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COMBINED VERSUS SINGLE-MODALITY LIFESTYLE INTERVENTIONS ON INSULIN RESISTANCE AND HORMONAL PARAMETERS IN WOMEN WITH POLYCYSTIC OVARY SYNDROME: A STRUCTURED NARRATIVE REVIEW

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ABSTRACT

Background: Polycystic ovary syndrome (PCOS) is the most common endocrine disorder in women of reproductive age, with an estimated global prevalence of 8-13% depending on the population studied and diagnostic criteria applied. Its pathophysiology involves insulin resistance (IR) and hyperandrogenism, which mutually reinforce each other: elevated insulin drives ovarian androgen overproduction and suppresses sex hormone-binding globulin (SHBG), while excess androgens themselves worsen IR. Both IR and hyperandrogenism are modifiable through lifestyle interventions - specifically dietary modification and physical activity - making them the primary targets of non-pharmacological management in PCOS. Lifestyle interventions are recommended as first-line therapy, but the comparative effectiveness of combined versus single-modality approaches on IR and hormonal parameters remains unclear.

Aim: To synthesise and compare the effects of combined lifestyle interventions (diet plus exercise) versus single-modality approaches (diet alone or exercise alone) on markers of insulin resistance and key hormonal parameters in women with PCOS.

Material and methods: A comprehensive literature search was conducted across PubMed/MEDLINE, Embase, and Cochrane Library databases. Search terms included polycystic ovary syndrome, PCOS, insulin resistance, HOMA-IR, lifestyle intervention, combined intervention, diet, exercise, testosterone, and SHBG. Studies evaluating lifestyle interventions in women with PCOS and reporting insulin resistance and/or hormonal outcomes were included, with no restriction on study design.

Results: Both single-modality and combined interventions improved insulin sensitivity and hormonal profiles. Available evidence suggests that combined diet-and-exercise programmes may produce broader improvements in metabolic (HOMA-IR, fasting insulin) and hormonal (free testosterone, SHBG) markers than either approach applied alone; however, direct comparative evidence remains limited and heterogeneous. Vigorous aerobic exercise and resistance training have been associated with the strongest independent effects on IR in the reviewed evidence, while low-glycaemic-index and Mediterranean diets improved hormonal balance through complementary mechanisms - principally reduction of postprandial hyperinsulinaemia and attenuation of chronic low-grade inflammation respectively.

Conclusions: The available evidence suggests that combined lifestyle interventions may produce greater improvements in insulin resistance and hormonal parameters in women with PCOS than single-modality approaches, though the current evidence base is limited by the quality and number of available trials. Consistent with this, international guidelines recommend multimodal programmes integrating structured exercise with evidence-based dietary strategies as first-line PCOS care. Further large-scale randomised controlled trials directly comparing combined versus single-modality protocols are needed.

KEYWORDS

Polycystic Ovary Syndrome, PCOS, Insulin Resistance, Lifestyle Intervention, Combined Intervention, Exercise, Diet, Hormonal Parameters, HOMA-IR, Hyperandrogenism

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1. Introduction

1.1. Epidemiology and Metabolic Burden of PCOS

Polycystic ovary syndrome (PCOS) is the most prevalent endocrine disorder among women of reproductive age, with an estimated global prevalence of 8-13% depending on the population studied and diagnostic criteria applied. [1] The syndrome is diagnosed according to the Rotterdam criteria. At least two of three features must be present: oligo- or anovulation, clinical or biochemical hyperandrogenism, and polycystic ovary morphology on ultrasound. [2] PCOS is not solely a reproductive disorder. Insulin resistance (IR) represents an intrinsic feature of the syndrome, independent of obesity, as shown by euglycaemic-hyperinsulinaemic clamp studies - with IR present in 75% of lean and 95% of overweight women with PCOS. [3]

The metabolic consequences of PCOS are substantial and lifelong. Women with PCOS have impaired glucose tolerance and face a significantly increased risk of type 2 diabetes compared with age-matched controls. [4] Cardiovascular risk is also heightened, driven by clustering of dyslipidaemia, hypertension, and central adiposity. [5] Given this cardiometabolic burden, international guidelines recommend lifestyle intervention as the cornerstone of first-line PCOS care. [6]

1.2. Pathophysiological Link Between Insulin Resistance and Hyperandrogenism

Insulin resistance in PCOS arises from post-receptor signalling defects. Specifically, increased serine phosphorylation of insulin receptor substrate-1 (IRS-1) selectively impairs metabolic but not mitogenic insulin pathways in skeletal muscle and adipose tissue. [7] The ovarian response to insulin is paradoxically preserved. Elevated circulating insulin therefore drives theca cell androgen synthesis via stimulation of cytochrome P450c17alpha enzyme activity. [8] Hyperinsulinaemia also suppresses hepatic synthesis of sex hormone-binding globulin (SHBG), increasing the biologically active free testosterone fraction. [8] The resulting hyperandrogenism worsens IR by reducing GLUT-4 expression and promoting visceral adiposity. A self-reinforcing bidirectional cycle is thereby established. [7, 8, 9]

1.3. Lifestyle Intervention as First-Line Treatment

The 2023 International Evidence-based Guideline for the Assessment and Management of PCOS recommends lifestyle modification as the first-line treatment for women with PCOS. [6] This includes dietary change, physical activity, and behavioural strategies. Both dietary and exercise interventions improve IR and hormonal parameters, but through partially distinct molecular pathways. Exercise primarily enhances skeletal muscle insulin sensitivity through AMPK-mediated GLUT-4 translocation. This mechanism operates largely independently of energy balance, meaning exercise improves IR even in the absence of weight loss. [10] Dietary approaches primarily reduce postprandial hyperinsulinaemia and exert anti-inflammatory effects through macronutrient quality. [11] Whether combining these two modalities produces greater improvements in IR and hormonal parameters than either applied alone remains an underexplored question in the literature. [11]

1.4. Aim of the Present Review

While dietary and exercise interventions have each been evaluated in women with PCOS, they engage insulin resistance through partially distinct molecular pathways and differ substantially in their hormonal effects, clinical applicability, and supporting evidence base. [10, 11] A focused comparative review is therefore timely and clinically relevant. This is particularly true given the increasing integration of exercise prescription and sports medicine principles into first-line PCOS management, and the ongoing uncertainty among clinicians and exercise professionals regarding whether combined approaches offer meaningful advantages over single-modality programmes. The present structured narrative review aims to: (1) synthesise the available evidence on dietary, exercise, and combined lifestyle interventions in women with PCOS; (2) compare their respective effects on insulin resistance markers and hormonal parameters; (3) characterise the biological mechanisms underlying differential responses to intervention modality; and (4) provide a foundation for evidence-based exercise prescription, sports medicine practice, and future research priorities.

Research Objective. To synthesise and compare the effects of combined lifestyle interventions (diet plus exercise) versus single-modality approaches (diet alone or exercise alone) on markers of insulin resistance and key hormonal parameters in women with polycystic ovary syndrome, with a view to informing evidence-based exercise prescription and sports medicine practice.

Research Problems. Do combined lifestyle interventions produce superior improvements in insulin resistance and hormonal parameters compared with single-modality approaches in women with PCOS? Which specific combinations of exercise modality, intensity, and dietary pattern yield the most clinically meaningful outcomes for sports medicine practitioners and exercise professionals working with this population?

Research Hypotheses. Combined lifestyle interventions integrating structured exercise prescription with dietary modification are hypothesised to produce broader improvements in HOMA-IR, fasting insulin, testosterone, and SHBG in women with PCOS than either single-modality approach applied alone. This hypothesis is based on the complementary and partially distinct biological mechanisms of each modality, and remains to be confirmed by adequately powered comparative trials.

2. Material and methods

This narrative review was conducted in accordance with the principles of the Scale for the Assessment of Narrative Review Articles (SANRA). A structured literature search was carried out across PubMed/MEDLINE, Embase, and Cochrane Library databases. The following terms were used individually and in combination: polycystic ovary syndrome, PCOS, insulin resistance, HOMA-IR, lifestyle intervention, combined intervention, diet, exercise, physical activity, aerobic training, resistance training, HIIT, testosterone, and SHBG. Studies considered for inclusion were randomised controlled trials, systematic reviews, meta-analyses, and narrative reviews that evaluated lifestyle interventions in women with PCOS and reported outcomes related to insulin resistance and/or hormonal parameters. No restriction on publication date was applied. The included references range from 1992 to 2026. Additional references were identified through manual screening of reference lists from key included publications.

All data presented in this review were drawn exclusively from the selected peer-reviewed publications. Extracted information was organised thematically by intervention type and synthesised qualitatively to identify consistent findings, points of convergence, and sources of variability between studies. Given the narrative design of this review, no standardised extraction protocol or formal quality appraisal tool was applied. No original statistical analyses were conducted. Quantitative findings including effect sizes, confidence intervals, and p-values are cited directly from the original publications where reported, with particular reference to the randomised controlled trials, systematic reviews, and meta-analyses included in this synthesis.

3. Results

3.1. Pathophysiology of Insulin Resistance in PCOS: Rationale for Multi-Target Interventions

As outlined in section 1.2, IR in PCOS is driven by post-receptor signalling defects that selectively impair metabolic insulin pathways in skeletal muscle. The resulting compensatory hyperinsulinaemia drives ovarian androgen overproduction and suppresses hepatic SHBG synthesis. [7, 8] Chronic low-grade inflammation further exacerbates IR through serine kinase activation. [7] Because exercise and dietary modification each target IR through partially distinct molecular pathways - AMPK-mediated skeletal muscle sensitisation versus reduction of postprandial hyperinsulinaemia respectively - their combination may produce additive metabolic benefits that neither modality achieves alone. [10, 11]

3.2. Effects of Dietary Interventions Alone

3.2.1. Low-Glycaemic-Index and Mediterranean Diets

Low-glycaemic-index (low-GI) diets attenuate postprandial glucose and insulin excursions. [12, 13] Reductions in fasting insulin, HOMA-IR, and free testosterone have been reported following low-GI dietary interventions, alongside improvements in menstrual regularity. [12, 13] The Mediterranean diet exerts additional anti-inflammatory effects through its polyphenol, omega-3 fatty acid, and fibre content, complementing direct metabolic benefits in women with PCOS. [12]

The hormonal effects of low-GI and Mediterranean dietary interventions in PCOS are mediated primarily through their impact on fasting and postprandial insulin. [12, 8] Reductions in chronic hyperinsulinaemia attenuate the insulinaemic drive to ovarian androgen synthesis. This restores hepatic SHBG production, producing downstream reductions in free testosterone and improvements in menstrual regularity. [8, 12] In randomised trials comparing a low-GI diet with a conventional healthy diet in women with PCOS, the low-GI group showed greater improvements in insulin sensitivity, menstrual cyclicity, and emotional wellbeing, despite similar degrees of weight loss in both groups. [13] These findings indicate that the hormonal benefits of the low-GI dietary pattern are at least partly independent of weight loss. They appear to be mediated through direct effects on postprandial insulin excursions and their downstream hormonal consequences. [12, 13]

3.2.2. Caloric Restriction and Weight Loss

In contrast to the direct hormonal mechanisms of dietary patterns described above, caloric restriction acts primarily through weight loss-mediated metabolic improvements. Caloric restriction reduces IR in overweight and obese women with PCOS through weight loss-mediated improvements in insulin sensitivity, reduction of adipose-derived inflammatory cytokines, and partial restoration of SHBG levels. [14, 15] A weight reduction of 5-10% of initial body weight produces clinically meaningful improvements in menstrual regularity, ovulation, and hormonal profiles. [14, 15] Dietary interventions alone do not engage the exercise-mediated pathways of skeletal muscle insulin sensitisation. These pathways - principally AMPK activation and GLUT-4 upregulation - operate largely independently of energy balance, meaning that the full insulin-sensitising benefits achieved through exercise cannot be replicated by dietary intervention alone. [10]

3.3. Effects of Exercise Interventions Alone

3.3.1. Aerobic Exercise and High-Intensity Interval Training

Physical exercise improves insulin sensitivity primarily through AMPK-mediated GLUT-4 translocation in skeletal muscle and increased mitochondrial oxidative capacity. [10] These effects are detectable even without weight loss. Exercise is therefore beneficial for both overweight and normal-weight women with PCOS. [10, 16] Vigorous-intensity aerobic training performed for a minimum of 120 minutes per week is linked to improvements in VO2peak, HOMA-IR, fasting insulin, BMI, and waist circumference in women with PCOS. [16] High-intensity interval training (HIIT) has emerged as an effective alternative to traditional moderate-intensity continuous training (MICT) - a protocol involving sustained moderate-intensity aerobic exercise at a constant intensity. HIIT improves insulin sensitivity, reduces BMI, and favourably modifies lipid profiles. [17, 18] No statistically significant superiority of HIIT over MICT has been found across anthropometric, metabolic, or hormonal outcomes. This suggests comparable effectiveness when both modalities are matched for total exercise energy expenditure. [19]

The effects of aerobic exercise on hormonal parameters in PCOS extend beyond insulin sensitivity. [20, 21] Vigorous aerobic exercise is linked to improvements in insulin measures in women with PCOS, and resistance or strength training has been reported to improve androgen levels in some studies, though findings are inconsistent and additional evidence is needed. [20] A systematic review and meta-analysis of exercise interventions in PCOS found no statistically significant overall effect of exercise on total testosterone or DHEA-S. The effect on SHBG was also noted to be less direct than previously assumed, potentially requiring more prolonged intervention strategies. [21] Where hormonal improvements have been observed, they are at least partly attributed to the exercise-induced reduction in fasting insulin and HOMA-IR, which reduces the insulinaemic drive to ovarian androgen production and may restore hepatic SHBG synthesis. [8]

3.3.2. Resistance Training

Resistance training increases skeletal muscle mass - the primary site of insulin-stimulated glucose disposal. It also improves whole-body insulin sensitivity independently of aerobic fitness gains. [16, 22] Studies incorporating resistance training have shown reductions in HOMA-IR and free androgen index (FAI) in women with PCOS. [16, 23, 22] A 2025 randomised controlled trial evaluated 8 weeks of combined resistance and endurance training in women with PCOS. Significant modulation of hormonal, metabolic, inflammatory, and oxidative stress biomarkers was observed, with greater improvements reported compared with endurance training alone. [24] Improvements in IR may be constrained without concurrent dietary modification. Exercise alone does not address dietary contributors to postprandial hyperinsulinaemia. [11, 25]

Taken together, available evidence indicates that physical activity improves insulin sensitivity in women with PCOS through AMPK-mediated GLUT-4 translocation in skeletal muscle, with these benefits detectable even in the absence of weight loss. [10, 16] Aerobic exercise, HIIT, and resistance training each contribute independent benefits, with comparable effectiveness reported when matched for total energy expenditure. [16, 19] However, the direct effects of exercise alone on testosterone and SHBG remain inconsistent across studies, suggesting that concurrent dietary modification may be needed to achieve more comprehensive hormonal improvements. [21, 11]

3.4. Effects of Combined Lifestyle Interventions

3.4.1. Evidence from Systematic Reviews and Meta-Analyses

Statistically beneficial effects of exercise on metabolic, anthropometric, and cardiorespiratory outcomes in women with PCOS have been reported. However, based on limited available data, no statistically significant differences were found between combined exercise-and-diet versus diet alone for most outcomes. This highlights the methodological limitations of existing trials and the need for further well-designed comparative studies. [23] A drop in HOMA-IR of -36.2% following vigorous-intensity exercise in women with PCOS has also been reported, with the magnitude of effect modified by baseline HOMA-IR values and dietary co-intervention. [16] The largest improvements have been observed in supervised aerobic interventions in overweight and obese participants. [23]

A Cochrane review of lifestyle changes in women with PCOS evaluated dietary, exercise, and combined interventions and found that lifestyle intervention produced improvements in body weight, hormonal profiles, and menstrual regularity compared with no active treatment. [25] The review highlighted that the combination of diet and exercise produced improvements across a broader range of outcomes than either modality alone. However, the evidence was of low to moderate quality and conclusions should be interpreted with caution pending higher-quality trials. [25] A further systematic review and meta-analysis examining exercise effects

on androgen levels and SHBG in women with PCOS found no statistically significant overall effect on total testosterone or DHEA-S. The effect on SHBG also remained unclear, underscoring the need for adequately powered trials designed specifically to evaluate hormonal endpoints. [21]

SHBG production in the liver is suppressed by hyperinsulinaemia, and combined interventions may reduce IR via exercise while simultaneously reducing postprandial insulin via dietary modification. [8] This dual mechanism may produce greater increases in SHBG than either approach alone, thereby reducing free testosterone more effectively. [8] Increased SHBG reduces the biologically active androgen fraction. Clinical improvements in hirsutism, acne, and menstrual regularity have been reported in studies of effective PCOS treatment. [8, 6]

3.4.2. International Guideline Endorsement

The 2023 International Evidence-based Guideline for the Assessment and Management of PCOS endorses combined lifestyle intervention as first-line therapy. [6] It recommends 150-300 minutes of moderate-intensity or 75-150 minutes of vigorous-intensity aerobic activity per week. Muscle-strengthening activities on two non-consecutive days are also recommended, alongside evidence-based dietary approaches. The guideline recommends combining dietary change with exercise, noting this is likely to yield greater clinical improvements than either approach alone. [6]

Taken together, available evidence suggests that combined dietary and exercise interventions may produce broader improvements in insulin resistance and hormonal parameters than either modality applied alone, principally through complementary effects on postprandial and basal hyperinsulinaemia and downstream restoration of SHBG production. [16, 25, 8] However, current meta-analytic evidence has not consistently shown statistically significant superiority of combined over single-modality approaches for most outcomes, and the scientific community emphasises the need for adequately powered comparative trials to confirm these effects on hormonal endpoints. [23, 16]

3.5. Differential Hormonal Responses to Intervention Modality

Hormonal assessment in PCOS typically covers several markers, each reflecting a different aspect of the underlying pathophysiology. The Homeostatic Model Assessment of Insulin Resistance (HOMA-IR) is a calculated index derived from fasting glucose and insulin that estimates whole-body insulin resistance. [7] Total testosterone and the free androgen index (FAI) - calculated as the ratio of total testosterone to sex hormone-binding globulin (SHBG) - quantify androgen excess, the clinical hallmark of PCOS. [8] SHBG itself is a hepatic protein that binds circulating sex hormones; its synthesis is suppressed by hyperinsulinaemia, and reduced SHBG levels increase the bioavailable fraction of testosterone. [8] The luteinising hormone to follicle-stimulating hormone (LH/FSH) ratio reflects pituitary gonadotropin imbalance and is elevated in many women with PCOS. [7] Anti-Müllerian hormone (AMH) is produced by ovarian follicles and is often elevated in PCOS, reflecting increased follicle number. [6] Each of these markers may respond differently to lifestyle intervention modalities, as outlined below.

Testosterone and free androgen index reportedly respond to interventions that substantially reduce IR, whether through exercise, dietary restriction, or their combination. Exercise alone may not produce significant hormonal changes in all studies. [16, 23, 21] SHBG responds particularly to combined interventions targeting both postprandial hyperinsulinaemia via diet and basal skeletal muscle insulin sensitivity via exercise. [8] The LH/FSH ratio is elevated in many women with PCOS due to abnormal GnRH pulsatility driven by insulin and androgen excess. It shows partial normalisation with lifestyle interventions that reduce IR, though the response is more variable than for testosterone and SHBG. [7] Studies report inconsistent AMH responses to lifestyle interventions. Reductions have been observed primarily in overweight women following combined interventions with a meaningful weight-loss component. [16, 23]

The response of individual hormonal parameters to lifestyle intervention may guide the selection of intervention modality in clinical practice. [6] Free androgen index and SHBG have shown more consistent responses to combined interventions targeting IR through both dietary and exercise mechanisms than to exercise alone. [16, 23] LH normalisation, where it occurs, is associated with improvements in menstrual regularity and ovulation rates - outcomes of particular clinical relevance for women with PCOS seeking to improve fertility. [6] Monitoring HOMA-IR, SHBG, free testosterone, and LH/FSH ratio may provide a more complete assessment of response to lifestyle intervention in clinical practice. [8, 6]

3.6. Practical Considerations: Adherence, Safety, and Individualisation

Available research identifies three commonly reported barriers to lifestyle modification in women with PCOS: low health literacy, inadequate personalised guidance, and limited psychological support. [26] Women with PCOS also face elevated rates of depression and anxiety, which reportedly reduce exercise adherence in this population. [6] High-volume training combined with severe caloric restriction carries the risk of relative energy deficiency in sport (RED-S). This can paradoxically worsen hormonal dysfunction by suppressing the hypothalamic-pituitary-ovarian axis. [27] Delivery within multidisciplinary teams incorporating exercise professionals, dietitians, and behavioural psychologists is recommended. [28]

Based on the reviewed evidence, the combination of vigorous-intensity aerobic exercise performed at a minimum of 120 minutes per week - supplemented by resistance training on at least two days per week - alongside a low-GI or Mediterranean dietary approach appears to produce the most consistent improvements in HOMA-IR and hormonal parameters in women with PCOS. [16, 6, 12, 13] Resistance training should be incorporated as a complement to aerobic exercise given its independent benefits on skeletal muscle insulin sensitivity and androgen levels. [22, 24] For sports medicine practitioners and exercise professionals, this means developing exercise prescriptions that specify both modality and intensity, while coordinating with a registered dietitian to ensure dietary strategies are compatible with training demands and do not create an energy deficit sufficient to suppress hypothalamic-pituitary-ovarian function. [28, 27]

Individualisation of combined lifestyle programmes is essential given the clinical and phenotypic heterogeneity of PCOS. The 2023 guideline recommends that lifestyle programmes be tailored to each woman's phenotype, body composition, exercise capacity, and dietary preferences. [6] Lean women with PCOS represent a distinct phenotype with IR that is not mediated by excess adiposity. They require exercise prescriptions tailored to their specific metabolic profile, as weight loss-focused interventions are inappropriate and may carry the risk of relative energy deficiency. [3, 27] The exercise and dietary components of combined interventions should be designed collaboratively by exercise professionals and registered dietitians with specific expertise in PCOS. Particular attention should be paid to ensuring that caloric targets and training volumes are compatible and do not create an energy deficit sufficient to suppress hypothalamic-pituitary-ovarian function. [6, 27, 28]

Taken together, lifestyle management of PCOS may benefit from a personalised, multidisciplinary approach that combines dietary and exercise interventions to improve insulin sensitivity, while carefully avoiding the excessive energy deficit associated with RED-S that could exacerbate hormonal disturbances in the hypothalamic-pituitary-ovarian axis. [6, 28, 27]

3.7. Comparison of Dietary Patterns: Differential Effects on Insulin Resistance and Hormonal Parameters

3.7.1. Low-Glycaemic-Index Versus Mediterranean Diet

Both the low-GI and Mediterranean diets improve IR markers and hormonal profiles in women with PCOS, as described in section 3.2.1. When compared directly, limited evidence exists to recommend one approach over the other, as modifications in protein, carbohydrate, and fat quality generally produced similar effects on PCOS presentations. [29] The key distinction between the two patterns lies in their primary mechanism: the low-GI diet acts principally through reduction of postprandial insulin excursions, while the Mediterranean diet exerts broader anti-inflammatory effects through its polyphenol and omega-3 content that complement its insulin-lowering benefits. [12]

The degree of adherence to the Mediterranean diet has been negatively correlated with the clinical severity of PCOS. Lower intake of fibre, fruit, vegetables, and monounsaturated fat was associated with more severe IR and hyperandrogenism. [12] Dietary quality - rather than any single macronutrient restriction - may therefore be the primary determinant of metabolic benefit in women with PCOS, given that adherence to overall dietary patterns has been more consistently associated with clinical outcomes than individual nutrient modifications. [12, 29]

Taken together, both dietary patterns appear to improve metabolic and hormonal parameters in PCOS, with the low-GI diet focusing primarily on stabilising postprandial insulin excursions, while the Mediterranean diet offers additional anti-inflammatory benefits. This suggests that the overall quality of the dietary pattern - rather than the restriction of specific macronutrients - is the key determinant of improvement in clinical and metabolic outcomes in women with PCOS. [12, 13, 29]

3.7.2. Ketogenic and Very-Low-Calorie Diets

Ketogenic diets are characterised by very low carbohydrate intake - typically below 50 g per day - and high fat intake. They have attracted growing interest in PCOS management. Their capacity to reduce fasting insulin and improve IR operates through mechanisms distinct from caloric restriction alone - principally ketone-mediated suppression of hepatic glucose production and reduction of postprandial insulin secretion. [30] Preliminary data suggest improvements in body weight, fasting insulin, triglycerides, and HOMA-IR following very-low-calorie ketogenic dietary protocols in women with PCOS. [30] However, the evidence base remains limited by small sample sizes and short intervention durations. Concerns regarding long-term sustainability, bone mineral density, and cortisol regulation restrict their broad clinical recommendation. [30] In the context of combined lifestyle interventions, ketogenic diets may be difficult to combine with high-intensity exercise protocols due to reduced glycogen availability, which is linked to impaired high-intensity exercise capacity. [10]

Taken together, the ketogenic diet appears to reduce insulin levels and improve metabolic parameters in PCOS through specific ketone-mediated mechanisms; however, its clinical application is limited by concerns about long-term health effects and the difficulty of combining this approach with high-intensity training due to reduced glycogen availability. [30, 10]

3.8. Synthesis: Combined Versus Single-Modality Interventions on Insulin Resistance and Hormonal Parameters

Synthesising the evidence reviewed across sections 3.2 to 3.7, a consistent pattern emerges regarding the comparative effects of combined versus single-modality lifestyle interventions on insulin resistance and hormonal parameters in women with PCOS. [16, 23, 25] Both dietary and exercise interventions independently improve HOMA-IR, fasting insulin, testosterone, and SHBG compared with no active treatment. [16, 23, 25] However, the available evidence - while limited by methodological heterogeneity and small sample sizes - suggests that combined interventions engaging both dietary and exercise pathways simultaneously may produce broader hormonal improvements than either modality alone. [16, 23]

For insulin resistance specifically, vigorous aerobic exercise combined with dietary co-intervention is linked to the largest reported reductions in HOMA-IR in meta-analytic analyses, with a pooled reduction of -36.2% reported in one review. [16] The magnitude of this effect was further modified by baseline HOMA-IR - women with the highest baseline IR showing larger absolute improvements - and by the presence of a dietary co-intervention, suggesting that the combination of exercise and dietary modification may produce greater IR reduction than exercise alone. [16] For SHBG, the combined mechanism addresses both the acute and chronic components of the hyperinsulinaemia that suppresses hepatic SHBG production. Dietary modification reduces postprandial insulin, while exercise reduces fasting insulin through skeletal muscle sensitisation. Together, these effects may produce greater SHBG increases than either modality alone. [8, 16]

For testosterone and free androgen index, available evidence suggests that reductions may be more consistently achieved through interventions that produce the greatest overall reduction in IR, whether through combined or single-modality approaches, as exercise alone has not been shown to produce significant reductions in testosterone in all meta-analytic analyses. [16, 21] For LH/FSH ratio and AMH specifically, the evidence is even more limited than for HOMA-IR and testosterone, with very few studies reporting these outcomes in directly comparative designs. Adequately powered comparative trials are needed across the full hormonal panel. [16, 23] Overall, these findings suggest the potential value of targeting IR through multiple complementary pathways - dietary and exercise - to achieve broader hormonal normalisation in women with PCOS.

Table 1. Comparative summary of the effects of single-modality versus combined lifestyle interventions on insulin resistance and hormonal parameters in women with PCOS.

Intervention	Primary Mechanism	HOMA-IR	Testosterone / FAI	SHBG	LH/FSH Ratio
Low-GI diet	Reduces postprandial insulin	↓	↓ / +/–	↑ / +/–	+/–
Mediterranean diet	Anti-inflammatory, reduces insulin	↓	↓ / +/–	↑ / +/–	+/–
Ketogenic diet	Suppresses hepatic glucose production	↓↓	↓ / +/–	↑ / +/–	+/–
Aerobic exercise	AMPK/GLUT-4 translocation	↓	+/–	+/–	+/–
HIIT	AMPK/GLUT-4 translocation	↓	+/–	+/–	+/–
Resistance training	Increases muscle mass, ↑ insulin sensitivity	↓	↓ / +/–	+/–	+/–
Combined diet + exercise	Dual pathway: dietary + skeletal muscle	↓↓	↓ / +/–	↑ / +/–	+/–

Source: Own elaboration based on included studies. Notes: ↓ = reported decrease; ↑ = reported increase; ↓↓ = stronger or more consistent reported decrease; +/– = inconsistent or insufficient evidence. The symbols summarise the direction of effects reported in the reviewed literature and should not be interpreted as pooled effect sizes. FAI = free androgen index; HOMA-IR = homeostatic model assessment of insulin resistance; SHBG = sex hormone-binding globulin; LH/FSH = luteinising hormone/follicle-stimulating hormone ratio; HIIT = high-intensity interval training.

4. Conclusions

Both dietary and exercise interventions have been associated with clinically meaningful improvements in insulin resistance and hormonal parameters in women with PCOS through partially distinct molecular pathways. Aerobic exercise and resistance training - the two primary exercise modalities available to sports medicine practitioners - improve IR through AMPK-mediated GLUT-4 translocation and skeletal muscle insulin sensitisation. [10] Low-GI and Mediterranean dietary approaches reduce postprandial hyperinsulinaemia and exert complementary anti-inflammatory effects. [12, 13] Vigorous-intensity exercise combined with dietary intervention is linked to the largest reductions in HOMA-IR in available meta-analytic evidence, with the magnitude of effect modified by baseline HOMA-IR values and dietary co-intervention. [16] These findings suggest that exercise prescriptions for women with PCOS may benefit from being developed within a broader lifestyle framework that integrates dietary strategies, rather than in isolation.

However, the evidence base for any advantage of combined over single-modality approaches remains limited by the quality and number of available trials. One major review found no statistically significant differences between combined exercise-and-diet versus diet alone for most outcomes, highlighting the methodological limitations of existing research. [23] Most available evidence is indirect, derived from multi-arm trials comparing different intervention types without ensuring equivalence of dose. [16, 23] As a narrative rather than systematic review, this study is also subject to selection bias and the broad scope of included literature precludes quantitative synthesis. [16, 23]

Sports medicine practitioners and exercise professionals working with women with PCOS are encouraged to consider combined lifestyle programmes as a recommended first-line approach, consistent with international guidelines. These programmes should integrate structured exercise prescription with evidence-based dietary strategies, with the recognition that the comparative evidence base is still developing. [6, 28] This review provides a synthesis of the available evidence on combined versus single-modality lifestyle interventions in PCOS and a framework for the well-designed comparative clinical trials that are now needed.

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