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Ketogenic versus Mediterranean Diet in the Management of PCOS: A Comparative Review on Endocrine Profiles, Lipid Remodeling, and Athletic Considerations.

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Abstract

Introduction. Polycystic ovary syndrome (PCOS) places unprecedented demands on the metabolic-endocrine system. Targeted dietary interventions and habitual modifications have proven highly effective in supporting reproductive reserve, thereby protecting the local ovarian microenvironment and boosting insulin sensitivity. Nevertheless, serious concerns stand regarding common therapeutic dilemmas and the risk of compromising skeletal muscle mass retention and high-intensity glycolytic exercise capacity.

Research objective. This literature review summarizes current knowledge on the pathophysiology, distinct kinetics, modulation of endocrine profiles, lipid remodeling, athletic considerations, and long-term compliance of the ketogenic diet (KD) versus the Mediterranean diet (MED) in patients with PCOS, with a focus on sequential clinical integration.

Methodology. The review is based on a structured search of peer-reviewed literature through May 2026. Keywords included: “polycystic ovary syndrome”, “ketogenic diet”, “Mediterranean diet”, “hyperandrogenism”, “insulin resistance”, and “sports performance”. Priority was given to clinical trials, randomized controlled trials, systematic reviews, and international consensus documents regarding the nutritional management of endocrine disorders. Articles were analyzed for their relevance to describing acute biochemical cutoff mechanisms versus gradual receptor optimization cascades.

Conclusions. Immediate carbohydrate restriction and targeted anti-inflammatory macronutrient models constitute significant progress in the care of patients with PCOS, helping to preserve ovulatory function and cardiometabolic health; however, their implementation requires careful monitoring of body composition parameters and avoidance of physical performance impairment. The use of a sequential therapeutic protocol is necessary to optimize endocrine balance, preserve lean tissue, and ensure sustainable, long-term lifestyle adherence.

Keywords: Polycystic ovary syndrome, ketogenic diet, Mediterranean diet, hyperandrogenism, sports performance.

Content

1. Introduction

From a medical perspective, polycystic ovary syndrome (PCOS) is no longer considered a gynecological issue solely. It is defined as a chronic metabolic-endocrine disorder rooted in tissue insulin resistance and secondary hyperinsulinemia [1, 2]. Importantly, this condition is not limited to patients with excess body fat, although in this group the cascade of clinical symptoms is significantly more severe [3]. The hormonal loop in this condition is not coincidental. Excess insulin circulating in the blood directly stimulates ovarian cells to overproduce androgens [4]. High levels of male hormones, in turn, effectively block normal ovulation and cause bothersome dermatological symptoms, such as acne or hirsutism [1]. These androgens also alter the patient's body composition, increasing fat accumulation in the abdominal region, which in turn exacerbates tissue resistance to insulin and closes this destructive, vicious cycle [5].

The second major aspect of the pathophysiology of PCOS is low-grade systemic inflammation and the accompanying chronic oxidative stress [6]. Inflammatory cytokines and free radicals directly damage insulin receptors, thus intensifying peripheral resistance and, in turn, driving the production of ovarian androgens. At the local level, the delicate ovarian microenvironment is severely disrupted, which leads to fertility issues and increases the risk of developing serious cardiovascular complications later in life [7]. The primary source of these inflammatory signals is linked to altered visceral adipose tissue. It continuously releases pro-inflammatory factors into the bloodstream, driving damage to the vascular endothelium [5].

Given this complex metabolic background, international clinical guidelines recognize lifestyle modification as the first-line therapy for all women with polycystic ovary syndrome [1, 8]. In this case, pharmacotherapy serves only as a supplement and cannot be treated as a substitute for changes in daily habits. For these to be effective, they must be based on the simultaneous implementation of a targeted weight-loss diet, consistent physical exercise lasting at least 150 minutes per week, and comprehensive behavioral support, including sleep hygiene and stress reduction [1, 3]. However, medicine does not impose a single, rigid dietary model, permitting both the ketogenic model—which serves as a rapid biochemical intervention—and the Mediterranean model —a highly stable, anti-inflammatory, long-term option [8]. This paper provides an in-depth comparison of both strategies in terms of their actual impact on patients' metabolism, fertility, and physical performance.

2. Nutritional Approaches: The Ketogenic Diet vs. the Mediterranean Diet

In PCOS, changing dietary habits is a key element of therapy aimed at supporting metabolic and hormonal parameters. The ketogenic and Mediterranean diets stand as distinct macronutrient strategies; nevertheless, both approaches present considerable therapeutic ability for reducing insulin resistance and chronic inflammation in patients with this diagnosis.

The fundamental difference between the two dietary models rests in the carbohydrate-to-fat ratio and the resulting metabolic state of the body. This entails a completely different selection of foods: those preferred and those eliminated from the diet.

The ketogenic diet is based on a radical restriction of carbohydrate intake, typically to 30–50 grams per day, with only 10–15 grams allowed during the induction phase to accelerate the onset of adaptive changes [1, 9]. Protein intake in this model is maintained at a level sufficient to preserve lean body mass, thereby classifying this diet as normal-protein [9, 10]. The main sources of energy are fats and proteins such as meat, eggs, fatty fish, cheese, butter, vegetable oils, avocados, and nuts and seeds. Permitted vegetables are limited to low-carbohydrate varieties, mainly leafy greens and cruciferous vegetables. Products high in carbohydrates and simple sugars, including grain products, most fruits, legume seeds, and starchy vegetables, are strictly limited or completely eliminated [4, 11, 12]. The goal of such a drastic restriction is to induce a state of nutritional ketosis, in which serum ketone body concentrations reach approximately 7–8 mmol/L. This enables the use of ketone bodies as energy substrates for peripheral tissues and the central nervous system, consequently minimizing the role of glucose. Interestingly, so-called Mediterranean-ketogenic versions are increasingly being implemented in clinical protocols for ketogenic diets, which aim to combine the metabolic state of ketosis with the intake of fats typical of the Mediterranean diet—namely olive oil and fish—at the expense of saturated fats of animal origin [4, 5, 12, 13, 14]. In studies on polycystic ovary syndrome, the very low-calorie ketogenic diet is particularly frequently analyzed; it is characterized by strict energy restriction to 500–800 kilocalories per day while maintaining the biochemical features of ketosis and a carbohydrate intake of 30–50 grams [1, 10].

Both dietary patterns analyzed demonstrate proven success in improving glycemia, reducing body weight, and optimizing the lipid profile. However, their biochemical mechanisms are different. The ketogenic diet is distinguished by a higher rate of initial weight loss and a rapid improvement in tissue insulin sensitivity, as indicated by a decrease in the HOMA-IR index [7, 10, 11]. Limitations comprise the risk of a temporary increase in LDL cholesterol levels and deficiencies in dietary fiber and certain micronutrients. It is also associated with relatively low long-term adherence due to strict dietary restrictions [10, 11, 15]. The Mediterranean diet, in turn, has a well-documented, unequivocally positive cardioprotective profile, strong antioxidant and anti-inflammatory effects. The nutritional density, long-term tolerability, and feasibility are also high [1, 6, 16]. In patients with type 2 diabetes and insulin resistance, both the ketogenic and Mediterranean diets are among the most effective dietary approaches for lowering glycated hemoglobin (HbA1c) and fasting glucose levels [1, 9, 11]. With regard to current scientific reports on PCOS, both diets effectively restore hormonal balance and lower insulin resistance. However, the evidence base for the ketogenic diet in this specific condition remains limited and is based primarily on short-term clinical interventions using a very low-calorie protocol [1, 6, 17].

3. Discussion

3.1. Endocrine Profiles and Androgen Suppression

In polycystic ovary syndrome, a multilevel pathophysiological loop plays a key role, in which peripheral insulin resistance, compensatory hyperinsulinemia, and hyperandrogenism mutually exacerbate one another. Excess circulating insulin interacts with luteinizing hormone (LH), directly stimulating ovarian cells to increase steroidogenesis and androgen secretion [1,

18]. At the same time, high insulin concentrations inhibit the synthesis of sex hormone-binding globulin (SHBG) in the liver. This results in a sharp increase in the free, bioavailable fraction of testosterone, which is responsible for the manifestation of clinical symptoms such as hirsutism, acne, and male-pattern baldness [3, 19]. Recent scientific reports indicate that hyperinsulinemia additionally enhances peripheral androgenogenesis by stimulating AKR1C3 expression in adipose tissue. Activation of this pathway leads to local, intracellular production of classic and 11-oxidized androgens (e.g., 11-ketotestosterone) directly within adipocytes, creating an independent, peripheral source of excess male hormones and worsening systemic metabolic dysfunction [20].

The implementation of a ketogenic diet, especially in a restrictive very low-calorie protocol (VLCKD), acts as an aggressive metabolic intervention. The elimination of glucose immediately cuts off stimulation of the insulin–androgen axis. The state of nutritional ketosis markedly lowers blood glucose and fasting insulin levels, accordingly improving peripheral tissue sensitivity, as evidenced by a decrease in the HOMA-IR index [17, 21]. During a typical 12-week intervention, SHBG and albumin concentrations increase significantly, resulting in a sharp drop in free, bioactive testosterone levels [1, 3]. At the central level, the ketogenic diet helps restore the function of the hypothalamic-pituitary-ovarian axis. This is demonstrated by reduced LH levels and an improved LH-to-FSH ratio, which, in turn, inhibits chronic stimulation of the theca cells [1, 6]. At the molecular level, this process is aided by the activation of AMPK and sirtuin 1 (SIRT1). Rapid loss of visceral fat also reduces aromatization in adipose tissue, thereby lowering peripheral estrogen production, resolving chronic hyperestrogenemia, and restoring normal hormonal feedback in the gonadotropin axis [1, 7].

In contrast to the ketogenic model, the Mediterranean diet and its low-carbohydrate modifications achieve endocrine goals gradually, relying on improved fatty acid quality and a high fiber intake. Randomized studies have shown that this model induces a gradual reduction in glucose, insulin, and HOMA-IR levels. It improves the QUICKI index, decreases total testosterone and basal LH levels, and normalizes the LH/FSH ratio [22]. This confirms that the resolution of hyperandrogenism is driven by long-term optimization of the insulin receptor signaling cascade, rather than a sudden biochemical cutoff [22]. Patients demonstrating high adherence to this model display lower baseline androgen and insulin concentrations and improved insulin sensitivity, even when energy intake is identical to that of the control groups [4]. It is connected to the presence of polyphenols and polyunsaturated fatty acids, which reduce oxidative stress and inflammation. Specifically, by stimulating proper Akt kinase phosphorylation and promoting the translocation of GLUT4 transporters to cell membranes [14, 23, 24]. It is important to refer to the role of a dietary fiber as well. Not only does it slow glucose absorption and flatten insulin peaks, but it also modulates the gut microbiome by increasing short-chain fatty acid (SCFA) production. Moreover, it supports glucose homeostasis and assures a stable, long-term increase in SHBG concentration [4, 5, 19].

The main difference between the two dietary models in the context of targeting hyperandrogenism boils down to the kinetics and starting point of the changes. The ketogenic diet has an immediate effect, causing a rapid drop in insulin levels and an immediate increase in hepatic SHBG synthesis after carbohydrate restriction. However, these data are primarily

derived from short-term time frames [3, 5, 17]. In contrast, the Mediterranean diet offers a proportionate approach. Decreases in androgens and increases in SHBG occur gradually. These are the long-term effects of improved receptor responsiveness and reduced chronic inflammation [4, 5, 22]. The ketogenic model rapidly lowers ovarian androgen production. It also reduces AKR1C3 activity in adipocytes by depleting glucose. In contrast, the Mediterranean model restores metabolic homeostasis in adipose and muscle tissues using high-quality nutrients. This leads to a slow, stable reduction in free testosterone over time [5, 6, 14, 23].

3.2. Ovulatory Function and the Ovarian Microenvironment

A substantial methodological disparity is apparent in the scientific literature: the evidence base for the ketogenic diet (KD) in a gynecological context is extensive, whereas data on the Mediterranean diet primarily focus on cardiometabolic markers. Clinical studies show that the KD shows a strong ability to restore menstrual cycle regularity, stimulate ovulation, and improve natural fertility rates [25, 26, 27, 28]. The regulatory effect of ketone bodies on neuroendocrine homeostasis may occur independently of absolute weight loss [29], and the implementation of short- and medium-term ketogenic restrictions can restore regular menstrual cycles and achieve a high pregnancy rate [27]. In obese patients undergoing in vitro fertilization (IVF), a very low-calorie ketogenic diet (VLCKD) protocol optimizes the ovulatory environment by lowering anti-Müllerian hormone (AMH) levels and the number of antral follicles, thus lowering the risk of ovarian hyperstimulation syndrome [30]. In contrast to the KD, the Mediterranean model shows significantly less pronounced reproductive changes, and its reproductive benefits are described primarily in general terms as an indirect effect of reduced peripheral insulin resistance [18, 30, 31].

The ketogenic model shows a clear kinetic advantage in resolving central endocrine dysfunctions in PCOS. Meta-analyses confirm that the KD induces a statistically significant reduction in luteinizing hormone (LH) levels, an increase in follicle-stimulating hormone (FSH), normalization of the LH/FSH ratio, and a decrease in total and free testosterone, accompanied by stimulation of hepatic SHBG synthesis [32]. Individual clinical protocols demonstrate that ketogenic interventions, including the VLCKD protocol, lead to a simultaneous increase in estradiol and progesterone on the twenty-first day of the cycle, which directly signals improved luteal function and normalization of ovarian function [25, 32]. Individual clinical protocols demonstrate that ketogenic interventions, including the VLCKD protocol, lead to a simultaneous increase in estradiol and progesterone on the twenty-first day of the cycle, which directly reflects improved luteal and ovarian function [33, 34, 35]. The Mediterranean diet is characterized by a significantly milder endocrine profile; although it improves gonadotropin levels, the extent of reduction in the pathological LH/FSH ratio and the rate of SHBG induction are significantly lower than in a state of ketosis [21, 35, 36]. Furthermore, the medical literature lacks systematic data evaluating the direct impact of the Mediterranean diet on AMH or progesterone levels, and in comparative studies of patients prior to IVF, only the VLCKD led to a rapid reduction in excessive, pathological ovarian reserve [18, 30, 31].

3.3. Lipid Remodeling and Cardiovascular Safety

Both dietary models show strong anti-inflammatory potential, reducing systemic cytokine secretion through weight loss and improved insulin sensitivity [18, 31, 37]. However, from a mechanistic perspective, the ketogenic diet displays a unique, direct signaling effect, in which the resulting beta-hydroxybutyrate and increased fatty acid oxidation can suppress the activity of the NLRP3 inflammasome and the NF-kappaB signaling pathway [38]. Blocking this inflammatory molecular complex interrupts the pathological cascade linking insulin resistance to hyperandrogenism, thereby protecting the local ovarian microenvironment and restoring normal folliculogenesis [38]. The Mediterranean diet exerts its antioxidant properties through a high intake of polyunsaturated fatty acids, fiber, and polyphenols that neutralize reactive oxygen species; however, current knowledge lacks studies measuring these markers directly in the ovarian tissue of PCOS patients, and evidence is limited to peripheral blood serum [18, 31, 37]. To summarize, the ketogenic diet exerts a stronger, faster short-term regulatory effect on the hypothalamic-pituitary-ovarian axis and on AMH, progesterone, and SHBG levels, thereby effectively restoring ovulatory cycles [25, 26, 27, 32]. The effect of the Mediterranean diet on direct ovarian markers is much milder and less well documented in the medical literature [18, 21, 30, 35].

Both dietary models competently modify the lipid profile in women with PCOS, employing different mechanisms and rates of change, although most of the available data are short-term. The ketogenic diet (VLCKD) acts as a rapid biochemical intervention, inducing a sharp decrease in triglyceride (TG) levels, total cholesterol (TC), and LDL-C, while simultaneously increasing HDL-C [19, 33]. Clinical trials have reported reductions in TC of nearly 40 mg/dL and LDL-C of 35 mg/dL, as well as increases in HDL-C from 50 to 65 mg/dL, findings confirmed by meta-analyses, though these indicate variable statistical significance of changes in HDL-C itself across certain models [32, 53]. In contrast, low-carbohydrate variants of the Mediterranean diet modify the lipid profile more gradually and harmoniously, demonstrating greater efficacy than classic low-fat diets [22]. However, the number of studies evaluating this approach straightforwardly in the PCOS population is significantly smaller, representing a gap in the current state of knowledge [17, 18].

Short-term randomized trials indicate that the ketogenic diet provides faster weight loss and a greater initial reduction in TG, TC, and LDL-C than the Mediterranean model, as confirmed by nine-week interventions [21, 35]. Nevertheless, in studies comparing the KD with a moderate-carbohydrate plant-based diet, the plant-based model more effectively improved the LDL-C, TG, and HDL-C profiles, despite greater weight loss and more pronounced regulation of hormonal parameters in the ketogenic group [55]. The PCOS phenotype is characterized by a highly atherogenic lipid profile with a predominance of small, dense LDL particles with high atherogenic potential and low HDL-C concentrations [37]. The ketogenic diet improves these indicators and lowers the TG/HDL ratio primarily by directly targeting insulin resistance [56], whereas Mediterranean-style diets leverage the protective effects of dietary fiber and the high-quality unsaturated fatty acids it provides [22].

Chronic dyslipidemia in PCOS increases the long-term risk of developing full-blown cardiovascular disease, and short-term implementation of a ketogenic diet reduces this risk

through rapid reduction of visceral fat and normalization of the HOMA-IR index, which relieves the vascular system [56]. However, the literature lacks data evaluating the long-term impact of high saturated fat intake in the ketogenic diet on the vascular endothelium and the development of atherosclerosis in young women over a multi-year period. The Mediterranean diet is a recognized standard of cardioprotection with the highest level of evidence, which permanently lowers blood pressure and reduces systemic inflammation [14]. Clinically, combining Mediterranean principles with controlled carbohydrate restriction represents the safest and most predictable long-term strategy [22], while the ketogenic model is an effective, rapid-acting tool for quickly overcoming metabolic blockage [17].

3.4. Sports Performance and Fitness Outcomes

Physical activity, particularly resistance and interval training, forms the basis of non-pharmacological treatment for insulin resistance in PCOS; therefore, the choice of dietary model must take into account its impact on exercise capacity. Carbohydrate-rich diets show a significant advantage over the ketogenic diet (KD) during high-intensity exercise and time trials, whereas ketosis does not improve maximum strength or peak power [39]. According to the position statement of the International Society of Sports Nutrition, carbohydrate restriction has a neutral or adverse effect on performance, particularly during maximal exercise [40]. Although adaptation to ketosis increases fatty acid oxidation, it does not improve performance in strength-power disciplines and sprints, where non-fat glycolysis remains the dominant energy pathway [41, 42, 43, 44]. Models with a higher carbohydrate intake provide higher peak power and a longer time to exercise failure [45]. Any performance metric benefits observed in the KD result solely from fat loss, not from actual optimization of muscle metabolism during lean-body sprints [46].

Increasing or preserving lean body mass is important for peripheral glucose uptake in women with PCOS. The ketogenic diet, despite its success in reducing body fat, carries a risk of muscle loss, which can account for up to one-third of total weight loss [15, 41]. Ketogenic restriction inhibits hypertrophic processes and limits strength and power gains, especially under energy-deficient conditions, even when adequate protein intake is maintained [40, 41, 44, 47]. Network evidence confirms that combining exercise with a ketogenic diet is one of the least efficient methods for retaining muscle mass in women trying to lose weight, yielding to classic calorie restriction and time-restricted feeding [48]. In contrast, the Mediterranean diet, defined by higher carbohydrate intake and anti-inflammatory potential, effectively preserves muscle glycogen stores and protects protein structures from catabolism, therefore reducing the risk of muscle loss associated with ketosis [15, 40, 48, 49, 50].

In a clinical context, in obese women with PCOS, the VLCKD protocol shows greater short-term success in reducing waist circumference, suppressing hyperandrogenism, and inducing ovulation compared to the Mediterranean diet [51]. However, high-intensity interval training (HIIT) requires immediate access to glucose, so the process of adapting to ketosis causes patients to experience energy slumps, chronic fatigue, and headaches, which impair adherence to training goals [40, 41, 42, 44]. The Mediterranean diet, based on low-glycemic-index carbohydrates and polyphenols, eliminates energy slumps and does not impair glycolytic capacity, consequently supporting performance during strength and high-intensity interval

exercises [45, 49, 50]. For active women with PCOS, the Mediterranean model represents a stable and safe long-term strategy for retaining muscle mass, whereas the ketogenic diet should be treated exclusively as a short-term, intensive tool for reducing severe obesity.

4. Conclusions and Applied Practices

4.1. Conclusions

Current clinical guidelines and systematic reviews indicate that the foundation of non-pharmacological treatment for polycystic ovary syndrome (PCOS) remains integrated lifestyle modification and a 5–10% reduction in body weight, rather than adopting a single, one-size-fits-all dietary model [1, 52]. Based on current medical knowledge, there is no international consensus favoring the ketogenic diet over the Mediterranean diet as the sole standard of care. This is because the key to boosting insulin sensitivity and restoring hormonal homeostasis is to generate a sustained energy deficit, which enables the effective use of both approaches, depending on the patient's individual phenotype and capabilities [2, 18, 28, 30].

A clinical analysis of both dietary strategies allows for a clear determination of their therapeutic role and optimal planning of interventions:

- Ketogenic diet (especially the VLCKD variant): It is a highly effective, short-term, intensive tool used as an emergency procedure [2, 17, 28, 30]. Meta-analyses confirm its powerful effects in rapidly reducing BMI, attenuating insulin resistance, suppressing free androgens, normalizing the gonadotropin ratio, and stimulating SHBG synthesis, thereby enabling the rapid restoration of ovulation prior to assisted reproductive procedures (IVF) [2, 28, 32, 51, 53]. Nevertheless, this model results in low long-term adherence—the dropout rate ranges from 27% to 49% due to high social pressure and biochemical rigor [28, 30]. Additionally, KD is associated with the risk of lean body mass loss, decreased tolerance for high-intensity interval training (HIIT), and numerous adverse effects (including acidosis, kidney stones, and dyslipidemia), which preclude it as a permanent lifestyle [1, 15, 40, 28].
- Mediterranean diet (MED/LC): It represents a safe, nutritionally complete, and strongly anti-inflammatory dietary pattern recommended as the default long-term model for PCOS [2, 28, 30, 36, 40]. It is defined by high patient adherence and an excellent organ safety profile [30, 32]. Thanks to its high intake of fiber, polyphenols, and MUFA and PUFA fatty acids, this model consistently improves lipid profiles, optimizes glucose-insulin metabolism, and provides long-term cardiometabolic protection, while also showing a positive impact on patients' psychological well-being and quality of life [2, 18, 40, 53, 54].

In practical terms, the most clinically justified strategy for women with severe obesity and severe metabolic and reproductive dysfunction is a sequential approach. It requires implementing a rigorous VLCKD protocol for a period of several to over a dozen weeks to rapidly break the health impasse, followed by a smooth transition of the patient to a

carbohydrate-controlled Mediterranean diet as a permanent, safe, and balanced lifestyle [2, 28, 30, 32, 40, 51].

The main paradigm and research perspective for the future persists the immediate need to conduct prospective, randomized head-to-head trials. These studies must directly compare the two models in a PCOS population over a multi-year period, with particular emphasis concerning their impact on specific ovarian parameters (including AMH), quality of life (PCOSQ questionnaire), risk of developing eating disorders (ED), and the prevalence of latent micronutrient deficiencies.

4.2. Practical Uses

Based on the synthesized evidence, physical performance and sports medicine professionals should implement the following recommendations for active women diagnosed with PCOS:

1. Training Customization: High-Intensity Interval Training (HIIT) and maximal strength disciplines require a steady glycolytic supply. For patients currently on a ketogenic protocol, training intensities must be adjusted downward during the first 2–4 weeks to reduce the acute metabolic energy slumps, chronic fatigue, and glycolytic impairment associated with adaptive ketosis.
2. Muscle Mass Protection: Practitioners must use targeted resistance training and ensure adequate protein intake (1.5–2.0 g/kg/day) when a patient uses the VLCKD approach, as carbohydrate restrictions under hypocaloric conditions are associated with significant skeletal muscle mass catabolism.
3. Long-Term Strategy: For long-term athletic preparation and stable cardiovascular performance, the Mediterranean diet remains the optimal baseline, as it prevents glycogen depletion and consistently supports the metabolic and high-intensity demands of concurrent athletic training.

Disclosure

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Conflicts of Interest

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