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Review Article

A Comprehensive Review of Polycystic Ovary Syndrome (PCOS): Pathophysiology, Diagnosis and Management

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A B S T R A C T

Polycystic Ovary Syndrome (PCOS) is one of the most prevalent endocrine and metabolic disorders affecting women of reproductive age worldwide. It is characterized by hyperandrogenism, ovulatory dysfunction, and polycystic ovarian morphology. Beyond reproductive abnormalities, PCOS is strongly associated with insulin resistance, obesity, dyslipidemia, and chronic low-grade inflammation, predisposing affected individuals to type 2 diabetes mellitus, cardiovascular disease, and endometrial hyperplasia. The pathophysiology of PCOS is multifactorial, involving genetic predisposition, neuroendocrine dysregulation, metabolic disturbances, and environmental influences. Diagnosis is primarily based on the Rotterdam consensus criteria. Management strategies include lifestyle modification, pharmacological therapy tailored to symptom profile, and fertility-directed interventions where required. This review comprehensively summarizes the epidemiology, mechanisms, clinical manifestations, diagnostic criteria, therapeutic approaches, and emerging trends in PCOS management.

Keywords: Polycystic Ovary Syndrome, Hyperandrogenism, Insulin Resistance, Anovulation, Metabolic Syndrome, Infertility

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INTRODUCTION:

Polycystic Ovary Syndrome (PCOS) is a complex endocrine disorder affecting approximately 6–20% of women of reproductive age, depending on diagnostic criteria used. It is characterized by excessive androgen production, ovulatory dysfunction, and polycystic ovarian morphology.

PCOS is no longer considered merely an ovarian disorder; rather, it is recognized as a systemic metabolic condition. Insulin resistance plays a central role in disease progression and contributes to long-term metabolic complications including type 2 diabetes and cardiovascular disease [1].

The etiology of PCOS is multifactorial, involving genetic susceptibility combined with environmental factors such as sedentary lifestyle, dietary imbalance, and obesity. Understanding these mechanisms is essential for effective long-term management [2].

Polycystic Ovary Syndrome (PCOS), commonly referred to as PCOD in certain regions, is a complex endocrine disorder affecting women of reproductive age.

Polycystic Ovarian Disease (PCOD) is a hormonal disorder commonly seen in women of reproductive age. It is characterized by irregular menstrual cycles, excess androgen levels, and the presence of multiple small cysts in the ovaries. The condition often leads to problems such as infertility, weight gain, acne, and excessive hair growth, and is strongly linked to insulin resistance and metabolic disturbances. PCOD is considered a multifactorial disorder influenced by genetic, lifestyle, and environmental factors, and it carries long-term health risks including diabetes and cardiovascular disease[3].

Table: 1 Difference between PCOD and PCOS

Aspect	PCOD (Polycystic Ovarian Disease)	PCOS (Polycystic Ovary Syndrome)
Nature	Condition where ovaries release immature or partially mature eggs that form cysts	A broader hormonal and metabolic syndrome with systemi
Severity	Relatively common and milder; often manageable with lifestyle changes	More serious and complex; linked to long-term health risks
Hormonal Imbalance	Present, but usually less severe	More pronounced, with higher androgen levels
Symptoms	Irregular periods, mild infertility, weight gain, acne, hair growth	Chronic anovulation, infertility, obesity, hirsutism, r disturbances
Health Risks	Limited; usually does not cause major complications if managed	Associated with diabetes, cardiovascular disease, and en cancer
Management	Lifestyle modification and simple medications often sufficient	Requires comprehensive treatment including hormonal the metabolic management
Impact on Fertility	Fertility usually preserved with proper management	Greater risk of infertility due to chronic anovulation

In short, PCOD is a milder ovarian condition, while PCOS is a more complex syndrome with systemic metabolic and hormonal consequences.

Metabolic and reproductive health are intricately connected, with disturbances in one system often manifesting in the other. Conditions such as obesity, insulin resistance, and diabetes disrupt hormonal regulation, leading to menstrual irregularities, infertility, and adverse pregnancy outcomes. Conversely, reproductive disorders like polycystic ovary syndrome (PCOS) not only impair fertility but also predispose individuals to long-term metabolic complications, including metabolic syndrome and cardiovascular disease. This bidirectional relationship underscores the importance of integrated approaches to prevention, diagnosis, and management in both reproductive and metabolic health[4].

Epidemiology

Polycystic Ovary Syndrome (PCOS) is one of the most common endocrine disorders affecting women of reproductive age worldwide. The estimated global prevalence ranges between 6% and 20%, depending on the diagnostic criteria applied and the population studied.Variability in prevalence is largely attributed to differences in diagnostic definitions such as the National Institutes of Health (NIH), Rotterdam, and Androgen Excess Society criteria. Studies suggest that the Rotterdam criteria tend to identify a higher number of cases due to broader diagnostic inclusion[5].

In India, the reported prevalence varies between 9% and 22%, with higher rates observed in urban populations compared to rural areas, likely due to sedentary lifestyle patterns, dietary habits, and increasing obesity rates.Adolescents and young women between 15 and 35 years represent the most affected demographic group. Familial clustering has also been observed, indicating a significant genetic component in disease susceptibility[6].

Recent epidemiological data further indicate a rising trend in PCOS incidence, correlating with increasing rates of obesity, metabolic syndrome, and insulin resistance globally. Given its association with long-term metabolic and cardiovascular complications, PCOS represents a substantial public health concern requiring early identification and preventive strategies[7].

Table 2: Epidemiological Overview of PCOS

Parameter	Data
Global prevalence	6 to 20%
Indian prevalence	9 to 22%
Most affected age group	15 to 35 years
Higher prevalence	Urban population
Major risk factors	Obesity, genetics, insulin resistance

Global vs Indian Perspective on PCOS

Polycystic Ovary Syndrome (PCOS) exhibits significant geographic variation in prevalence, clinical characteristics, and metabolic risk profiles. Globally, the estimated prevalence ranges between 6% and 20%, depending on the diagnostic criteria used and the population studied. Differences in ethnicity, lifestyle patterns, and healthcare access contribute to this variability. Broader diagnostic frameworks tend to identify a higher number of affected individuals due to expanded inclusion criteria[8].

In India, reported prevalence rates range from approximately 9% to 22%, suggesting a comparatively substantial disease

burden. Urban populations appear to demonstrate higher rates than rural populations, which may be attributed to rapid urbanization, sedentary behavior, dietary transitions toward energy-dense foods, and increasing obesity prevalence. Furthermore, Indian women with PCOS frequently exhibit pronounced metabolic abnormalities, including insulin resistance, central adiposity, and dyslipidemia, often occurring at lower body mass index thresholds compared to Western populations [9].

An additional concern in the Indian context is the rising incidence of PCOS symptoms during adolescence and early reproductive years. Limited awareness, delayed medical consultation, and sociocultural factors may contribute to underdiagnosis and inadequate management in certain regions. Overall, while PCOS remains a global endocrine disorder, the Indian population appears to experience a relatively higher metabolic risk burden, highlighting the need for early screening, preventive strategies, and population-specific management approaches [10].

Table 3: Comparative overview of epidemiological and metabolic characteristics of PCOS in global and Indian populations.

Parameter	Global Scenario	Indian Scenario
Estimated prevalence	6–20% (varies by diagnostic criteria)	9–22% (higher reported burden)
Diagnostic variability	Influenced by NIH, Rotterdam, AES criteria	Mostly Rotterdam criteria used
Urban vs rural trend	Higher in urban populations	Significantly higher in urban and semi-urban areas
Age group affected	Predominantly 15–35 years	Increasing incidence in adolescents and young women
Obesity association	Strong correlation with obesity	Metabolic risk observed even at lower BMI
Insulin resistance	Common metabolic feature	More pronounced metabolic abnormalities
Type 2 diabetes risk	Elevated compared to general population	Higher relative risk due to early metabolic syndrome
Cardiovascular risk	Increased long-term risk	Potentially higher due to early onset insulin resistance
Awareness & screening	Better awareness in developed nations	Limited awareness in rural areas; delayed diagnosis common

Table 4: BMI Cut-off Comparison – Asian vs Western Standards

BMI Category	Western (WHO Standard	Asian/Indian Standard	Clinical Relevance in PCOS
Underweight	< 18.5 kg/m²	< 18.5 kg/m²	May still have PCOS despite low BMI
Normal weight	18.5 – 24.9 kg/m²	18.5 – 22.9 kg/m²	Asians may show insulin resistance within this range
Overweight	25.0 – 29.9 kg/m²	23.0 – 24.9 kg/m²	Increased metabolic risk begins earlier in Asians
Obesity Class I	30.0 – 34.9 kg/m²	25.0 – 29.9 kg/m²	Higher risk of insulin resistance and anovulation
Obesity Class II	35.0 – 39.9 kg/m²	≥ 30.0 kg/m²	Markedly elevated cardiometabolic risk
Obesity Class III	≥ 40 kg/m²	—	Severe metabolic complications

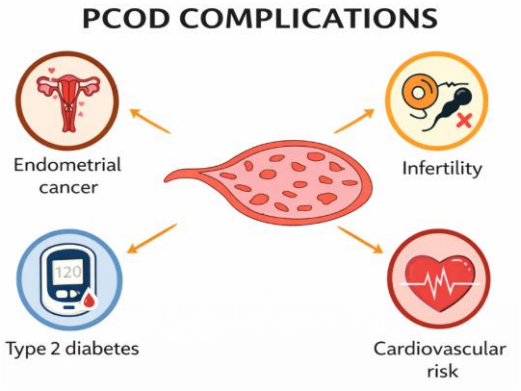

Figure: 1 PCOD Complications

Symptoms of PCOS:

Irregular periods: PCOS disrupts the balance of reproductive hormones (estrogen, progesterone, LH, FSH). This imbalance prevents the ovaries from releasing eggs regularly, leading to delayed, missed, or unpredictable menstrual cycles. Some women may have very heavy bleeding when periods do occur[11].

Ovulation issues: Because eggs are not released consistently, ovulation becomes irregular or absent. This is one of the main reasons PCOD is linked to infertility. Even if

ovulation happens, the hormonal environment may not support proper implantation[12].

Weight gain (especially around the abdomen): PCOD is often associated with insulin resistance, meaning the body doesn't use insulin effectively. This leads to higher insulin levels, which promote fat storage, particularly in the abdominal area. Weight gain further worsens hormonal imbalance, creating a cycle[13].

Acne and oily skin: Elevated levels of androgens (male hormones like testosterone) stimulate sebaceous (oil) glands in the skin. This excess oil clogs pores, causing persistent acne, especially on the face, chest, and back[14].

Hirsutism (excess hair growth): Androgens also trigger hair growth in areas where women typically have little or no hair, such as the chin, upper lip, chest, and abdomen. This symptom can be distressing and affect self-esteem[15].

Thinning hair or hair loss: While androgens cause excess hair growth in some areas, they also shrink hair follicles on the scalp. This leads to thinning hair or male-pattern baldness, especially near the crown[16].

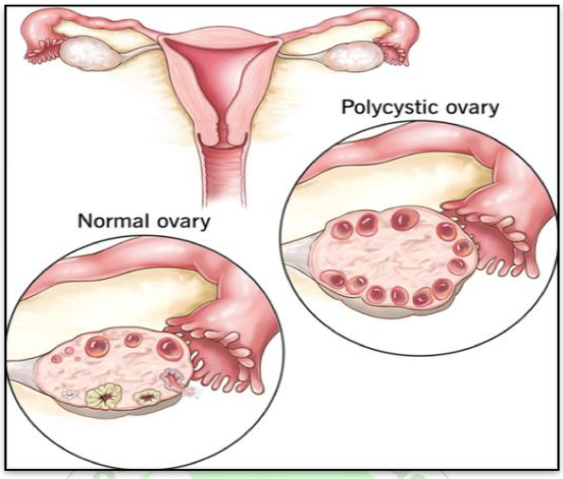

Figure: 2 Normal vs Polycystic ovary

Fatigue and mood changes: Hormonal fluctuations, insulin resistance, and poor sleep quality (sometimes linked to PCOD-related weight gain or sleep apnea) can cause chronic tiredness. Mood swings, anxiety, and depression are also common due to both biological and emotional factors [17].

Cysts in ovaries: The name "polycystic" comes from the presence of multiple small fluid-filled sacs (follicles) in the ovaries. These are immature eggs that fail to mature and release. Not all women with PCOD have visible cysts, but when present, they are detected via ultrasound [18].

PCOS is not just about the ovaries-it's a systemic condition involving hormones, metabolism, and mental health. That's why symptoms can range from cosmetic (acne, hair changes) to reproductive (infertility) and metabolic (weight gain, insulin resistance)[19].

Table 5: Summary of Pathophysiological Mechanisms

Mechanism	Effect	Clinical Outcome
Insulin resistance	↑ Insulin levels	↑ Androgen production
Hyperandrogenism	Follicular arrest	Anovulation
LH/FSH imbalance	Poor follicle development	Infertility
Chronic inflammation	Endothelial dysfunction	CV risk

Pathophysiology of PCOS:

PCOS results from interconnected endocrine and metabolic disturbances.

Hyperandrogenism

Elevated luteinizing hormone (LH) stimulates ovarian theca cells, increasing androgen production. Reduced follicle-stimulating hormone (FSH) impairs follicular maturation, leading to chronic anovulation[20].

Insulin Resistance

Peripheral insulin resistance results in compensatory hyperinsulinemia, which enhances ovarian androgen synthesis and reduces sex hormone-binding globulin (SHBG), increasing free testosterone levels[21].

Neuroendocrine Dysfunction

Increased gonadotropin-releasing hormone (GnRH) pulse frequency leads to LH predominance, further exacerbating androgen excess[22].

Chronic Inflammation

Low-grade systemic inflammation contributes to endothelial dysfunction and cardiovascular risk.

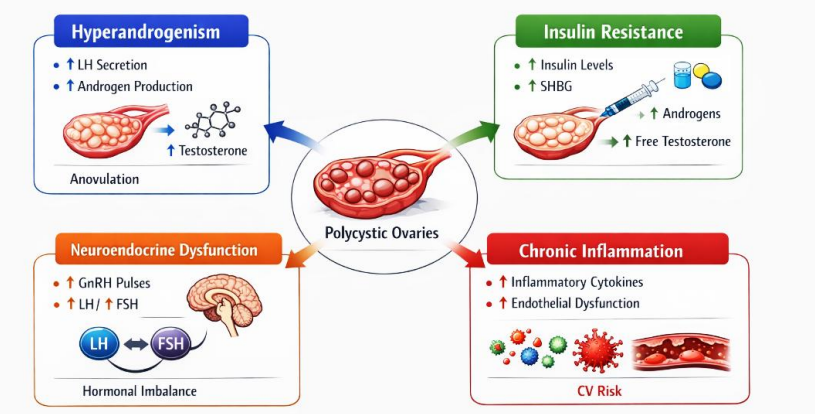

Figure: 3 Pathophysiology of PCOS

Etiology of Polycystic Ovary Syndrome (PCOS)

Polycystic Ovary Syndrome (PCOS) is a multifactorial disorder resulting from complex interactions among genetic, hormonal, metabolic, and environmental factors. Although the exact cause remains incompletely understood, several contributory mechanisms have been identified [23].

Genetic Predisposition

Familial clustering of PCOS suggests a strong hereditary component. Women with a first-degree relative affected by PCOS are at increased risk of developing the disorder. Genetic variations influencing insulin signaling pathways, androgen synthesis, and ovarian folliculogenesis have been implicated. However, PCOS is considered polygenic rather than caused by a single gene mutation [24].

Insulin Resistance

Insulin resistance is a central etiological factor in many patients with PCOS. Reduced cellular sensitivity to insulin leads to compensatory hyperinsulinemia. Elevated insulin levels stimulate ovarian theca cells to produce excess androgens and decrease hepatic production of sex hormone-binding globulin (SHBG), thereby increasing free circulating testosterone levels. This hormonal imbalance contributes to anovulation and hyperandrogenic symptoms [25].

Hyperandrogenism

Excess androgen production from the ovaries and, in some cases, adrenal glands is a hallmark feature of PCOS. Increased luteinizing hormone (LH) secretion promotes androgen synthesis. Elevated androgen levels disrupt normal follicular maturation, resulting in follicular arrest and formation of multiple small ovarian follicles [26].

Neuroendocrine Dysfunction

Altered hypothalamic–pituitary signaling leads to increased gonadotropin-releasing hormone (GnRH) pulse frequency, favoring LH secretion over follicle-stimulating hormone (FSH). This imbalance impairs normal follicular development and ovulation [27].

Obesity and Lifestyle Factors

Obesity, particularly central adiposity, exacerbates insulin resistance and hyperandrogenism. Sedentary lifestyle, high-calorie diets, and chronic stress contribute to metabolic dysfunction, thereby worsening PCOS manifestations. Notably, PCOS can also occur in lean women, although metabolic severity may differ [28].

Chronic Low-Grade Inflammation

Women with PCOS often exhibit elevated inflammatory markers, indicating a state of chronic low-grade inflammation. Inflammatory mediators may impair insulin signaling and ovarian function, further contributing to disease progression [29].

Environmental and Epigenetic Influences

Environmental factors such as endocrine-disrupting chemicals, intrauterine exposure to excess androgens, and early-life metabolic disturbances may influence gene expression through epigenetic mechanisms. These changes can predispose individuals to PCOS later in life[30].

Clinical Manifestations

Common features include:

- Irregular menstrual cycles
- Hirsutism
- Acne
- Obesity (central adiposity)
- Infertility
- Scalp hair thinning
- Mood disturbances

Diagnosis

Diagnosis is based on the **Rotterdam criteria (2003)** requiring at least two of the following three features after exclusion of related disorders [31].

1. Clinical or biochemical hyperandrogenism
2. Oligo-ovulation or anovulation
3. Polycystic ovarian morphology on ultrasound

Table 6: Rotterdam Diagnostic Criteria

Criteria	Description
Hyperandrogenism	Clinical (hirsutism/acne) or biochemical
Ovulatory dysfunction	Oligomenorrhea or anovulation
Polycystic ovaries	≥12 follicles (2–9 mm) or ovarian volume >10 mL
Diagnosis	Any 2 of 3

Complications of PCOD:

Infertility and Reproductive Dysfunction:

According to IJPS reviews, PCOD is a leading cause of anovulatory infertility. The hormonal imbalance and excess androgen production prevent normal follicular maturation and ovulation. Even when ovulation occurs, the endometrium may not be adequately prepared for implantation, which further reduces fertility potential[32].

Pregnancy Complications:

Women with PCOS who conceive are at higher risk of gestational diabetes, preeclampsia, miscarriage, and preterm birth. These complications stem from underlying insulin resistance, chronic inflammation, and hormonal imbalance[33].

Cardiovascular Risks:

Review literature notes that women with PCOS have higher rates of dyslipidemia, hypertension, and endothelial dysfunction, all of which predispose them to cardiovascular disease. Chronic low-grade inflammation further exacerbates vascular risk, increasing the likelihood of ischemic heart disease later in life[34]

Psychological and Emotional Impact:

Meta-analyses and WJPSR reviews highlight the psychological burden of PCOS. Women with the condition show higher prevalence of depression, anxiety, and reduced quality of life. Symptoms such as hirsutism, acne, and obesity contribute to body image concerns, while infertility adds emotional distress[35].

Endometrial Hyperplasia and Cancer:

Because PCOD frequently leads to prolonged anovulation, the endometrium is exposed to unopposed estrogen without progesterone. WJPSR reviews emphasize that this hormonal environment increases the risk of endometrial hyperplasia and, in severe cases, endometrial carcinoma[36].

Metabolic Syndrome and Type 2 Diabetes:

Insulin resistance is a central feature of PCOD, and WJPSR reviews consistently link it to metabolic syndrome-a cluster of obesity, hypertension, and dyslipidemia. Elevated insulin levels worsen androgen excess and increase the risk of type 2 diabetes, making PCOD a systemic metabolic disorder rather than just a reproductive condition[36].

Pharmaceutical Management:

Combined Oral Contraceptives (COCs):

Review articles consistently identify COCs as the **first-line therapy** for women not seeking pregnancy. They regulate menstrual cycles, reduce androgen levels, and improve acne and hirsutism. Importantly, they protect the endometrium from hyperplasia and carcinoma by balancing estrogen with progestin[37].

Anti-Androgen Therapy:

Agents such as **spironolactone, flutamide, and finasteride** are used to treat hirsutism and acne. They act by blocking androgen receptors or inhibiting androgen synthesis. Reviews caution that these drugs must be combined with contraception due to teratogenic risks if pregnancy occurs[38].

Insulin Sensitizers:

Metformin is widely discussed as a cornerstone therapy in PCOD with insulin resistance. It improves insulin sensitivity, lowers circulating insulin levels, and indirectly reduces ovarian androgen production. Metformin also helps restore ovulation and improves metabolic parameters, reducing the risk of type 2 diabetes. Some reviews also mention thiazolidinediones and newer agents like GLP-1 receptor agonists, though their use is less established[39].

Ovulation Induction Agents:

For women desiring pregnancy, clomiphene citrate has traditionally been the first-line ovulation induction drug. More recent review articles highlight letrozole (an aromatase inhibitor) as superior in terms of ovulation and live birth rates. In resistant cases, gonadotropins may be used, though they carry a higher risk of multiple pregnancies and ovarian hyperstimulation[40].

Adjunct Therapies:

Eflornithine cream is used topically to reduce facial hair growth.

Statins have been explored for improving lipid profiles and reducing inflammation, though their routine use in PCOD remains debated.

Weight management drugs are occasionally considered in obese women, but lifestyle modification remains the cornerstone[41].

Table 7: Pharmacological Treatment Summary

Drug Class	Examples	Mechanism	Indication
COCs	Ethinyl estradiol + Progestin	Suppress LH	Cycle regulation
Insulin sensitizer	Metformin	↑ Insulin sensitivity	IR, anovulation
Ovulation induction	Letrozole	Aromatase inhibitor	Infertility
Anti-androgen	Spironolactone	AR blocker	Hirsutism

Essential Nutrients Required in PCOD:

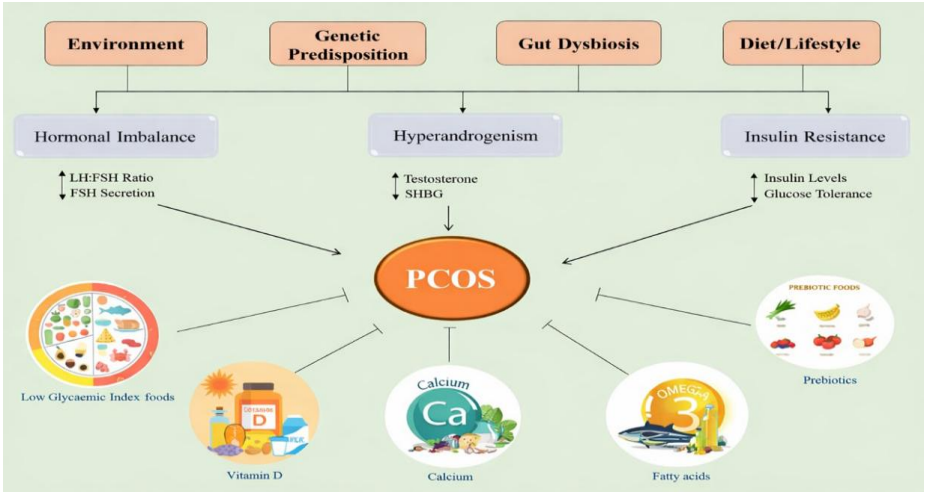

Figure 4: Managing PCOS: Key Factors and Solutions

Dietary Fiber:

High fiber intake is crucial in PCOD because it slows glucose absorption, reduces post-meal insulin spikes, and improves overall insulin sensitivity. This helps control blood sugar levels and reduces the risk of type 2 diabetes, which is common in PCOD. Whole grains, legumes, vegetables, and fruits are emphasized as primary sources[42].

Lean Protein:

Adequate protein intake supports muscle health, improves satiety, and helps regulate appetite. Unlike refined carbohydrates, lean protein does not cause sharp rises in blood sugar, making it beneficial for women with insulin resistance. Sources include fish, poultry, eggs, soy, and pulses[43].

Omega-3 Fatty Acids:

Omega-3 fatty acids have strong anti-inflammatory properties, which are important because PCOD is associated with chronic low-grade inflammation. They also improve lipid profiles and reduce cardiovascular risk. Fatty fish (salmon, mackerel), flaxseeds, walnuts, and chia seeds are excellent sources[43].

Vitamin D:

Vitamin D deficiency is highly prevalent in women with PCOD. Supplementation improves insulin sensitivity, menstrual regularity, and even ovulation rates. Sunlight exposure, fortified dairy products, and supplements are common sources [44].

Vitamin B12 and Folate:

These nutrients are essential for metabolic health and red blood cell formation. Women taking metformina common drug in PCOD management-are at risk of B12 deficiency, making supplementation important. Folate also supports reproductive health and reduces the risk of neural tube defects in pregnancy. Sources include leafy greens, eggs, dairy, and fortified cereals[45].

Magnesium:

Magnesium plays a role in glucose metabolism and insulin action. Adequate intake reduces inflammation and improves insulin sensitivity. Nuts, seeds, whole grains, and green leafy vegetables are rich sources[46]

Zinc:

Zinc supports reproductive health, regulates hormone balance, and helps reduce acne and hirsutism. It also plays a role in immune function. Pumpkin seeds, legumes, and seafood are good dietary sources[47].

Antioxidants (Vitamin E, Selenium):

Oxidative stress is elevated in PCOD, contributing to inflammation and metabolic dysfunction. Antioxidants such as vitamin E and selenium help neutralize free radicals, improving overall cellular health. Sources include nuts, seeds, vegetable oils, and Brazil nuts[48].

Table 8: Nutritional Support in PCOS

Nutrient	Role	Sources
Dietary fiber	Improves insulin control	Whole grains
Omega-3	Anti-inflammatory	Fish
Vitamin D	Enhances ovulation	Sunlight
Zinc	Hormonal regulation	Seeds
Magnesium	Glucose metabolism	Nuts

Lifestyle Modification Strategies:

Dietary Management:

The balanced nutrition-rich in fiber, lean protein, and omega-3 fatty acids-improves insulin sensitivity and reduces androgen levels. Low-glycemic index diets are particularly effective in regulating menstrual cycles and metabolic outcomes[49].

Weight Reduction and Exercise:

Even modest weight loss (5–10% of body weight) can restore ovulation and improve fertility. Regular aerobic exercise combined with resistance training reduces insulin resistance, lowers BMI, and improves cardiovascular health in PCOD patients[50].

Behavioral and Psychological Support:

A lifestyle modification should extend beyond diet and exercise to include stress management, sleep hygiene, and psychological counseling. Addressing depression and anxiety improves adherence to lifestyle interventions and enhances overall quality of life[51].

Holistic Lifestyle Interventions:

Combined interventions-dietary changes, structured physical activity, and behavioral therapy-are more effective than single interventions. Multidisciplinary approaches significantly improve reproductive outcomes, metabolic parameters, and emotional well-being[52].

Beyond Diet and Exercise:

A lifestyle management should also address sleep quality, smoking cessation, and reduction of sedentary behavior. These factors contribute to better hormonal balance and reduced long-term risks such as diabetes and cardiovascular disease[53].

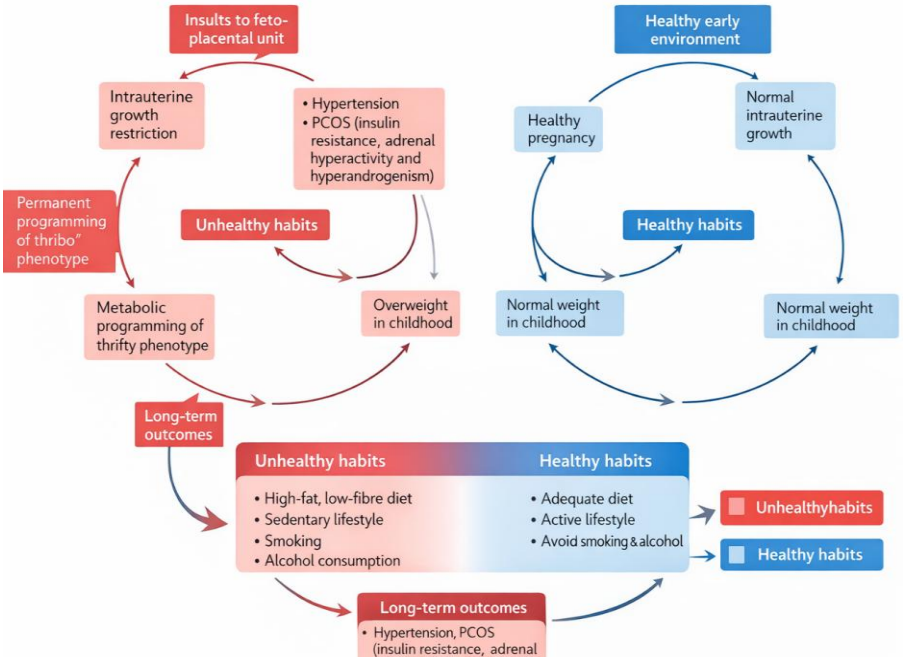

Figure 5: Healthy vs unhealthy habit cycles

CONCLUSION:

Polycystic Ovary Syndrome (PCOS) is a complex and multifactorial endocrine–metabolic disorder with significant reproductive, metabolic, and psychological implications. It extends beyond ovarian dysfunction and represents a systemic condition characterized by hyperandrogenism, insulin resistance, chronic low-grade inflammation, and neuroendocrine dysregulation. The heterogeneity of clinical presentation—including menstrual irregularities, infertility, metabolic disturbances, and long-term cardiometabolic risk—highlights the need for comprehensive and individualized patient assessment.

Early diagnosis based on standardized criteria and appropriate exclusion of differential conditions is essential to prevent long-term complications such as type 2 diabetes mellitus, cardiovascular disease, and endometrial hyperplasia. Current management strategies emphasize a multidisciplinary approach integrating lifestyle modification, pharmacological therapy, metabolic risk reduction, and psychological support. Weight optimization, insulin sensitization, hormonal regulation, and fertility-directed treatments remain central components of therapy.

Emerging interventions—including inositol supplementation, GLP-1 receptor agonists, microbiome modulation, and personalized metabolic profiling—offer promising avenues for future therapeutic advancement. However, further large-scale, long-term clinical trials are required to establish safety, efficacy, and standardized treatment algorithms.

In summary, PCOS should be approached as a lifelong metabolic and reproductive disorder requiring early intervention, patient education, and sustained follow-up. A shift toward precision medicine and preventive strategies may significantly improve quality of life and reduce the global health burden associated with this condition.

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