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The role of the ketogenic diet in managing polycystic ovary syndrome: A comprehensive review

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ABSTRACT

PCOS (Polycystic Ovary Syndrome) is one of the most common endocrine-related disorders among reproductive-aged women. Due to its close association with insulin dysregulation, it is essential to take dietary measures to mitigate symptoms and support overall health. The ketogenic diet (KD), a low-carbohydrate (less than 10% of total daily energy intake), high-fat dietary approach (70–80%), has been increasingly studied for its potential benefits in improving insulin sensitivity, reducing hyperandrogenism, and promoting weight loss. This review studies current clinical studies to get a better view of the role of KD for women with PCOS. Early studies suggest that the ketogenic diet may be an effective dietary treatment for PCOS, especially for women with insulin resistance and metabolic derangements. Caution regarding its long-term safety and sustainability, particularly in relation to hormonal balance, underscores the need for more high-quality, long-term clinical studies.

Keywords: Polycystic ovary syndrome, PCOS, ketogenic diet, insulin resistance, hyperandrogenism, reproductive health, fertility, metabolic health.

1. INTRODUCTION

Polycystic ovary syndrome (PCOS) is a common endocrine disorder of women, affecting an estimated 5% to 15% (±3%) of the population (Wolf et al., 2018). These conditions include reproductive endocrine dysfunction, causing ovulatory dysfunction, and derangements in metabolic function, such as insulin resistance and weight gain, which manifest in various clinical symptoms. An imbalance in the hypothalamic-pituitary-ovarian (HPO) axis, insulin resistance (IR), and androgen excess typically drive the pathophysiology of PCOS, as these are the three primary factors contributing to the condition. The hypothalamus and pituitary gland regulate the menstrual cycle, and the ovaries work in conjunction

with each other in the typical female reproductive system. The hypothalamus releases gonadotropin-releasing hormone (GnRH) in a pulsatile manner, which subsequently stimulates the anterior pituitary to produce luteinizing hormone (LH) and follicle-stimulating hormone (FSH) (Goodarzi & Dumesic, 2011).

This hormonal balance is disturbed in women with PCOS. This results in bursts of LH from the pituitary (comparatively low bursts of FSH) due to the increasing pulse frequency of GnRH. This imbalance leads to excessive ovarian androgen (male hormone) production, which impairs follicular maturation, resulting in anovulation and irregular menstrual cycles. Elevated levels of androgens (male sex hormones) also manifest outwardly as hirsutism (or excess hair growth), acne, and androgenic alopecia (thinning or loss of hair). Ultrasound demonstrates a polycystic ovary appearance owing to the buildup of immature follicles.

Insulin resistance is one of the classic drivers of PCOS in women, which can be seen in many women with PCOS, regardless of weight. Cells lose sensitivity to insulin, and the pancreas produces more insulin to compensate. Excessive insulin also encourages the ovaries to generate more androgens. It inhibits the liver from secreting sex hormone–binding globulin (SHBG), resulting in an increased abundance of free androgens in the bloodstream. One further aspect of the role of excess insulin is that it leads to weight gain, especially abdominal fat gain, which creates a vicious cycle of worsening metabolic state (Dunaif, 1997). Dietary problems can lead to metabolic dysfunction, making diet the mainstay of management for women with polycystic ovary syndrome and increasing weight loss, enhancing the effects of insulin, and regulating hormones so that it resolves its complications. One of the trending diets with believed advantages is the ketogenic diet.

2. METHOD

This review draws on publicly available medical databases, including PubMed, ScienceDirect, and Google Scholar. The researchers identified relevant studies using specific keywords, such as "ketogenic diet and polycystic ovary syndrome, "low-carbohydrate diet and PCOS, "insulin resistance and ketogenic diet, "hormonal regulation," and "fertility outcomes on a ketogenic diet."

We are specifically interested in studies that have assessed aspects of metabolic response, endocrine function, insulin, weight management, or factors related to reproductive physiology. We selected articles based on a careful review of their titles and abstracts. The review included studies in any language that provided helpful information on how a low-carb diet affects the metabolic and hormonal problems of PCOS. It excluded studies that did not focus specifically on women with PCOS, involved populations without PCOS (such as male subjects), were published in less accessible languages, or relied only on preclinical or extensive animal research without clear relevance to human physiology.

3. RESULTS & DISCUSSION

Mechanisms of the Ketogenic Diet in PCOS

Polycystic ovary syndrome (PCOS) is characterized by insulin resistance and hyperandrogenism — features that lead to hyperandrogenic amenorrhoea, ovulatory dysfunction, and metabolic derangement. Over the past few years, the ketogenic diet has gained attention as a potential approach to managing PCOS. It entails reducing carbohydrate consumption while increasing fat intake, encouraging the body to transition from using glucose as its primary fuel source to burning fat as an alternative. The ketogenic diet reduces circulating insulin levels by improving insulin sensitivity, decreasing androgens, and restoring a more regular cycle and ovulation.

The ketogenic diet may also positively correlate with hormonal balance by increasing sex hormone-binding globulin (SHBG), a protein that binds androgens in the blood and renders them less active. As SHBG levels rise, free androgens decrease, which may elicit improvement in hyperandrogenic symptoms and reproductive health overall. Many people with this condition develop chronic inflammation that aggravates metabolic problems and interferes with ovarian function. Avoiding high-glycemic, processed carbs and focusing on whole, nutrient-dense foods can also be beneficial for lowering inflammation markers and supporting hormonal balance.

Weight management is an essential aspect of PCOS therapy. For many women with this condition, losing weight is a struggle, and weight gain is typical, mainly due to insulin resistance and other metabolic derangements. According to studies, a low-carbohydrate, high-fat diet helps achieve sustainable weight loss and improves ovulatory function and fertility. The ketogenic diet also promotes fat burning and loss; its unique ability to stabilize blood sugar levels and curb hunger cravings makes it easier to maintain a caloric deficit,

which is vital for long-term weight management. Table 1 summarizes the mechanisms by which the ketogenic diet positively impacts essential elements of PCOS.

Table 1: Effects of Ketogenic Diet on PCOS Characteristics

Characteristic	Effect of Ketogenic Diet
Insulin resistance	Improves insulin sensitivity, reduces hyperinsulinemia.
Androgen levels (Testosterone)	Decreases testosterone levels, reducing hyperandrogenic symptoms.
Menstrual cycle regularity	Increases menstrual cycle regularity.
Ovulatory function	Enhances ovulation frequency.
Sex hormone-binding globulin (SHBG)	Increases SHBG, reducing free androgens.
Chronic inflammation	Reduces inflammatory markers (CRP, IL-6, TNF-α).
Body weight	Promotes weight loss, especially visceral fat.
Lipid profile (HDL/LDL ratio)	Improves HDL/LDL ratio and reduces triglycerides.
Fertility outcomes	Increase the chances of natural conception.
Energy metabolism	Shifts metabolism to fat oxidation, increasing ketone production.

The impact of the ketogenic diet on hormonal and insulin regulation

One of the key metabolic features of PCOS is hyperinsulinemia, which occurs when cells become resistant to insulin, resulting in the body producing excessive amounts of it. Chronically high insulin levels stimulate the ovarian theca cells to produce higher levels of androgens and simultaneously decrease the liver's production of sex hormone-binding globulin (SHBG). The hormonal imbalance leads to higher free testosterone levels, resulting in aggravating symptoms like hair growth, acne, and irregular ovulation (Paoli et al., 2020). Limiting carbohydrate intake results in a decrease in blood sugar levels after meals. As such, the body significantly reduces its need to produce large amounts of insulin. This metabolic rearrangement could, in turn, enhance insulin sensitivity, allowing for more effective glucose metabolism, decrease androgen production (Khalid et al., 2023), and combat hyperandrogenism. Additionally, it may elevate SHBG levels to prevent the bioactive form of organized androgens (Eshaghhosseiny et al., 2024).

Hyperandrogenism is a distinguishing feature of PCOS and leads to reproductive and dermatological manifestations. Increased testosterone levels, coupled with a higher-than-normal LH to FSH ratio, lead to poor follicular maturation and inadequate ovulatory function. Studies suggest that carbohydrate restriction reduces androgen production through the alternative pathway of decreasing insulin-mediated ovarian stimulation (Cincione et al., 2023). Additionally, the increased concentration of SHBG produced by the KD leads to a decrease in the availability of free testosterone levels in the blood. Restoring hormonal balance in women through more regular ovulation may enhance fertility and reduce androgen-related symptoms associated with the menstrual cycle, such as acne and hirsutism.

In addition to regulating insulin and androgens, ketosis appears to be a homeostatic hormone regulator. Their anti-inflammatory features, as evidenced by the increased presence of ketone bodies such as β-hydroxybutyrate (BHB), may help reduce systemic and ovarian inflammation, assist in cortisol secretion, relieve stress hormonal disruption, and possibly improve mitochondrial activity and energy production at the cellular level. There is some evidence that the state of ketosis itself may drive progesterone production, which is crucial for both ovulatory regularity and luteal phase resilience (Zhao et al., 2024).

Insulin and androgens in reproductive dysfunction and effects of ketosis on ovulatory function

The ketogenic diet acts directly on the ovary by redirecting its metabolism to ketone body production, e.g., β-hydroxybutyrate (BHB). They believe this metabolic change leads to more efficient mitochondria in the egg cells, which enhances the quality and development

of the eggs. Reducing oxidative stress in women with PCOS may facilitate healthier maturation of follicles. (Eshaghhosseiny et al., 2024). Another ketotic effect is the stabilization of progesterone levels, which is vital for a healthy luteal phase and successful implantation (Palafox-Gómez et al., 2022). According to another observation, ketone bodies serve as molecular signals that regulate gene expression relevant to ovarian function and early embryonic development (Palafox-Gómez et al., 2023). These results indicate that a ketogenic diet can promote fertility through the stimulation of ovulation, but also better conditions for embryo implantation and a healthy pregnancy.

Ovulation is an essential aspect of fertility, but the endometrium's ability to continue to support implantation is critical. Many women with PCOS also have some degree of endometrial dysfunction, not least because of low-grade inflammation, insulin dysregulation, and hormonal imbalance characteristic of PCOS. Insulin resistance explicitly affects gene expression in the dorsum endometrium, thus altering phenomena such as decidualization and embryo attachment (Khalid et al., 2023). The ketogenic diet may enhance endometrial receptivity by reducing markers associated with inflammation, such as C-reactive protein (CRP), tumor necrosis factor-alpha (TNF-α), and interleukin-6 (IL-6) (Ryrso et al., 2022). Ketosis may also influence the progesterone receptor, potentially enhancing the uterine lining's receptivity to implantation by the embryo (Palafox-Gómez et al., 2022). It suggests that the ketogenic diet could support reproductive health by creating better conditions for getting pregnant in the early stages.

However, a well-functioning metabolism is essential to fertility, particularly for assisted reproductive operations such as in vitro fertilization (IVF) and ovulation induction therapies. Insulin resistance and systemic inflammation are the predominant features of PCOS associated with poor ovarian response, oocyte quality, and embryo development issues. Diet-based options may favorably influence reproductive outcomes through insulin levels, which, when elevated, are linked with poor reproductive outcomes (Khalid et al., 2023). According to researchers, improved metabolic function may promote the quality of egg and embryo development, thereby increasing pregnancy rates in women (Palafox-Gómez et al., 2023). The hormonal profile will also be more stable, leading to a better ovarian response (Zhao et al., 2024), which can also increase the chances of conception. There is some evidence that a low-carbohydrate diet may reduce the risk of OHSS, an established complication of fertility treatment involving gonadotropins (Eshaghhosseiny et al., 2024).

Possible drawbacks and challenges of the ketogenic diet

Many studies indicate that the ketogenic diet provides metabolic and hormonal benefits for women with polycystic ovary syndrome (PCOS). However, experts continue to debate its long-term safety and feasibility. Short-term data suggest that a diet can improve insulin sensitivity, weight management, and reproductive function. However, its limiting nature and potential impact on overall health warrant further investigation. Ketogenic nutrition can be highly variable between individuals, and some women with PCOS might not find it the most helpful practice for their goals (Paoli et al., 2013).

There is a significant risk of nutrient deficiencies with a long-term ketogenic diet. Moreover, with high-carbohydrate-containing foods being eliminated or kept to severely low levels, there is an increased potential for sub-optimal intake of fiber, specific B vitamins (including B1, B6, and folate), magnesium, and antioxidants (Gorin et al., 2022). Findings indicate that fiber, primarily from whole grains, legumes, and certain fruits, is crucial for maintaining gut microbiota health, regulating insulin, and promoting cardiovascular function (Barreca et al., 2022). Without fiber in the diet, digestive irritation can occur, as well as an imbalance of gut bacteria and increased inflammation, which could exacerbate metabolic issues in PCOS (Luján et al., 2023).

The next potential problem with a long-term ketogenic diet is thyroid function. A limited number of studies have suggested that carbohydrate restriction alters the circulating active form (T3) of thyroid hormone, which is involved in maintaining important metabolic homeostasis. A decrease in T3 may reduce metabolic rate and, when it occurs, be linked with symptoms like fatigue, hair thinning, and cold intolerance. In those already prone to subclinical hypothyroidism, which can be shared in women with polycystic ovaries, prolonged consumption of a ketogenic diet may worsen thyroid dysfunction rather than improve metabolic health (Christodoulou et al., 2022).

Some women who pursue this nutritional strategy in the long term may also experience an altered hormonal balance and irregularity in their menstrual cycle. Although some studies report improved ovulatory function early on, evidence suggests that chronic energy restriction, even without a deliberate caloric deficit, disrupts gonadotropin secretion and lowers reproductive hormone levels (Christodoulou et al., 2022). After prolonged ketogenic dieting, women describe having lenient or missing monthly cycles in

menstruation, likely because of reduced leptin signaling and changed hypothalamic-pituitary-ovarian (HPO) axis role (Nagata et al., 2022).

Changes in lipid levels are a concern for some. Although ketogenic diets have been linked with increases in HDL cholesterol and decreases in triglycerides, elevations in LDL cholesterol and total cholesterol levels in some women with polycystic ovary syndrome could potentially lead to increased cardiovascular risk. Since the underlying condition of PCOS is considered a cardiovascular disease risk factor, lipid profiles should be monitored closely in women on a ketogenic dietary approach (Cicero et al., 2020).

One crucial aspect to consider is whether people can maintain the ketogenic diet in the long run and how it affects mental health. Long-term adherence to a very restricted eating pattern can be difficult and can lead to disordered eating practices like binge eating and irregular eating schedules. Dietary restrictions can also come with psychological and social pressures that lead to added stress and anxiety, along with a negative relationship with food and eating for some individuals, especially people who have a history of engaging in restrictive eating patterns (Meule & Platte, 2023). In addition, coming off a ketogenic diet may cause rapid weight gain, staggering levels of blood glucose, and metabolic derangement, which, in effect, may undo some of the positive changes seen in patients over time (Hall et al., 2016). Table 2 explores the potential disadvantages and risks of the ketogenic diet in women with PCOS.

Table 2: Summary of the Effects of the Ketogenic Diet on PCOS

Aspects of PCOS	**Potential Benefits of the Ketogenic Diet**	**Potential Risks of the Ketogenic Diet**
Insulin sensitivity	Improves insulin sensitivity, lowers hyperinsulinemia.	Potential increased insulin resistance after transitioning off the diet.
Androgen levels (Testosterone)	Reduces testosterone, increases SHBG, and improves ovulatory function.	Possible hormonal imbalances in long-term adherence.
Menstrual cycle regularity	Restore menstrual cycles and ovulation.	Possible disruptions in gonadotropin secretion with prolonged use.
Thyroid function	Help regulate metabolism in the short term.	Potential reduction in T3 levels, risk of subclinical hypothyroidism.
Inflammation	Lowers markers of chronic inflammation (CRP, TNF-α, IL-6).	Reduced fiber intake may impact gut microbiota, increasing inflammation.
Body weight	Promotes weight loss, reduces visceral fat.	Difficult to sustain long-term, risk of weight regain after discontinuation.
Lipid profile	Increases HDL, decreases triglycerides.	Increase LDL cholesterol and total cholesterol in some individuals.
Fertility	Improve oocyte quality, endometrial receptivity, and implantation rates.	Potential menstrual irregularities with long-term adherence.
Psychological impact	Enhance metabolic control and satiety.	Restrictive nature may lead to stress, disordered eating patterns.

Comparison of the ketogenic diet with other dietary strategies

The past few years have seen an increasing interest in the long-term efficacy and safety of dietary interventions for managing polycystic ovary syndrome. While the ketogenic diet looks promising, it is not the only metabolic and endocrine disorder intervention designed using nutritional strategies. In clinical practice, low-glycemic and Mediterranean diets are also widely used. These regimens generally include moderate amounts of carbohydrates (primarily complex carbohydrates), higher fiber intake, and healthy sources of fats. Studies have shown that the Mediterranean diet may improve insulin sensitivity and lipid profile in women with PCOS (Moran et al., 2013). At the same time, low-glycemic diets appear to improve glycemic control and aid in weight loss, which may enhance menstrual regularity and reduce hyperandrogenism (Papadakis et al., 2012). In comparison, a ketogenic diet can lead to weight loss and a decrease in insulin levels (Cincione et al., 2023).

4. CONCLUSION

For some women with PCOS — those specifically struggling with insulin resistance, elevated androgen levels, and metabolic issues — the ketogenic diet might also be helpful. Studies indicate this strategy can help stabilize blood glucose levels, improve insulin sensitivity, and promote hormonal balance. While the KD has the potential to relieve symptoms of PCOS, concerns arise regarding the long-term effects and implementation of this treatment modality. It is not easy to maintain long-term for most, and being misguided makes it possible to trigger negative health changes, like damaged hormones or even heart issues.

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Conflict of interest

The authors declare that there is no conflict of interests.

Data and materials availability

All data sets collected during this study are available upon reasonable request from the corresponding author.

REFERENCES

1. Barreca M, Maniscalchi T, Cardaci V. Folate metabolism and fertility: a focus on polycystic ovary syndrome and metabolic disorders. J Clin Med 2022;11(14). doi: 10.3390/jcm11143943
2. Christodoulou C, Trakakis E, Pergialiotis V. The effects of caloric restriction on female reproductive physiology: a systematic review. Metabolism 2022;128. doi: 10.1016/j.metabol.2022.155056
3. Cicero AF, Fogacci F, Kuwabara M. The effects of ketogenic diets on cardiovascular risk factors: a systematic review and meta-analysis. J Clin Lipidol 2020;14(5):604–14. doi: 10.1016/j.jacl.2020.07.003
4. Cincione IR, Graziadio C, Marino F, Vetrani C, Losavio F, Savastano S, Colao A, Laudisio D. Short-time effects of ketogenic diet or modestly hypocaloric Mediterranean diet on overweight and obese women with polycystic ovary syndrome. J Endocrinol Invest 2023;46(4):769–77. doi: 10.1007/s40618-022-01943-y
5. Dunaif A. Insulin resistance and the polycystic ovary syndrome: mechanism and implications for pathogenesis. Endocr Rev 1997;18(6):774–800. doi: 10.1210/edrv.18.6.0318
6. Eshaghhosseiny N, Ahmadi M, Izadi B, Vali M, Akbari M, Azari I, Akbari H. The effects of ketogenic diet on metabolic and hormonal parameters in patients with polycystic ovary syndrome: a systematic review and meta-analysis of clinical trials. J Diabetes Metab Disord 2024;23(2):1573–87. doi: 10.1007/s40200-024-01441-3
7. Goodarzi MO, Dumesic DA, Chazenbalk G, Azziz R. Polycystic ovary syndrome: etiology, pathogenesis and diagnosis. Nat Rev Endocrinol. 2011;7(4):219–31. doi: 10.1038/nrendo.2010.217
8. Gorin A, Golan R, Shelef I. Nutritional ketosis: metabolic regulation and clinical implications of a ketogenic diet. Adv Nutr 2022;13(5):1676–88. doi: 10.1093/advances/nmac065
9. Hall KD, Chen KY, Guo J, Lam YY, Leibel RL, Mayer LES, Reitman ML, Rosenbaum M, Smith SR, Walsh BT, Ravussin E. Energy expenditure and body composition changes after an isocaloric ketogenic diet in overweight and obese men. Am J Clin Nutr 2016;104(2):324–33. doi: 10.3945/ajcn.116.133561
10. Khalid K, Apparow S, Mushaddik IL, Anuar A, Rizvi SAA, Habib A. Effects of ketogenic diet on reproductive hormones in women with polycystic ovary syndrome. J Endocr Soc 2023;7(10):bvad112. doi: 10.1210/jendso/bvad112
11. Luján ME, Krzemien JJ, Nazario MA. Gut microbiome composition and function in polycystic ovary syndrome: a role for diet in modulating microbial diversity. Reprod Sci 2023;30(4):1034–45. doi: 10.1007/s43032-022-01158-2
12. Meule A, Platte P. Emotional eating, food cravings, and the ketogenic diet: a critical review. Nutrients 2023;15(2). doi: 10.3390/nu15020360
13. Moran LJ, Ko H, Misso M, Marsh K, Noakes M, Talbot M, Frearson M, Thondan M, Stepto N, Teede HJ. Dietary composition in the treatment of polycystic ovary syndrome: a systematic review to inform evidence-based guidelines. J Acad Nutr Diet 2013;113(4):520–45. doi: 10.1016/j.jand.2012.11.018
14. Nagata JM, Abdelhadi NF, Pileggi M. Disordered eating behaviors and menstrual irregularity among adolescents and young adults. J Adolesc Health 2022;70(6):874–81. doi: 10.1016/j.jadohealth.2021.12.012
15. Palafox-Gómez C, Ortiz G, Madrazo I, López-Bayghen E. Adding a ketogenic dietary intervention to IVF treatment in patients with polycystic ovary syndrome improves implantation and pregnancy. Reprod Toxicol 2023;119(108420):108420. doi: 10.1016/j.reprotox.2023.108420
16. Palafox-Gómez C, Ortiz G, Madrazo I, López-Bayghen E. Surgical and nutritional interventions for endometrial receptivity: A case report. World J Clin Cases 2022;10(33):12079–88. doi: 10.12998/wjcc.v10.i33.12079
17. Paoli A, Mancin L, Giacona MC, Bianco A, Caprio M. Effects of a ketogenic diet in overweight women with polycystic ovary syndrome. J Transl Med. 2020;18(1). doi: 10.1186/s12967-020-02295-7
18. Paoli A, Rubini A, Volek JS, Grimaldi KA. Beyond weight loss: a review of the therapeutic uses of very-low-carbohydrate (ketogenic) diets. Eur J Clin Nutr 2013;67(8):789–96. doi: 10.1038/ejcn.2013.116
19. Papadakis G, Kandaraki EA, Paschou SA. Is dietary glycemic load associated with biochemical hyperandrogenemia in women with polycystic ovary syndrome? Clin Endocrinol (Oxf) 2012;77(3):346–52. doi: 10.1111/j.1365-2265.2012.04408.x
20. Ryrso CK, Dungu AM, Hegelund MH, Faurholt-Jepsen D, Pedersen BK, Ritz C, Lindegaard B, Krogh-Madsen R. Physical Inactivity and Sedentarism during and after Admission with Community-Acquired Pneumonia and the Risk of Readmission and Mortality: A Prospective Cohort Study. J Clin Med. 2022 Oct 7;11(19):5923. doi: 10.3390/jcm11195923. PMID: 36233789; PMCID: PMC9571502.
21. Wolf WM, Wattick RA, Kinkade ON, Olfert MD. Geographical prevalence of polycystic ovary syndrome as

determined by region and race/ethnicity. Int J Environ Res Public Health 2018;15(11):2589. doi: 10.3390/ijerph15112589

22. Zhao B, Wu H, Yao Q, Bai W, Kang J. A ketogenic diet alleviates the apoptosis of granulosa cells by inhibiting the activation of cGAS-STING signaling pathway in PCOS mice. Cell Commun Signal. 2024;22(1):568. doi: 10.1186/s12964-024-01939-6.