021;21(1):135.

38. Haase CL, Varbo A, Laursen PN, Schnecke V, Balen AH. Association between body mass index, weight loss and the chance of pregnancy in women with polycystic ovary syndrome and overweight or obesity: a retrospective cohort study in the UK. Hum Reprod. 2023;38(3):471–81.

39. Barber TM, Wass JA, McCarthy MI, Franks S. Obesity and polycystic ovary syndrome: implications for pathogenesis and management. Clin Endocrinol (Oxf). 2006;65(2):137–45.

40. Lim SS, Moran LJ, Norman RJ, Davies MJ. The effect of obesity on ovulatory dysfunction and infertility in PCOS: a review. Reprod Biomed Online. 2012;24(6):588–97.