International Journal of Advanced Research in Biological Sciences
ISSN: 2348-8069
www.ijarbs.com
(A Peer Reviewed, Referred, Indexed and Open Access Journal)
DOI: 10.22192/ijarbs Coden: IJARQG (USA) Volume 12, Issue 12-2025

Review Article

DOI: http://dx.doi.org/10.22192/ijarbs.2025.12.12.012

# Lifestyle and Metabolic Dimensions of Polycystic Ovarian Disease among Indian Women: An Integrative Review

***Rajaiah Vailla[1], Swamy Uppula[1] and Shamitha Gangupanthula[2]**

[1]Department of Zoology, Kakatiya Government College, Hanumakonda, (T.S), India
[1]Department of Zoology, University College Kakatiya University, Warangal, (T.S), India
*Corresponding Author: *rajaiahvailla@gmail.com*

## Abstract

Polycystic Ovarian Disease (PCOD) has emerged as a major reproductive and metabolic health concern among women of childbearing age in India. The increasing incidence of this condition is closely associated with profound lifestyle changes accompanying urbanization, including altered dietary patterns, reduced physical activity, and heightened psychosocial stress. Although frequently conflated with Polycystic Ovary Syndrome (PCOS), PCOD is predominantly a functional ovarian disorder characterized by reversible metabolic and hormonal disturbances. This review critically examines the current understanding of PCOD pathophysiology, with particular emphasis on insulin resistance, androgen excess, and ovarian dysfunction. It further analyzes the contribution of lifestyle behaviours, environmental exposures, and socio-cultural factors to the growing prevalence of PCOD in the Indian population. Beyond its clinical manifestations, the review highlights the broader implications of PCOD on mental health, fertility, quality of life, and economic productivity. Recognising the unique socio-cultural context of Indian women, this article advocates for cost-effective, culturally sensitive, and sustainable preventive and therapeutic strategies. Lifestyle modification, community awareness, and early intervention are underscored as essential components in mitigating the expanding burden of PCOD in India.

**Keywords:** Polycystic Ovarian Disease, Indian women, lifestyle factors, insulin resistance, metabolic dysfunction, hyperandrogenism, reproductive health, urbanisation, preventive strategies.

## Objectives

This review sets out to explore the growing occurrence of Polycystic Ovarian Disease (PCOD) among women in India. It will delve into the biological, lifestyle, and socio-environmental factors that play a role in its development. Additionally, it aims to clarify the differences between PCOD and Polycystic Ovary Syndrome (PCOS) while assessing practical prevention and management strategies tailored to the Indian socio-cultural landscape.

## 1. Introduction

Polycystic ovary syndrome (PCOS) represents a complex and heterogeneous endocrine disorder that extends beyond isolated reproductive dysfunction. Although its clinical features have been widely described, the underlying mechanisms driving disease onset and progression remain incompletely understood. The primary aim of this review is to examine the physiopathological basis of PCOS by bringing together current evidence from endocrinology, metabolism, and inflammation research.

Specifically, this review focuses on the dysregulation of the hypothalamic–pituitary–ovarian axis, excess androgen production, and abnormalities in gonadotropin secretion, which together form the endocrine foundation of PCOS. In addition, metabolic disturbances such as insulin resistance, hyperinsulinaemia, dyslipidaemia, and altered glucose homeostasis are examined for their contributory role in amplifying hormonal imbalance and disease severity (Dunaif, 1997; Diamanti-Kandarakis & Dunaif, 2012).

Emerging evidence also indicates that chronic low-grade inflammation is a key component of PCOS pathophysiology, influencing both endocrine and metabolic processes and contributing to long-term complications (González, 2012). By integrating these interconnected pathways, this review seeks to provide a comprehensive and cohesive understanding of how endocrine, metabolic, and inflammatory mechanisms collectively drive the clinical manifestations and systemic consequences of PCOS.

The purpose of this review is not only to summarise existing knowledge but also to highlight unresolved questions and research gaps within these interrelated domains. By adopting an integrated, multidisciplinary perspective, the review aims to support future research efforts and inform more effective diagnostic and therapeutic strategies for PCOS management.

Polycystic Ovary Syndrome (PCOS) is one of the most common reproductive endocrine disorders affecting women of reproductive age worldwide. It is characterized by distinctive ovarian morphological features, notably the presence of multiple small, immature follicles arranged along the ovarian periphery. Globally, PCOS affects approximately 10–13% of women during their reproductive years, making it a significant public health concern (Azziz et al., 2004). Clinically, the disorder disrupts normal menstrual cyclicity, frequently presenting as oligomenorrhea or secondary amenorrhea. In more severe cases, PCOS contributes to infertility, recurrent pregnancy loss, and adverse reproductive outcomes, thereby substantially impairing women's reproductive health and quality of life (Balen et al., 2016).

The condition was first described in 1935 by Stein and Leventhal, who identified a characteristic triad of amenorrhea, hirsutism, and polycystic ovaries; consequently, the disorder is also known as Stein–Leventhal syndrome (Stein & Leventhal, 1935). Subsequent research has demonstrated that PCOS is a heterogeneous and multifactorial disorder resulting from complex interactions among endocrine, metabolic, and inflammatory pathways.

The pathophysiology of PCOS involves dysregulation of the hypothalamic–pituitary–ovarian (HPO) axis, marked by altered gonadotropin-releasing hormone (GnRH) pulsatility and increased secretion of luteinizing hormone (LH) from the pituitary gland (Marshall

& Eagleson, 1999). These hormonal disturbances promote excessive ovarian androgen production, which represents a hallmark feature of PCOS. Hyperandrogenism manifests clinically as acne, hirsutism, and androgenic alopecia and is a key diagnostic criterion of the disorder (Azziz et al., 2009). Concurrently, insulin resistance is highly prevalent among women with PCOS and plays a pivotal role in disease progression by intensifying hyperandrogenism and disrupting glucose metabolism (Dunaif, 1997).

Insulin resistance reduces cellular responsiveness to insulin, resulting in compensatory hyperinsulinemia, weight gain, and an increased risk of obesity. These metabolic abnormalities markedly elevate the likelihood of developing type 2 diabetes mellitus in individuals with PCOS (Legro et al., 1999). Beyond reproductive dysfunction, PCOS is increasingly recognized as a systemic disorder associated with long-term nonreproductive complications, including cardiovascular disease (Wild et al., 2011), dyslipidemia, and metabolic syndrome (Diamanti-Kandarakis & Dunaif, 2012). Additionally, chronic low-grade inflammation contributes to disease severity by exacerbating endocrine and metabolic imbalance (González, 2012).

Despite considerable advances in understanding the molecular and clinical features of PCOS, its precise pathogenesis remains incompletely elucidated. This review aims to integrate findings from both basic and clinical research to provide a comprehensive overview of the physiological and pathological mechanisms underlying PCOS. Specifically, it examines endocrine alterations such as HPO axis dysfunction and elevated levels of anti-Müllerian hormone (AMH), androgens, and prolactin, along with metabolic abnormalities including insulin resistance and lipid dysregulation. Furthermore, the role of inflammatory mediators in the development and progression of PCOS is evaluated.

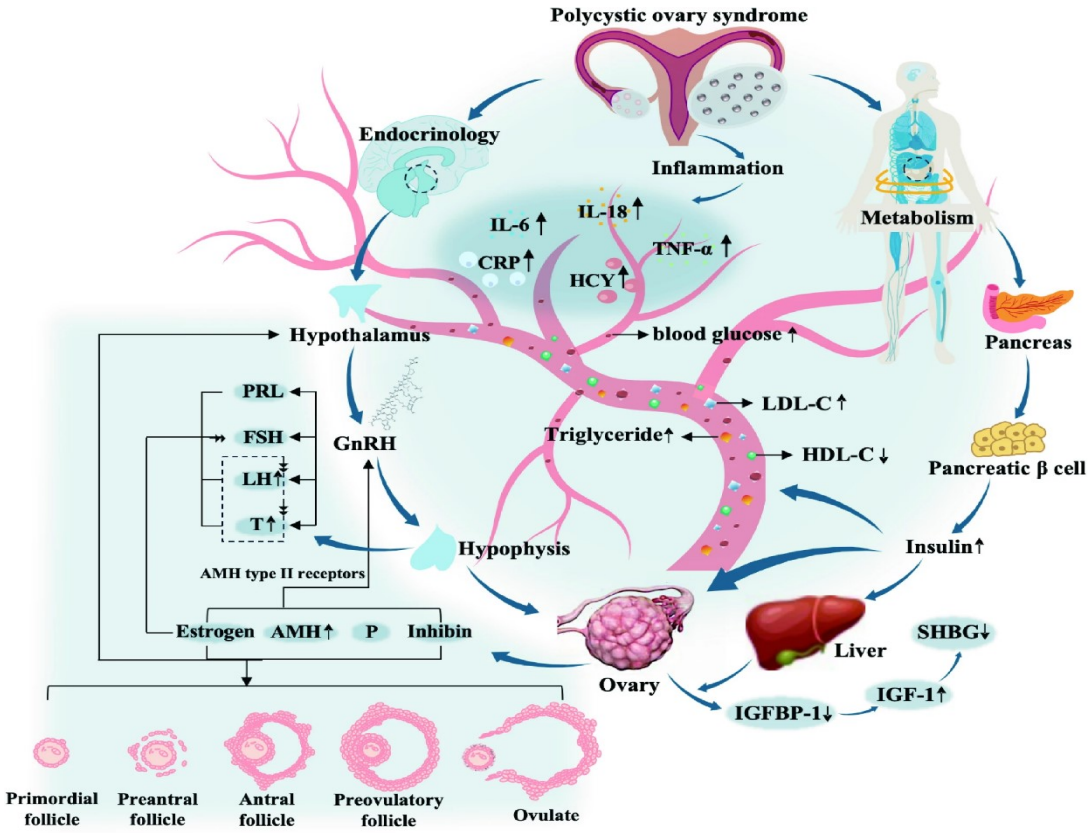


**Fig 1. Physiopathological mechanisms involved in polycystic ovary syndrome**

By clarifying the interconnected nature of endocrine, metabolic, and inflammatory pathways, this review highlights how these mechanisms collectively drive the clinical manifestations and complications of PCOS. In addition, existing knowledge gaps and unresolved questions in the fields of endocrinology, metabolism, and immunology are identified, and future research directions are proposed. Emphasising interdisciplinary collaboration, this review underscores the importance of integrated approaches combining endocrinology, metabolomics, and immunology to advance a holistic understanding of PCOS pathogenesis. Integrated overview of the physiopathology of polycystic ovary syndrome (PCOS), highlighting the complex interactions between endocrine dysfunction, metabolic abnormalities, and inflammatory processes. Dysregulation of the hypothalamic–pituitary–ovarian axis results in altered gonadotropin secretion and excess androgen production, leading to impaired follicular development and ovulatory dysfunction. Metabolic disturbances, particularly insulin resistance and hyperinsulinaemia, further exacerbate androgen excess and contribute to obesity, dyslipidaemia, and glucose intolerance. Concurrently, chronic low-grade inflammation acts as a reinforcing factor, intensifying hormonal and metabolic imbalance. The convergence of these pathways underlies the reproductive, metabolic, and systemic manifestations of PCOS.(Fig. 1).

## 2 Explain Endocrine Dysregulation in PCOS:

Normal female reproductive function is regulated by the coordinated activity of the hypothalamic–pituitary–ovarian (HPO) axis. This finely balanced endocrine system ensures regular menstrual cyclicity, ovarian follicle development, ovulation, and appropriate hormonal secretion necessary for fertility and overall reproductive health.The hypothalamus initiates this regulatory process by releasing gonadotropin-releasing hormone (GnRH) in a pulsatile manner. The frequency and amplitude of GnRH pulses are critical, as they determine the secretion of gonadotropins from the anterior pituitary gland, namely luteinising hormone (LH) and follicle-stimulating hormone (FSH). FSH plays a central role in stimulating the growth and maturation of ovarian follicles, while LH supports androgen synthesis by theca cells and triggers ovulation during the mid-cycle surge (Guyton & Hall, 2016).Within the ovary, follicular development progresses through a series of well-defined stages, from primordial follicles to preovulatory follicles. Granulosa cells convert androgens to oestrogens through aromatase activity under the influence of FSH, leading to rising oestrogen levels during the follicular phase of the menstrual cycle. Increasing oestrogen concentrations exert feedback effects on the hypothalamus and pituitary, initially inhibiting and later stimulating gonadotropin release to generate the LH surge required for ovulation (Yen & Jaffe, 1998).

Ovulation marks the release of a mature oocyte from the dominant follicle, followed by formation of the corpus luteum, which secretes progesterone during the luteal phase. Progesterone prepares the endometrium for potential implantation and exerts negative feedback on GnRH, LH, and FSH secretion to regulate the length and stability of the menstrual cycle. In the absence of pregnancy, declining progesterone and oestrogen levels lead to menstruation and initiation of a new cycle.The integrity of this hormonal feedback system is essential for normal ovarian function. Any disruption in GnRH pulsatility, gonadotropin secretion, or ovarian steroidogenesis can impair follicular development and ovulation. Understanding this normal endocrine framework provides a crucial baseline for interpreting the hormonal and ovarian abnormalities observed in polycystic ovary syndrome.

Discuss abnormalities in GnRH pulsatility, luteinising hormone hypersecretion, follicle-stimulating hormone imbalance, and excess androgen production. Explain how these endocrine changes impair follicular maturation and lead to ovulatory dysfunction.

## 3: Metabolic Alterations in Polycystic Ovary Syndrome

Metabolic dysfunction is a central component of the pathogenesis of polycystic ovary syndrome and plays a critical role in the progression and severity of the disorder. Among these abnormalities, insulin resistance and the resulting hyperinsulinaemia are the most consistently observed features, affecting both lean and overweight women with PCOS.

Insulin resistance in PCOS is characterised by reduced responsiveness of peripheral tissues, particularly skeletal muscle and adipose tissue, to circulating insulin. To maintain normal glucose levels, pancreatic β-cells increase insulin secretion, leading to chronic hyperinsulinaemia. Elevated insulin concentrations have direct and indirect effects on ovarian function, including stimulation of androgen production by ovarian theca cells and suppression of hepatic synthesis of sex hormone-binding globulin, thereby increasing the bioavailability of circulating androgens (Dunaif, 1997; Diamanti-Kandarakis & Dunaif, 2012).

This insulin-driven amplification of androgen excess contributes to the persistence of hyperandrogenic features and disrupts normal follicular development. In addition, insulin resistance promotes weight gain and central adiposity, which further aggravate metabolic and hormonal imbalance. Excess adipose tissue alters adipokine secretion and lipid metabolism, contributing to dyslipidaemia characterised by elevated triglycerides and reduced high-density lipoprotein cholesterol levels (Wild et al., 2011).

The metabolic consequences of PCOS extend beyond reproductive health. Prolonged insulin resistance and impaired glucose tolerance significantly increase the risk of developing type 2 diabetes mellitus at an earlier age compared with women without PCOS (Legro et al., 1999). These metabolic disturbances also predispose affected individuals to cardiovascular complications and metabolic syndrome, reinforcing the view of PCOS as a systemic disorder rather than a condition limited to ovarian dysfunction.

Collectively, insulin resistance, hyperinsulinaemia, obesity, and lipid abnormalities form a self-perpetuating cycle that intensifies both metabolic and endocrine dysfunction in PCOS. Understanding these interrelated metabolic alterations is essential for effective disease management and for reducing long-term health risks associated with the syndrome.

## 4: Inflammatory Mechanisms in Polycystic Ovary Syndrome

Increasing evidence indicates that polycystic ovary syndrome is associated with a state of chronic low-grade inflammation, which plays a significant role in disease pathogenesis and progression. This inflammatory milieu is present in both obese and lean women with PCOS, suggesting that inflammation is an intrinsic component of the disorder rather than merely a consequence of excess adiposity.

Women with PCOS commonly exhibit elevated circulating levels of inflammatory markers such as C-reactive protein, tumour necrosis factor-α, interleukin-6, and interleukin-18. These mediators interfere with insulin signalling pathways, thereby aggravating insulin resistance and promoting compensatory hyperinsulinaemia (González, 2012). Impaired insulin action further enhances ovarian androgen production, reinforcing endocrine imbalance and disrupting normal follicular development.

Inflammatory cytokines also exert direct effects on ovarian tissue. They influence steroidogenesis, folliculogenesis, and oocyte quality by altering the local ovarian microenvironment. Chronic exposure to inflammatory mediators may impair granulosa cell function and contribute to follicular arrest, a characteristic feature of polycystic ovaries (Repaci et al., 2011). In addition,

inflammation-induced oxidative stress further exacerbates cellular dysfunction and hormonal irregularities.

The interaction between inflammation and metabolic dysfunction creates a self-perpetuating cycle in PCOS. Adipose tissue dysfunction leads to altered secretion of adipokines, such as decreased adiponectin and increased leptin, which promote pro-inflammatory states and worsen insulin resistance. Over time, this sustained inflammatory burden contributes to the development of long-term complications, including cardiovascular disease, endothelial dysfunction, and increased susceptibility to type 2 diabetes mellitus (Diamanti-Kandarakis et al., 2006).

Taken together, chronic low-grade inflammation acts as a critical link between endocrine and metabolic abnormalities in PCOS. Understanding the inflammatory component of the syndrome is essential for identifying novel therapeutic targets and for developing comprehensive management strategies aimed at reducing both reproductive and metabolic complications.

## 5: Integration of Endocrine, Metabolic, and Inflammatory Pathways

Polycystic ovary syndrome represents a multifactorial disorder in which endocrine, metabolic, and inflammatory pathways are closely interconnected, creating a self-perpetuating cycle that exacerbates clinical manifestations. Dysregulation of the hypothalamic–pituitary–ovarian (HPO) axis leads to excessive luteinising hormone (LH) secretion and hyperandrogenism, which impairs follicular development and ovulation (Marshall & Eagleson, 1999). Elevated androgen levels also influence adipose tissue distribution, promoting central obesity and altering adipokine secretion.

Metabolic dysfunction, particularly insulin resistance and compensatory hyperinsulinaemia, amplifies endocrine disturbances by stimulating ovarian androgen production and reducing sex hormone-binding globulin levels, thereby increasing free circulating androgens (Dunaif, 1997; Diamanti-Kandarakis & Dunaif, 2012). This endocrine–metabolic interplay contributes to menstrual irregularities, anovulation, and hyperandrogenic symptoms such as hirsutism and acne.

Chronic low-grade inflammation further reinforces this cycle. Pro-inflammatory cytokines, including tumour necrosis factor-α and interleukin-6, impair insulin signalling and exacerbate insulin resistance, which in turn intensifies hyperandrogenism (González, 2012). Inflammation also disrupts ovarian function directly by affecting granulosa and theca cell activity, thereby contributing to follicular arrest and impaired oocyte quality (Repaci et al., 2011).

The convergence of these pathways produces systemic consequences beyond reproductive dysfunction, including dyslipidaemia, impaired glucose tolerance, and increased cardiovascular risk. Figure 1 illustrates this integrated model, highlighting how endocrine, metabolic, and inflammatory mechanisms interact synergistically to drive both ovarian pathology and broader metabolic complications in PCOS. Understanding these interconnections is essential for developing holistic treatment strategies that target multiple aspects of the syndrome simultaneously.

## 6: Clinical Manifestations and Complications of PCOS

The complex interplay between endocrine, metabolic, and inflammatory pathways in polycystic ovary syndrome (PCOS) is directly reflected in its diverse clinical features. Disruption of the hypothalamic–pituitary–ovarian axis and hyperandrogenism lead to characteristic reproductive abnormalities. Women with PCOS often present with menstrual irregularities, including oligomenorrhea or amenorrhea, due to impaired follicular maturation and anovulation (Balen et al., 2016). Hyperandrogenism manifests as hirsutism, acne, androgenic alopecia, and in

some cases, seborrhoea, significantly impacting quality of life and psychological well-being (Azziz et al., 2009).

Infertility is a common consequence of PCOS, arising from chronic anovulation and altered ovarian morphology. Women with PCOS may also experience recurrent pregnancy loss, further highlighting the reproductive implications of the syndrome (Fauser et al., 2012). Beyond reproductive health, metabolic abnormalities such as insulin resistance, hyperinsulinaemia, and central obesity contribute to the development of dyslipidaemia, impaired glucose tolerance, and type 2 diabetes mellitus (Dunaif, 1997; Legro et al., 1999).

Chronic low-grade inflammation further amplifies these metabolic disturbances and increases the risk of cardiovascular complications. Women with PCOS often exhibit endothelial dysfunction, hypertension, and a pro-inflammatory lipid profile, which collectively heighten long-term cardiovascular risk (Wild et al., 2011). Metabolic syndrome is observed with increased prevalence among women with PCOS, reflecting the systemic nature of the disorder and its wide-ranging health implications (Diamanti-Kandarakis & Dunaif, 2012).

In summary, the clinical presentation of PCOS encompasses reproductive, dermatological, metabolic, and cardiovascular manifestations. Understanding the mechanistic links between endocrine dysfunction, metabolic disturbances, and inflammation provides critical insight into the diverse clinical features and long-term complications of the syndrome. This holistic understanding is essential for designing targeted diagnostic and therapeutic interventions.

## 7: Future Research Directions in Polycystic Ovary Syndrome

Despite significant advances in understanding polycystic ovary syndrome (PCOS), several aspects of its pathophysiology and clinical management remain inadequately elucidated.

Future research should prioritise an interdisciplinary approach that integrates endocrinology, metabolomics, immunology, and clinical sciences to generate a comprehensive understanding of the syndrome.From an endocrinological perspective, further studies are required to elucidate the precise mechanisms regulating hypothalamic–pituitary–ovarian axis dysregulation and androgen excess. Investigating the molecular pathways governing ovarian steroidogenesis and gonadotropin dynamics may reveal novel targets for therapeutic intervention (Legro et al., 2013).

Metabolomics offers an opportunity to characterise metabolic alterations in PCOS more accurately. High-throughput profiling of metabolites in serum, urine, and follicular fluid could improve early diagnosis, identify biomarkers of disease severity, and clarify the relationship between insulin resistance, obesity, and hyperandrogenism (Zhang et al., 2020).

Immunological studies should aim to define the role of chronic low-grade inflammation and oxidative stress in disease progression. Understanding how inflammatory cytokines interact with endocrine and metabolic pathways could inform the development of anti-inflammatory or immunomodulatory therapies (Repaci et al., 2011).

Additionally, translational and clinical research is essential to evaluate the effectiveness of personalised treatment strategies, including lifestyle interventions, pharmacological agents, and assisted reproductive technologies. Longitudinal cohort studies across diverse populations, including region-specific studies such as those in India, are required to assess the influence of genetics, environment, and socio-cultural factors on PCOS prevalence and outcomes (Azziz et al., 2016).Finally, interdisciplinary collaborations combining endocrinology, metabolism, immunology, and clinical expertise are critical to advance holistic understanding and management of PCOS. Such approaches may facilitate the development of precision medicine strategies, improve diagnostic

criteria, and optimise prevention and therapeutic interventions, ultimately reducing the long-term reproductive and metabolic complications of the syndrome.

## Conclusion

Polycystic ovary syndrome (PCOS) is a multifactorial disorder arising from the intricate interplay of endocrine, metabolic, and inflammatory pathways. Dysregulation of the hypothalamic–pituitary–ovarian axis, hyperandrogenism, insulin resistance, and chronic low-grade inflammation collectively contribute to the reproductive, metabolic, and systemic manifestations of the syndrome. These interconnected mechanisms not only drive clinical features such as menstrual irregularities, hyperandrogenic symptoms, and infertility but also predispose affected women to long-term complications, including type 2 diabetes mellitus, cardiovascular disease, and metabolic syndrome.

A comprehensive understanding of PCOS requires an integrated approach that considers the convergence of hormonal, metabolic, and inflammatory processes. Future research should adopt interdisciplinary strategies, combining endocrinology, metabolomics, immunology, and clinical sciences, to elucidate unresolved pathophysiological mechanisms and to identify novel diagnostic markers and therapeutic targets.

Effective management of PCOS must therefore extend beyond reproductive health, encompassing lifestyle interventions, pharmacological therapies, and personalised approaches tailored to metabolic and inflammatory profiles. By advancing a holistic understanding of the syndrome, such strategies can improve both short-term clinical outcomes and long-term health, ultimately reducing the burden of PCOS on women worldwide.

## References

Azziz, R., Carmina, E., Dewailly, D., Diamanti-Kandarakis, E., Escobar-Morreale, H. F., Futterweit, W., Janssen, O. E., Legro, R. S., Norman, R. J., Taylor, A. E., & Witchel, S. F. (2009). The Androgen Excess and PCOS Society criteria for the polycystic ovary syndrome. The Journal of Clinical Endocrinology & Metabolism, 94(2), 456–459. https://doi.org/10.1210/jc.2008-2642

Azziz, R., Woods, K. S., Reyna, R., Key, T. J., Knochenhauer, E. S., & Yildiz, B. O. (2004). The prevalence and features of the polycystic ovary syndrome in an unselected population. The Journal of Clinical Endocrinology & Metabolism, 89(6), 2745–2749. https://doi.org/10.1210/jc.2003-032046

Azziz, R., Woods, K. S., Reyna, R., Key, T. J., Knochenhauer, E. S., & Yildiz, B. O. (2016). The prevalence and features of the polycystic ovary syndrome in an unselected population. The Journal of Clinical Endocrinology & Metabolism, 89(6), 2745–2749.

Balen, A. H., Morley, L. C., Misso, M., Franks, S., Legro, R. S., Wijeyaratne, C. N., Stener-Victorin, E., Fauser, B. C. J. M., & Norman, R. J. (2016). The management of polycystic ovary syndrome: An update. Human Reproduction, 31(12), 261–271. https://doi.org/10.1093/ humrep/dew255

Diamanti-Kandarakis, E., & Dunaif, A. (2012). Insulin resistance and the polycystic ovary syndrome revisited: An update on mechanisms and implications. Endocrine Reviews, 33(6), 981–1030. https://doi.org/10.1210/er.2011-1034

Diamanti-Kandarakis, E., Paterakis, T., Kandarakis, H., & Panidis, D. (2006). Indices of low-grade chronic inflammation in polycystic ovary syndrome and the beneficial effect of metformin. Human Reproduction, 21(6), 1426–1431.

Dunaif, A. (1997). Insulin resistance and the polycystic ovary syndrome: Mechanism and implications for pathogenesis. Endocrine Reviews, 18(6), 774–800. https://doi.org/10.1210/ edrv.18.6.0318

Fauser, B. C., Tarlatzis, B. C., Rebar, R. W., Legro, R. S., Balen, A. H., Lobo, R.,

Carmina, E., Chang, J., Yildiz, B. O., Laven, J. S., Boivin, J., Petraglia, F., & Hodgen, G. D. (2012). Consensus on women's health aspects of polycystic ovary syndrome (PCOS): The Amsterdam ESHRE/ASRM-Sponsored 3rd PCOS Consensus Workshop Group. Fertility and Sterility, 97(1), 28–38.

González, F. (2012). Inflammation in polycystic ovary syndrome: Underpinning of insulin resistance and ovarian dysfunction. Steroids, 77(4), 300–305. https://doi.org/10.1016/j.steroids.2011.12.003

Guyton, A. C., & Hall, J. E. (2016). Textbook of medical physiology (13th ed.). Elsevier.

Legro, R. S., Arslanian, S. A., Ehrmann, D. A., Hoeger, K. M., Murad, M. H., Pasquali, R., & Welt, C. K. (2013). Diagnosis and treatment of polycystic ovary syndrome: An Endocrine Society clinical practice guideline. The Journal of Clinical Endocrinology & Metabolism, 98(12), 4565–4592.

Legro, R. S., Kunselman, A. R., Dodson, W. C., & Dunaif, A. (1999). Prevalence and predictors of risk for type 2 diabetes mellitus and impaired glucose tolerance in polycystic ovary syndrome. The Journal of Clinical Endocrinology & Metabolism, 84(1), 165–169. https://doi.org/10.1210/jcem.84.1.5393

Marshall, J. C., & Eagleson, C. A. (1999). Neuroendocrine aspects of polycystic ovary syndrome. Endocrine Reviews, 20(3), 351–378. https://doi.org/10.1210/edrv.20.3.0368

Repaci, A., Gambineri, A., & Pasquali, R. (2011). The role of low-grade inflammation in the polycystic ovary syndrome. Molecular and Cellular Endocrinology, 335(1), 30–41.

Stein, I. F., & Leventhal, M. L. (1935). Amenorrhea associated with bilateral polycystic ovaries. American Journal of Obstetrics and Gynecology, 29, 181–191.

Wild, R. A., Rizzo, M., Clifton, S., & Carmina, E. (2011). Lipid levels in polycystic ovary syndrome: Systematic review and meta-analysis. Fertility and Sterility, 95(3), 1073–1079. https://doi.org/10.1016/j.fertnstert.2010.12.027

Yen, S. S. C., & Jaffe, R. B. (1998). Reproductive endocrinology: Physiology, patho-physiology, and clinical management (4th ed.). Saunders.

Zhang, H., Wu, X., Sun, H., Xu, W., & Lu, X. (2020). Metabolomic insights into polycystic ovary syndrome: Pathophysiology, diagnosis and therapy. Frontiers in Endocrinology, 11, 574.

| Access this Article in Online | |
|---|---|
| Quick Response Code | Website: www.ijarbs.com |
| | Subject: Pathophysiology |
| DOI:10.22192/ijarbs.2025.12.12.012 | |

How to cite this article:
Rajaiah Vailla, Swamy Uppula and Shamitha Gangupanthula. (2025). Lifestyle and Metabolic Dimensions of Polycystic Ovarian Disease among Indian Women: An Integrative Review. Int. J. Adv. Res. Biol. Sci. 12(12): 123-131.
DOI: http://dx.doi.org/10.22192/ijarbs.2025.12.12.012