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Original Article

Precision Nutrition For PCOS/PMOS: Translating Dietary Patterns, Metabolic Phenotypes, And Nutraceutical Evidence Into Practical Care

Prasanna Cheemalamarri*

Clinical Dietitian, GBR Hospital, Hyderabad, India

ABSTRACT

Polycystic ovary syndrome (PCOS), renamed polyendocrine metabolic ovarian syndrome (PMOS) through a 2026 global consensus process, is a heterogeneous endocrine-metabolic condition in which insulin resistance, hyperandrogenism, adipose dysfunction, reproductive disturbance, and psychological burden interact across the life course. Nutrition is first-line care, yet a single "PCOS diet" is not supported by current evidence. This narrative review synthesizes recent guidelines, systematic reviews, meta-analyses, and selected randomized trials to develop a practical precision-nutrition framework for PCOS/PMOS. Evidence supports sustainable healthy eating patterns rather than one universal macronutrient prescription. Energy restriction is useful when clinically indicated; dietary quality remains important independent of weight change. DASH, Mediterranean-style, low-glycemic-index/load, and other minimally processed, fiber-rich patterns improve selected anthropometric and metabolic outcomes, while certainty for reproductive endpoints is lower. Lower-carbohydrate and ketogenic strategies may produce short-term benefits but require attention to nutritional adequacy, adherence, lipid response, fertility plans, and eating-disorder risk. Inositol, vitamin D, omega-3 fatty acids, and probiotics may be considered as adjuncts for selected patients, but heterogeneity in products, doses, and study quality prevents routine universal supplementation. A phenotype-informed approach should integrate glycemic status, body composition, lipid and liver risk, reproductive goals, medications, culture, food access, sleep, mental health, and patient preference. The review proposes an assessment algorithm, evidence-to-practice tables, and a food-first plate model. Nutrition should be framed as supportive, individualized chronic-disease care, not a cure, moral test, or replacement for medical treatment.

Keywords: Polycystic ovary syndrome; polyendocrine metabolic ovarian syndrome; precision nutrition; insulin resistance; dietary pattern; nutraceuticals

INTRODUCTION

Polycystic ovary syndrome has traditionally been defined by combinations of ovulatory dysfunction, clinical or biochemical hyperandrogenism, and polycystic ovarian morphology after exclusion of alternative diagnoses. The 2023 International Evidence-based Guideline emphasized that PCOS is not only a reproductive disorder; it is associated with metabolic, cardiovascular, dermatological, sleep, pregnancy, and psychological consequences that require longitudinal, person-centered care.[1] In 2026, a multistep global consensus recommended the name polyendocrine metabolic ovarian syndrome (PMOS) to better represent the condition's systemic biology and to reduce the misleading emphasis on ovarian "cysts."[2] Because both terms remain in clinical records and the literature, this review uses PCOS/PMOS.

The prevalence of PCOS varies substantially with diagnostic criteria, age, ethnicity, ascertainment, and access to care. Recent global synthesis estimates indicate that approximately one in ten reproductive-aged women may meet commonly used criteria, with important regional variation.[3] The burden is amplified by delayed diagnosis, fragmented care, weight stigma, infertility distress, acne and hirsutism, and elevated risks of impaired glucose tolerance and type 2 diabetes. Cardiovascular risk factors are common, and a large meta-analysis prepared for the international guideline reported higher odds of composite

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cardiovascular disease, ischemic heart disease, myocardial infarction, and stroke among women with PCOS.[4] These observations make nutrition relevant not merely to body weight but to lifelong metabolic risk reduction.

Insulin resistance is present across body-size categories, although its severity is often greater with central adiposity, sleep disturbance, fatty liver, inactivity, and genetic susceptibility. Compensatory hyperinsulinemia can stimulate ovarian androgen synthesis, reduce hepatic sex hormone-binding globulin, and interact with hypothalamic-pituitary-ovarian signaling. At the same time, androgen excess may promote visceral adiposity and worsen insulin action, creating a bidirectional cycle. Low-grade inflammation, adipokine dysregulation, oxidative stress, and alterations in the gut microbiome are plausible amplifiers rather than isolated causes. Nutrition may influence several nodes in this network, but no food or supplement has been shown to "reverse" the syndrome in every phenotype.[5,6]

Lifestyle management, including dietary change, physical activity, behavioral support, sleep, and reduction of sedentary time, is recommended for all people with PCOS/PMOS, including those who do not seek weight loss.[1,7] Health benefits can occur without a change on the scale. When higher weight contributes to metabolic or reproductive risk and weight reduction is a shared goal, a modest loss can improve insulin sensitivity, ovulation, and biochemical hyperandrogenism. However, repeated restrictive dieting, shame-based counseling, and unrealistic targets can worsen disengagement and eating pathology. Contemporary care therefore balances metabolic risk reduction with weight-inclusive communication, patient autonomy, and screening for disordered eating.

Popular media commonly prescribe dairy-free, gluten-free, very-low-carbohydrate, "hormone-balancing," or supplement-heavy regimens as uniquely necessary for PCOS. These claims often extrapolate from mechanistic studies, uncontrolled cohorts, or short trials. The 2023 guideline found no evidence that one diet composition is superior for the full range of outcomes and recommended sustainable eating consistent with general healthy dietary guidance, individualized to preferences and goals.[1] Since then, network meta-analysis and systematic reviews have refined, not overturned, this conclusion: specific patterns may rank better for selected metabolic outcomes, but certainty, durability, and head-to-head evidence remain limited.[8,9]

The objective of this review is to translate recent nutrition evidence into a clinically usable framework. It asks five questions: ([1]) Which dietary patterns have the strongest support for metabolic and reproductive outcomes? ([2]) How should macronutrient quality and meal structure be operationalized? ([3]) When are supplements reasonable, uncertain, or potentially harmful? ([4]) How can recommendations be adapted to metabolic phenotype, reproductive goals, culture, medication, and food access? and ([5]) Which research gaps prevent genuinely precise nutrition care? The focus is adult PCOS/PMOS; adolescent, pregnancy, and fertility-treatment considerations are highlighted where relevant but require specialist adaptation.

Genetic susceptibility + life-course exposures	Insulin resistance and hyperinsulinemia	Ovarian/adrenal androgen excess	Ovulatory and follicular dysfunction
Adipose dysfunction; low-grade inflammation	Dyslipidemia, MASLD, glycemic risk	Gut microbiome and intestinal barrier	Sleep, stress, stigma, mental health
Nutrition intervention targets: energy balance when indicated; carbohydrate quality; fiber and plant diversity; adequate protein; unsaturated fats; micronutrient repletion; culturally acceptable behavior change			

Figure 1. Nutrition-relevant systems model of PCOS/PMOS. Original conceptual synthesis by the author; arrows indicate interacting pathways rather than proven linear causality.

MATERIALS AND METHODS

A narrative review design was selected because the objective was clinical translation across heterogeneous evidence rather than estimation of a single pooled effect. The evidence search was updated through July 2026. PubMed/MEDLINE and publicly available guideline and journal sources were searched using combinations of "polycystic ovary syndrome," "polyendocrine metabolic ovarian syndrome," "diet," "nutrition," "dietary pattern," "Mediterranean," "DASH," "glycemic index," "low carbohydrate," "ketogenic," "time-restricted eating," "inositol," "vitamin D," "omega-3," "probiotic," "lifestyle," "systematic review," "meta-analysis," and "randomized trial." Reference lists of key reviews were used to identify foundational studies.

Priority was given to the 2023 International Evidence-based Guideline, the 2026 global terminology consensus, systematic reviews and meta-analyses published from 2023 onward, and randomized trials that illustrated clinically important benefits or limitations. Earlier high-quality evidence was retained when it addressed a question not superseded by recent work, such as glycemic index/load. Studies were considered in relation to anthropometry, insulin resistance, glycemia, lipids, inflammation, androgen-related outcomes, menstrual function, fertility, adherence, safety, and patient burden.

Because dietary trials differ in energy prescription, comparator diets, duration, phenotype, medication use, outcome definition, and adherence, numerical effects were not pooled de novo. Conclusions were graded qualitatively as relatively strong, moderate, emerging, insufficient, or potentially harmful. "Relatively strong" indicates consistent direction across higher-level syntheses for a defined outcome, not certainty that the intervention is superior for every patient. The review did not include individual patient data, human participants, or animal experiments; therefore ethics committee approval was not required.

The term precision nutrition is used pragmatically rather than genomically. It refers to aligning dietary strategy with clinical phenotype, preferences, context, and response monitoring. This definition avoids premature claims that microbiome, metabolomic, or genetic tests can currently select a proven optimal PCOS diet. Recommendations were additionally screened for feasibility, nutritional adequacy, weight stigma, supplement quality, pregnancy safety, medication interaction, and risk of disordered eating.

Domain	Clinical meaning	Nutrition relevance
PCOS/PMOS terminology	PCOS is the established literature term; PMOS is the 2026 consensus name emphasizing polyendocrine and metabolic features.	Use both terms during transition. The name change reinforces life-course metabolic assessment without implying every patient has obesity or insulin resistance.
Ovulatory dysfunction	Irregular or absent ovulation after appropriate assessment.	Energy adequacy, weight trajectory, glycemic health, and eating-disorder screening matter. Severe restriction can itself disrupt cycles.
Hyperandrogenism	Clinical signs such as hirsutism/acne or biochemical elevation.	May improve indirectly through reduced hyperinsulinemia; nutrition is adjunctive and should not delay effective dermatologic or endocrine therapy.
Ovarian morphology/AMH	Imaging or biomarker criteria used within age-appropriate diagnostic algorithms.	No dietary pattern should be judged solely by changes in ovarian morphology or AMH.

Metabolic phenotype	Glycemic abnormality, central adiposity, dyslipidemia, hypertension, fatty liver, or sleep apnea risk.	Determines urgency and monitoring; supports cardiometabolic dietary patterns and medical co-management.
Psychological context	Depression, anxiety, body-image distress, and disordered eating are common.	Counseling must avoid moralizing food or weight. Restrictive plans require caution and specialist support.

Table 1. Terminology, Diagnostic Domains, And Nutrition Relevance

RESULTS AND DISCUSSION

Dietary Patterns: No Universal Winner, But Meaningful Differences By Outcome

The strongest cross-cutting finding is that dietary intervention can improve PCOS/PMOS outcomes, but superiority depends on what is measured. A 2024 network meta-analysis of randomized trials ranked low-calorie diets highly for body weight and body mass index, while DASH performed well for fasting glucose, fasting insulin, HOMA-IR, and triglycerides.[8] Ranking probabilities should be interpreted cautiously because networks inherit limitations of small trials, short follow-up, heterogeneous comparators, and varying energy deficits. Still, the results support choosing patterns by metabolic objective rather than searching for a single syndrome-specific diet.

Mediterranean-style eating emphasizes vegetables, legumes, fruits, whole grains, nuts, seeds, olive oil, fish, and minimally processed foods, with flexible inclusion of dairy, eggs, and poultry. A systematic review across female reproductive health found favorable associations, although PCOS-specific trial evidence was limited.[10] More recent PCOS trials suggest feasibility and improvements in diet quality and selected metabolic endpoints, but robust evidence for ovulation, live birth, or long-term disease events remains insufficient. The Mediterranean pattern is best viewed as a high-quality template that can be adapted to local cuisines, not as a requirement to consume Mediterranean foods.

DASH shares many Mediterranean features but places greater structure around vegetables, fruits, whole grains, legumes, nuts, low-fat dairy, lean proteins, and sodium reduction. In PCOS trials, calorie-restricted DASH interventions have improved insulin resistance, triglycerides, inflammatory markers, and anthropometry. The network meta-analysis ranked DASH particularly well for several glycemic outcomes.[8] Its limitations include intensive trial support, difficulty separating energy restriction from food-pattern effects, and uncertainty in lean PCOS.

Low-glycemic-index or low-glycemic-load strategies prioritize carbohydrate quality and portion, replacing refined grains, sweets, and sugar-sweetened beverages with intact whole grains, legumes, vegetables, and appropriately portioned fruit. Earlier meta-analysis of randomized trials found improvements in insulin sensitivity and selected reproductive outcomes.[11] Clinical teaching should emphasize minimally processed carbohydrate, fiber, protein and fat pairing, and meal consistency rather than memorizing numerical GI lists.

Energy-restricted diets can improve weight-related and metabolic outcomes when higher weight and positive energy balance are clinically relevant. The method of creating a deficit is less important than safety, adequacy, adherence, and preservation of lean mass. For people with normal weight, recent unintentional weight loss, pregnancy, active eating disorder, or high training demands, energy restriction may be inappropriate.

Lower-carbohydrate diets are heterogeneous. A 2024 randomized trial comparing a portfolio moderate-carbohydrate diet with a ketogenic diet reported improvements in both groups, with outcome-specific differences.[12] Ketogenic interventions may be difficult to sustain and can reduce fiber and micronutrient variety; LDL cholesterol may rise in susceptible individuals. They should not be presented as necessary for PCOS or used casually during pregnancy attempts, pregnancy, lactation, or in patients with eating-disorder risk.

Time-restricted eating and intermittent fasting modify timing rather than food identity. Evidence in PCOS/PMOS remains emerging, and small studies cannot establish long-term reproductive safety or superiority. Until stronger evidence exists, time restriction should be optional, modest, and stopped when it impairs adequacy, menstrual health, mental health, or adherence.

Vegetarian and plant-forward diets have not been sufficiently tested as distinct PCOS interventions, yet their components align with cardiometabolic care. A well-planned vegetarian or vegan diet can be suitable, with attention to protein, vitamin B^{12}, iron, iodine, calcium, vitamin D, zinc, and omega-^{3} sources.

Dietary strategy	Weight	Insulin resistance	Lipids	Androgens/cycles	Long-term safety
Mediterranean-style	Moderate	Moderate	Moderate	Emerging	Moderate
DASH	Moderate	Relatively strong	Moderate	Emerging	Moderate
Low-GI/GL	Emerging	Moderate	Emerging	Emerging	Moderate
Energy-restricted	Relatively strong	Moderate	Emerging	Emerging	Moderate
Lower-carbohydrate	Moderate	Moderate	Moderate	Emerging	Emerging
Ketogenic	Moderate	Moderate	Emerging	Emerging	Insufficient
Time-restricted eating	Emerging	Emerging	Insufficient	Insufficient	Insufficient

Figure 2. Qualitative certainty and clinical usefulness of dietary strategies. Ratings synthesize recent reviews and guideline conclusions and are not a formal GRADE assessment.

Pattern	Potential advantages	Important limitations	Best-fit context
Mediterranean-style	High fiber and unsaturated fat; cardiometabolic alignment; flexible and food-based.	PCOS-specific reproductive outcomes remain uncertain; access and cultural translation matter.	Long-term default pattern, dyslipidemia, inflammation, family-based eating.
DASH	Strong signal for insulin resistance, glucose, triglycerides, and blood pressure.	Often tested with calorie restriction and intensive support.	Hypertension risk, insulin resistance, high sodium or ultra-processed intake.
Low GI/GL	Improves carbohydrate quality; may support insulin sensitivity and menstrual outcomes.	GI tables can be burdensome and do not capture the whole meal.	Postprandial symptoms, prediabetes, structured carbohydrate guidance.

Energy-restricted balanced diet	Most direct evidence for weight loss when indicated.	Risk of weight cycling, lean-mass loss, micronutrient inadequacy, stigma.	Higher weight with shared weight-loss goal and monitoring.
Moderate lower-carbohydrate	May reduce triglycerides and glycemic excursions while retaining food variety.	Definitions vary; benefit may reflect energy reduction.	Marked insulin resistance, high refined-carbohydrate intake.
Ketogenic/very low carbohydrate	Short-term weight and metabolic changes in some trials.	Limited long-term data; constipation, micronutrient gaps, LDL response.	Selected, closely monitored adults; not a universal first-line diet.
Time-restricted eating	Simple rule for some patients; may reduce late eating.	Small evidence base; may worsen disordered eating.	Optional experiment in carefully selected adults with monitoring.

Table 2. Dietary Patterns In PCOS/PMOS: Likely Benefits, Limitations, And Best-Fit Contexts

Macronutrient Quality And Meal Architecture

Carbohydrate should be discussed in terms of quality, quantity, distribution, and context. The most defensible target is replacement of refined grains, confectionery, and sugar-sweetened beverages with fiber-rich foods. A gradual fiber goal, often approximately 25-[35] g/day depending on energy needs and tolerance, can improve satiety, stool quality, and glycemic response.

Protein supports satiety and lean mass, particularly during energy restriction. A practical approach is to include a protein-rich food at meals and distribute intake across the day. Kidney disease, pregnancy, athletic training, and food insecurity require individualized targets.

Fat quality is more important than extreme fat avoidance. Replacing saturated and trans fats with monounsaturated and polyunsaturated fats supports lipid management. Omega-3-rich foods are preferable to indiscriminate high-dose supplements.

Meal frequency should reflect symptoms, medication, schedule, and preference. Pairing carbohydrate with protein, fiber, and unsaturated fat often improves satisfaction and postprandial response. Continuous glucose monitors may be educational in diabetes, but routine use in normoglycemic PCOS can increase anxiety and is not yet evidence-based.

Ultra-processed food is a useful population concept but a blunt individual label. Clinicians can identify high-impact substitutions: water instead of sugary drinks, whole fruit instead of juice, and beans or lentils in place of part of refined starch or processed meat.

Nutrition target	Practical translation	Monitoring/cautions
Carbohydrate quality	Choose legumes, intact whole grains, vegetables, whole fruit, unsweetened dairy; reduce sugary drinks and refined snacks.	Adjust to glycemia, activity, medication, tolerance. Gluten avoidance unnecessary without clinical indication.
Fiber and plant diversity	Increase vegetables, pulses, fruit, whole grains, nuts, seeds gradually; aim for weekly variety.	Titrate for gastrointestinal tolerance; maintain hydration.
Protein adequacy	Include a protein-rich food at each main meal; plant and animal sources per preference.	Higher intake is not automatically better; adapt in kidney disease, pregnancy, athletic training.
Fat quality	Replace butter, excess ghee, fatty/processed meat, and trans fats with unsaturated oils, nuts, seeds, fish.	Monitor LDL cholesterol with ketogenic or very-high-fat approaches.
Energy density	Use high-volume vegetables, soups, pulses, lean protein when weight loss is a shared goal.	Avoid aggressive deficits, chronic hunger, fatigue, or menstrual deterioration.
Meal structure	Use regular meals or a planned eating window; pair carbohydrate with protein/fiber/fat.	Screen for binge-restrict cycles; accommodate shift work, medication, culture.

Table 3. Food-First Macronutrient Translation

Micronutrients And Nutraceuticals

Supplements occupy a disproportionate share of PCOS marketing. The appropriate sequence is to identify a plausible indication, review food intake and laboratory data, consider product quality and interactions, define an outcome, and reassess. Supplements should not be stacked without a plan.

Inositol has the most developed PCOS-specific evidence among commonly used supplements. A systematic review conducted for the international guideline found potential improvements in selected metabolic measures and possible reproductive benefits, but substantial heterogeneity and low-to-moderate certainty limited firm recommendations.[13] A separate meta-analysis of randomized trials also reported improvements in several metabolic and hormonal outcomes.[14] It is reasonable to discuss inositol as an optional adjunct while explaining uncertainty regarding live birth and superiority to established treatments.

Vitamin D deficiency should be identified and treated according to standard clinical guidance. A 2024 dose-response meta-analysis evaluated vitamin D status in PCOS, but supplementation evidence for syndrome-specific outcomes remains inconsistent.[15] Testing and replacement are preferable to universal high-dose therapy.

Omega-^{3} fatty acids may improve triglycerides and selected inflammatory or metabolic measures.[16] Food sources such as oily fish are preferred when acceptable. Claims that omega-^{3} supplements reliably restore ovulation or fertility are not established.

Probiotics, prebiotics, and synbiotics are supported by a growing mechanistic literature linking gut microbial ecology with insulin resistance, inflammation, and androgen metabolism. A 2024 systematic review of randomized trials reported improvements in selected

glycemic, inflammatory, and hormonal markers, but strains, doses, duration, and products were heterogeneous.[17] Another review emphasized that microbiome findings are not yet sufficient to prescribe a universal "PCOS probiotic."[18]

Magnesium, zinc, selenium, chromium, N-acetylcysteine, coenzyme Q^{10}, carnitine, cinnamon, curcumin, berberine, and herbal mixtures have been studied with mixed results. Recent comprehensive reviews catalogue possible changes in biochemical markers but repeatedly identify small samples, short duration, and uncertain clinical significance.[19,20] Berberine and concentrated herbal products can interact with medicines and are not automatically safe in pregnancy.

Folate is indicated before conception and during early pregnancy according to national guidance. Vitamin B^{12} deserves attention in vegans and in long-term metformin users. Calcium and vitamin D should be assessed when dairy is excluded.

Supplement	Possible role	Why certainty is limited	Practical caution
Inositol	Selected metabolic and ovulatory outcomes may improve.	Formulations, ratios, dose, comparators, and fertility outcomes vary.	Discuss as adjunct, not replacement for proven treatment; choose quality-assured product.
Vitamin D	Correct documented deficiency; possible metabolic/reproductive effects.	Benefits may reflect correction of deficiency rather than a PCOS-specific effect.	Test when appropriate; avoid chronic high-dose use without monitoring.
Omega-3 fatty acids	May lower triglycerides and influence inflammation.	Dose/formulation vary; reproductive outcomes uncertain.	Prefer food sources; consider bleeding risk and product quality.
Probiotic/synbiotic	Selected glycemic, inflammatory, and androgen markers may improve.	Strain-specific effects, small trials, short follow-up.	Do not generalize one product's evidence to all products.
N-acetylcysteine/CoQ10	Emerging evidence for insulin sensitivity or fertility-treatment adjuncts.	Limited clinical endpoints and inconsistent protocols.	Review medication interactions and pregnancy-treatment context.
Herbal products/berberine	Potential metabolic effects in small studies.	Variable composition, contamination, pharmacologic activity.	Avoid unsupervised use during conception, pregnancy, liver disease, polypharmacy.

Table 4. Selected Supplements: Evidence-Informed Use And Cautions

Phenotype-informed Precision Nutrition

Precision begins with assessment rather than a branded diet. At minimum, nutrition care should consider age, diagnostic confidence, menstrual pattern, hyperandrogenic symptoms, blood pressure, waist or central adiposity when acceptable, weight trajectory, glycemic status, lipids, liver risk, sleep apnea symptoms, medications, reproductive plans, mental health, disordered eating, gastrointestinal symptoms, dietary pattern, food security, cultural

practices, cooking resources, and readiness for change.

For insulin-resistant or prediabetic phenotypes, the priority is a sustainable cardiometabolic pattern with high fiber, reduced sugary beverages and refined starch, adequate protein, regular activity, and weight reduction when indicated. Metformin or anti-obesity medication may be appropriate through medical care; nutrition should anticipate gastrointestinal effects and vitamin B^{12} monitoring with long-term metformin.

For lean PCOS/PMOS, automatic calorie restriction is inappropriate. Insulin resistance can still occur, so glycemic screening remains necessary, but the focus may be meal quality, adequate energy, resistance exercise, sleep, and management of reproductive or androgenic symptoms.

For dyslipidemia or fatty liver risk, Mediterranean-style or DASH eating, unsaturated fat substitution, soluble fiber, weight management when indicated, and reduction of alcohol and sugar-sweetened beverages are priorities. Very-low-carbohydrate diets require lipid follow-up because triglycerides may fall while LDL cholesterol rises.

For fertility and preconception, the goal is nutritional adequacy and metabolic optimization, not maximal short-term weight loss. Letrozole remains first-line pharmacologic ovulation induction in appropriate anovulatory infertility under specialist care; nutrition can support but should not delay fertility evaluation.[1]

Adolescents require age-appropriate diagnosis and protection from weight stigma. Pregnancy and lactation require obstetric and dietetic adaptation; ketogenic diets, fasting, and nonessential supplements should not be assumed safe. The psychological phenotype can determine whether a theoretically effective diet is harmful; warning signs include fear-driven food elimination, binge-restrict cycling, compulsive weighing, and avoidance of social eating.

Clinical context	First nutrition priorities	Avoid/monitor
Prediabetes/marked insulin resistance	DASH, Mediterranean, or low-GI structure; reduce liquid sugar/refined starch; fiber, protein distribution, activity.	Monitor glycemia, medication effects, overly restrictive carbohydrate targets.
Higher weight with weight-loss goal	Moderate energy deficit, high-volume foods, adequate protein, resistance exercise.	Weight stigma, rapid loss, lean-mass loss, micronutrient gaps.
Lean PCOS/PMOS	Adequate energy, diet quality, resistance exercise, sleep, glycemic screening.	Routine calorie restriction or assuming insulin resistance is absent.
High triglycerides/fatty liver risk	Unsaturated fats, soluble fiber, reduce sugary drinks/alcohol, weight management if appropriate.	Lipid/liver monitoring; caution with high-saturated-fat ketogenic diets.
Pregnancy planning/fertility treatment	Folate/micronutrient adequacy, stable nourishing pattern, coordinated fertility care.	Extreme diets, unverified herbs, delayed medical evaluation.
Disordered eating/body-image distress	Regular adequate meals, flexible structure, weight-neutral goals, specialist referral.	Fasting, rigid elimination, frequent weighing, moral language about food.

Table 5. Phenotype-Informed Nutrition Priorities

Non-starchy vegetables + whole fruit	Healthy fats (nuts, seeds, olive oil, fish)
High-fiber carbohydrate	Protein-rich foods
Water / unsweetened drinks	

Figure 3. A practical PCOS/PMOS plate framework. Portions should be individualized; the model is not intended as a rigid prescription.

From Assessment To Prescription: A Practical Clinical Pathway

Step 1 is to define the patient's goals in her own terms. Common goals include more predictable cycles, reduced diabetes risk, improved energy, fertility, weight change, less food preoccupation, better gastrointestinal comfort, or easier family meals.

Step 2 is risk stratification. Glycemic testing, lipids, blood pressure, sleep symptoms, family history, and liver risk determine clinical urgency. Any red flag, such as rapid virilization, severe hyperglycemia, suspected eating disorder, pregnancy complication, or significant depression, requires medical assessment rather than nutrition-only management.

Step 3 is selection of the least restrictive effective dietary pattern. For many patients this is a culturally adapted Mediterranean/DASH hybrid: half the plate non-starchy vegetables when available, a quarter protein-rich foods, a quarter high-fiber carbohydrate, plus unsaturated fat and water.

Step 4 is one to three measurable behaviors, such as replacing sugary drinks with unsweetened alternatives, adding pulses at meals, or eating a protein-rich breakfast on workdays. Behavioral specificity matters more than a long list of prohibitions.

Step 5 is reassessment after an appropriate interval, commonly 8-[12] weeks for behavior and selected metabolic markers. Outcomes should include symptoms, adherence, quality of life, menstrual pattern, energy, gastrointestinal tolerance, and psychological burden, not only weight.

Eating occasion	Example components	Purpose/adaptation
First meal	Vegetable omelet with whole-grain roti/toast and fruit; or unsweetened yogurt/soy yogurt with oats, berries, nuts, and seeds.	Protein + fiber; dairy-free option per preference or intolerance.
Midday meal	Large vegetable portion; lentils/beans, fish, chicken, tofu, or paneer; brown rice, millet, quinoa, or whole-grain roti.	Culturally adaptable plate structure; starch portion varies with activity and goals.
Optional snack	Fruit with nuts; roasted chickpeas; yogurt; hummus with vegetables; or no snack if not hungry.	Prevents extreme hunger for some; not mandatory.
Evening meal	Vegetable and bean soup with whole-grain bread; or stir-fry with tofu/fish and mixed vegetables.	Emphasize vegetables, protein, and minimally processed carbohydrate.
Treat foods	Planned portion of preferred dessert or snack within an otherwise adequate pattern.	Flexible inclusion may improve adherence and reduce all-or-nothing eating.

Table 6. Example One-Day Flexible Meal Framework

What The Evidence Supports With Greatest Confidence

Nutrition and lifestyle are foundational for all PCOS/PMOS phenotypes, but foundational does not mean sufficient as monotherapy. Hormonal contraception, metformin, anti-obesity medicines, antiandrogens, fertility treatment, dermatologic care, and psychological treatment may be needed alongside nutrition, not instead of it.

Overall dietary quality and sustainability matter more than allegiance to a named diet. The common denominator across better-performing patterns is reduction of refined and ultra-processed foods, greater fiber and plant-food diversity, adequate protein, substitution of unsaturated for saturated fat, and an energy intake aligned with needs and goals.[8]

Weight loss can be therapeutic when higher adiposity contributes to risk, yet weight-neutral improvements are also clinically meaningful. Supplement evidence remains adjunctive and uneven: inositol is a reasonable shared-decision option but not a proven cure; vitamin D should primarily correct deficiency; omega-3 and probiotic interventions may target selected metabolic features.

Areas Of Controversy

The carbohydrate debate illustrates how binary messaging can distort evidence. Insulin resistance does not imply that carbohydrate is toxic. Very-low-carbohydrate diets may improve short-term markers through energy reduction and weight loss, but similar mechanisms can be achieved with less restrictive patterns.

Dairy and gluten elimination are widely promoted despite absence of a PCOS-specific indication. Gluten avoidance is essential in celiac disease and may help diagnosed non-celiac sensitivity, but routine elimination raises cost and can reduce fiber and fortification.

Ketogenic diets create a tension between short-term efficacy and long-term implementation. A supervised trial may be acceptable to a well-informed adult, but the evidence base is too small to normalize ketogenic eating as first-line reproductive care.

The microbiome is scientifically compelling but commercially premature. Direct-to-consumer microbiome tests cannot currently prescribe a validated PCOS diet. The PMOS name change may improve recognition of metabolic and multisystem burden, but it also carries risk if "metabolic" is interpreted as synonymous with obesity or personal responsibility.

Implementation In Diverse Settings

Cultural adaptation is not an optional add-on. Legumes, vegetables, whole grains, fermented foods, nuts, seeds, herbs, and unsaturated fats exist across culinary traditions. Counseling should begin with the patient's staples and identify preparation, portion, and pairing changes.

Food insecurity changes the prescription. Frozen produce, canned beans, eggs, tinned fish, seasonal fruit, oats, and fortified staples may be more feasible than specialty "PCOS foods." A plan that requires expensive berries, protein powders, or imported grains is not precision care.

Interdisciplinary care is ideal: dietitians translate evidence into meals; clinicians evaluate endocrine and metabolic risk; psychologists address distress and eating behavior; exercise professionals support fitness and muscle health; fertility specialists coordinate conception care.

Research Priorities

Future trials should recruit across body-size, ethnic, socioeconomic, adolescent, and life-stage groups and report diagnostic phenotype, with sufficient duration to assess menstrual regularity, fertility, weight regain, diabetes progression, cardiovascular markers, and safety.

Core outcome sets are needed. Trials frequently report dozens of biochemical measures while neglecting quality of life, hunger, adherence, cost, eating-disorder symptoms, live birth, and patient-defined benefit. Supplement trials need independently verified products, transparent dose and formulation, adverse-event reporting, and pregnancy surveillance.

Gap	Why it matters	Recommended design feature
Long-term comparative effectiveness	Most diet trials are short and cannot establish durability or clinical events.	Pragmatic head-to-head trials of major patterns for 12+ months.
Reproductive endpoints	Biochemical changes do not guarantee ovulation, pregnancy, or live birth.	Standardized menstrual measures and adequately powered fertility outcomes.
Lean and diverse phenotypes	Evidence is dominated by overweight/obese adult cohorts.	Stratified recruitment by BMI, ethnicity, age, and metabolic phenotype.
Eating-disorder safety	Restrictive diets may worsen a common comorbidity.	Baseline/follow-up validated screening, adverse-event reporting.
Supplement quality	Commercial formulations differ from trial products.	Independent assay, formulation transparency, interaction/pregnancy monitoring.
Implementation and equity	Food access, cost, and cultural fit determine real-world impact.	Co-designed interventions, cost-effectiveness, community settings.

Table 7. Priority Research Gaps

CONCLUSION

Nutrition is essential in PCOS/PMOS care, but the best evidence does not support a single curative diet. Sustainable patterns rich in vegetables, legumes, whole grains, fruit, adequate protein, and unsaturated fats, with reduced sugary drinks, refined grains, and excess ultra-processed foods, provide a defensible foundation. DASH, Mediterranean-style, and low-GI approaches can be selected according to glycemic, lipid, blood-pressure, weight, reproductive, and cultural goals. Energy restriction is useful only when clinically indicated, desired, and safely supported. Lower-carbohydrate, ketogenic, and time-restricted approaches may help selected adults but require careful monitoring and should not be universal prescriptions. Supplements are adjuncts: inositol has promising but uncertain evidence; vitamin D should correct deficiency; omega-3 and probiotics may target selected metabolic outcomes; unregulated combinations warrant caution. Precision nutrition in PCOS/PMOS is therefore a structured process of risk assessment, shared goal setting, least-restrictive pattern selection, behavioral support, and multidimensional follow-up, integrated with appropriate endocrine, reproductive, and mental-health care.

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AUTHOR CONTRIBUTIONS

[Author initials — REQUIRED]: conceptualization, literature review, evidence synthesis, writing (original draft), writing (review and editing), and approval of the final manuscript.

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