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# Metabolic consequences of obesity and insulin resistance in polycystic ovary syndrome: diagnostic and methodological challenges

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## Abstract

Women with polycystic ovary syndrome (PCOS) have a considerable risk of metabolic dysfunction. This review aims to present contemporary knowledge on obesity, insulin resistance and PCOS with emphasis on the diagnostic and methodological challenges encountered in research and clinical practice. Variable diagnostic criteria for PCOS and associated phenotypes are frequently published. Targeted searches were conducted to identify all available data concerning the association of obesity and insulin resistance with PCOS up to September 2016. Articles were considered if they were peer reviewed, in English and included women with PCOS. Obesity is more prevalent in women with PCOS, but studies rarely reported accurate assessments of adiposity, nor split the study population by PCOS phenotypes. Many women with PCOS have insulin resistance, though there is considerable variation reported in part due to not distinguishing subgroups known to have an impact on insulin resistance as well as limited methodology to measure insulin resistance. Inflammatory markers are positively correlated with androgen levels, but detailed interactions need to be identified. Weight management is the primary therapy; specific advice to reduce the glycaemic load of the diet and reduce the intake of pro-inflammatory SFA and advanced glycation endproducts have provided promising results. It is important that women with PCOS are educated about their increased risk of metabolic complications in order to make timely and appropriate lifestyle modifications. Furthermore, well-designed robust studies are needed to evaluate the mechanisms behind the improvements observed with dietary interventions.

**Key words:** **Polycystic ovary syndrome: Obesity: Diabetes: Insulin resistance**

## Introduction

Polycystic ovary syndrome (PCOS) is a common endocrine–metabolic disorder affecting 12–18 % of women, depending on the diagnostic criteria used[1]. Women with PCOS are at lifelong risk of metabolic dysfunction and have increased risk of insulin resistance (IR), type 2 diabetes mellitus (T2DM), dyslipidaemia and atherosclerosis[2]. The pathophysiology of PCOS is complex and to some extent remains unclear; PCOS has a genetic heritability and several potential genes have been identified. However, research in this area is still in its infancy[3,4] and is influenced by environmental factors, most notably diet and increased body weight[5]. The progression of research and translation into clinical practice are hindered by the variability of diagnostic and methodological issues endemic in studies of PCOS[6].

Therefore the aim of this review is to present current knowledge on the association between obesity, IR and PCOS and in doing so highlight some of the diagnostic and methodological challenges encountered in research and clinical practice. Targeted searches were conducted to identify all available data concerning the association of obesity and IR with PCOS in order to summarise current knowledge on this topic up to September 2016. PubMed and Science Direct were utilised in order to locate relevant scientific reports and papers and ensure an extensive review of the literature. Articles were considered if they were peer reviewed, in English and included women with PCOS (all diagnostic methods).

PCOS is a heterogeneous condition, the symptoms of which often first present during the teenage years[7], though the symptoms vary in their severity and can also change with age. The symptoms include: menstrual irregularity, acne, excess male pattern hair (hirsutism), male pattern alopecia, central adiposity and fertility issues. Many of the symptoms associated with PCOS have been shown to lead to a reduction in health-related quality of life[8] and consequently depression and anxiety are commonly reported in women with PCOS, independent of body-weight status[9]. Although obesity is a common feature of PCOS, lean women with PCOS are also at increased disease risk when compared with matched controls and can also suffer debilitating symptoms[3]. IR is not within any of the diagnostic criteria; however, the majority of lean and overweight women with PCOS have a form of IR intrinsic to PCOS and the compensatory hyperinsulinaemia drives many of the phenotypic features of PCOS[10]. Hyperinsulinaemia promotes ovarian hyperandrogenism which is present in 60–80 % of women with

**Abbreviations:** AGEs, advanced glycation endproducts; GI, glycaemic index; GL, glycaemic load; IGT, impaired glucose tolerance; IR, insulin resistance; NAFLD, non-alcoholic fatty liver disease; PCOS, polycystic ovary syndrome; T2DM, type 2 diabetes mellitus; VAI, visceral adiposity index.

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**Table 1.** Diagnostic criteria for polycystic ovary syndrome (PCOS)[18]

| Criteria | NIH 1990 | Rotterdam 2003* | AE-PCOS |
|---|---|---|---|
| Oligo-ovulation leading to oligomenorrhoea, or anovulation leading to amenorrhoea | + | +/− | +/− |
| Hyperandrogenism: clinical (hirsutism, male pattern alopecia, acne) and/or biochemical | + | +/− | + |
| Polycystic ovarian morphology on ultrasound | +/− | +/− | +/− |

NIH, National Institutes of Health; AE, androgen excess.
* The presence of two of the following three features with the exclusion of other endocrine disorders.

PCOS; the androgen excess predominately results from increased synthesis and release of ovarian androgens[10]. In combination, hyperinsulinaemia and hyperandrogenaemia can disrupt follicle growth; this is accompanied by menstrual irregularity, anovulatory sub-fertility and accumulation of immature follicles[5].

The most widely used criteria for clinical diagnosis of PCOS involve the presence of two of the three features listed in Table 1, with the exclusion of other endocrine disorders as developed by the European Society of Human Reproduction and Embryology (ESHRE) and the American Society for Reproductive Medicine (ASRM)[11], and are otherwise known as the Rotterdam criteria.

There are distinct phenotypic presentations associated with PCOS, and over the years several sub-categories have been suggested. In 2012 at the National Institutes of Health workshop there was a general consensus[12] and these categories are as follows:

Androgen excess + ovulatory dysfunction;
Androgen excess + polycystic ovarian morphology;
Ovulatory dysfunction + polycystic ovarian morphology;
Androgen excess + ovulatory dysfunction + polycystic ovarian morphology.

Several studies have highlighted that women with PCOS who have regular ovulatory cycles often have normal insulin sensitivity with milder metabolic abnormalities compared with women with PCOS with ovulatory dysfunction[13,14]. Women who are anovulatory with hyperandrogenism are considered to be at the greatest metabolic risk[15]. Women with hyperandrogenic PCOS generally present with a worse cardiometabolic profile compared with women with non-hyperandrogenic PCOS[16–18]. It is generally agreed that the heterogeneity in the PCOS population, studies not reporting specific PCOS phenotypes, and the different methodologies used to report IR and obesity/visceral fat have led to controversy in the relationship between obesity, IR and PCOS[6,19].

## Obesity and polycystic ovary syndrome

Obesity is a common feature of PCOS. A meta-analysis of cross-sectional and retrospective studies illustrated there is a greater prevalence of obesity in PCOS; BMI $>30\,kg/m^2$ (relative risk 2·77; 95 % CI 1·88, 4·10; PCOS *n* 4165; controls *n* 4885) compared with healthy women[20]. The variability of data reporting the prevalence of obesity in women with PCOS is likely to reflect geographical, environmental and population variability between studies. There is also uncertainty as to whether PCOS causes weight gain and obesity or if obesity is linked to the development of PCOS[21,22]. Furthermore, clarification of the role of adiposity in PCOS entails measurements other than the measurement of BMI.

A meta-analysis clearly indicated that overweight or obese women with PCOS had significantly worse metabolic and reproductive outcomes when compared with healthy-weight women with PCOS[23]. Palomba *et al.*[24] confirmed that obesity has a bidirectional relationship with PCOS and noted that excessive weight gain could unmask a latent PCOS condition. IR promotes the development of visceral adiposity in women with PCOS, whereas the role of androgens is less clear[25]. Tosi *et al.*[26] have recently shown adiposity to be correlated with IR and free testosterone levels (fat mass determined by dual-energy X-ray absorptiometry (DXA), IR with euglycaemic clamp and serum free testosterone by liquid chromatography–MS; *n* 100 women with PCOS). However, insulin sensitivity, but not adiposity, was found to be an independent predictor of free testosterone concentrations. The central:peripheral fat ratio identified by DXA was shown to be related to androgen levels in twenty-four women with PCOS[27]. Details of the complex relationship between androgens and obesity are gradually unfolding[28]; adipocytes seem to be prone to hypertrophy when exposed to androgen excess, and both adipose tissue hypertrophy and hyperandrogenism are related to IR[29]. What is known is that weight reduction, even as little as 5 %, improves PCOS symptoms in overweight or obese women with PCOS[30,31].

Whether women with PCOS present with increased abdominal adiposity compared with women in the general population continues to be debated. A diagnostic method of abdominal obesity has not yet been defined and thus a range of methods is used. Visceral adiposity index (VAI) is a simple surrogate marker of visceral adiposity which utilises BMI, waist circumference, TAG and HDL measurements and has been increasingly used since it was established in 2010[32]. Anthropometric studies that include waist circumference measurements have shown that, regardless of BMI, women with PCOS typically have an increased abdominal fat distribution[33–36]. A meta-analysis by Lim *et al.*[20] also reported that women with PCOS had increased prevalence of central obesity (risk ratio 1·73; 95 % CI 1·31, 2·30; PCOS *n* 1191; controls *n* 2396) (Fig. 1).

When body composition has been assessed by MRI, amounts of visceral adipose tissue have been found to be similar in women with PCOS and controls with comparable BMI (*n* 31), despite increased waist:hip ratios[37] in the women with PCOS. However, it is worth noting that in this study a number of participants were only 1 week after ceasing the insulin-sensitising agent, metformin, which has been shown to reduce visceral adiposity in women with PCOS[38,39]. Until relatively recently, studies infrequently reported PCOS phenotypes, which are known to have an impact on metabolic risk. Panidis *et al.*[40], in a relatively small study (*n* 100 PCOS), reported significantly higher VAI in the hyperandrogenism and oligo/anovulation phenotype, when compared with other phenotypes. Additionally, Androulakis *et al.*[41] reported that women with PCOS with menstrual disorders had significantly


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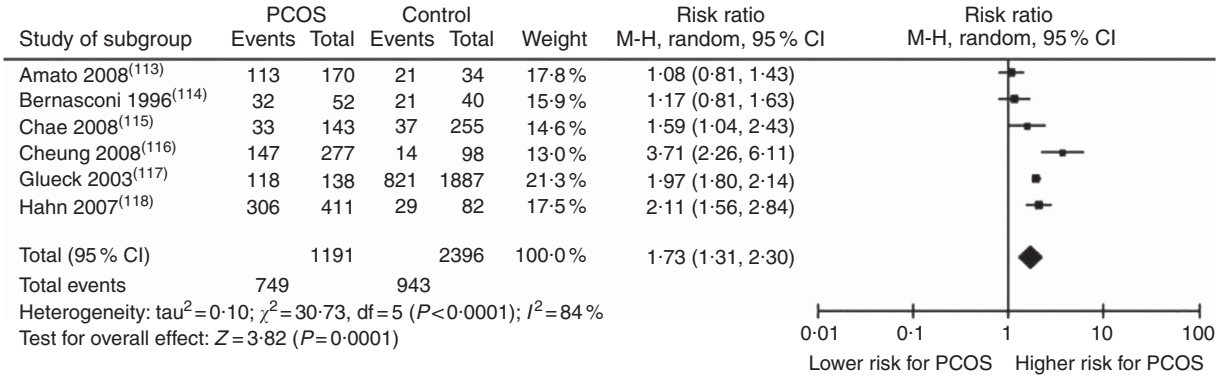


**Fig. 1.** Meta-analysis[113–118] of the prevalence of central obesity in women with and without polycystic ovary syndrome (PCOS) (taken from Lim *et al.*[20]). M-H, Mantel–Haenszel.

higher VAI when compared with PCOS without menstrual disorders. A more recent, albeit relatively small, study[15] reported a higher VAI in PCOS women with hyperandrogenism and oligo/annovulation compared with other phenotypes. Further research is clearly warranted with more accurate assessments of adiposity for different PCOS phenotypes to help clarify the link between obesity and PCOS.

Visceral adipose tissue is an important endocrine organ, producing and releasing several adipokines; any specific differences in resistin, leptin and visfatin in PCOS women compared with controls remain uncertain; however, a clear role for adiponectin in PCOS has been reported. Adiponectin is downregulated in obesity and is associated with IR; it also has strong insulin-sensitising, anti-inflammatory and anti-diabetic functions. Adiponectin circulates in different polymer complexes; it has been suggested that its actions are mediated primarily by the high-molecular-weight form of adiponectin[42]. In 2014[43], a meta-analysis based on thirty-eight trials (*n* 1944 PCOS; *n* 1654 healthy controls) highlighted significantly lower total adiponectin levels in PCOS women compared with healthy controls, independent of BMI (weighted mean difference −2·67; 95 % CI −3·22, −2·13). Visceral fat secretes less adiponectin compared with subcutaneous fat[44], so the proposed increased visceral adiposity in women with PCOS could be a reason behind decreased high-molecular-weight adiponectin levels. Metformin, a common treatment option for PCOS, was associated with significantly elevated serum adiponectin concentrations in women with PCOS (standard mean differences −0·43, 95 % CI −0·75, −0·11)[45]. In addition, it is likely that adiponectin levels are different in the PCOS phenotypes as suggested by Cankaya *et al.*[46]; in a small study (*n* 40), adolescents with androgen excess PCOS phenotype had significantly lower high-molecular-weight adiponectin levels compared with non-hyperandrogenic women. Testosterone has been found to contribute to high-molecular-weight adiponectin variance[47]; however, in a 2009 meta-analysis testosterone levels were not associated with adiponectin levels[48]. Further high-quality studies are needed to help clarify the role of testosterone and adiponectin in PCOS.

Obesity is evidently complex and the location of excess adiposity is key and all too rarely measured in combination with presenting PCOS phenotypes. In particular, visceral fat plays a key role in generating the insulin-resistant state which leads to impaired glucose tolerance (IGT) and is a significant and independent risk factor for T2DM[49].

## Insulin resistance in polycystic ovary syndrome

A link between PCOS and IR was first highlighted in 1980, whereby obese women with PCOS were shown to have an increased insulin response to an oral glucose tolerance test compared with obese controls[50]. Prevalence rates of IR have been reported between 44 and 85 %[10,51]. This variability is in part due to differences in PCOS phenotype and ethnicity[15,17]. Reports of the prevalence of IR are limited by the methods used to determine insulin sensitivity; the euglycaemic insulin clamp technique is the most accurate method to diagnose IR, but its high cost limits use; thus the surrogate markers, fasting insulin and homeostasis model assessment of insulin resistance (HOMA-IR)[52], are often reported.

IR and compensatory hyperinsulinaemia are fundamental to the exacerbation of PCOS symptoms and metabolic complications[53]. Several studies have shown that the majority of women with PCOS, both lean and overweight, have a form of IR that is intrinsic to the syndrome[10]. Researchers have shown that obesity and PCOS have a separate and synergistic relationship with IR[54]. Defects in both insulin sensitivity and secretion are present in lean women with PCOS[55]. The pathogenesis of IR in PCOS is complex and incompletely understood, though it is agreed there is a post-binding defect in insulin receptor signalling[51]. There is an increasing body of evidence to suggest a genetic susceptibility to PCOS and its associated IR as well as evidence to suggest tha PCOS may also have fetal origins due to androgen exposure at critical periods of growth or due to intra-uterine growth restrictions[4].

IR is a common feature in annovulatory women with PCOS; however, it is not as frequently observed in ovulatory women with PCOS, even when hyperandrogenism is present[56]. Bil *et al.*[15] reported significantly greater HOMA-IR in the androgen excess PCOS phenotype compared with non-hyperandrogenic women (*n* 100). Some older studies have failed to demonstrate IR in lean women with PCOS[57,58],


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although this may be accounted for by the inability to distinguish between the different PCOS phenotypes which resulted in the inclusion of women who were ovulatory.

Chronic inflammation and its role in IR in PCOS continues to be investigated[59]. The increase in multiple markers of inflammation such as leucocyte count, C-reactive protein, IL-18, protein-1, and monocyte chemo-attractant in addition to increased oxidative stress and endothelial dysfunction are evidence that PCOS is commonly coupled with low-level systemic inflammation[60]. However, there are limited studies that assess the presence of chronic inflammation independent of adiposity in women with PCOS. Circulating markers of oxidative stress are abnormal in women with PCOS independent of weight excess and IR[61]. The close association between oxidative stress and inflammation make it difficult to determine their individual contribution to PCOS and the mechanism of oxidative stress-induced IR remains unclear[62]. Oxidative stress and inflammation markers are positively correlated with androgen levels in PCOS patients[63]; thus they seem to contribute to hyperandrogenaemia, but detailed interactions still need to be identified, and as of yet few investigations have been done.

Insulin acts directly on the ovary by stimulating the thecal cells to increase androgen production and by activation of the cytochrome P450c17α[57,64]. Hyperinsulinaemia further exacerbates the pathogenesis of PCOS by inhibiting the production of insulin-like growth factor-1 (IGF-1) binding protein in the liver, leading to elevated circulating levels of IGF-1, which in turn stimulates ovarian thecal cell androgen production[65]. Hyperinsulinaemia reduces hepatic production of sex hormone-binding globulin, thus increasing free testosterone levels[66]. In combination, hyperinsulinaemia and hyperandrogenaemia can disrupt follicle growth; this is accompanied by menstrual irregularity, anovulatory sub-fertility and accumulation of immature follicles[51]. Diamanti-Kandarakis & Dunaif[51] in a review described the modulating action of insulin on ovarian steroidogenesis as well as the importance of the insulin signalling pathway in the control of ovulation.

Women with PCOS commonly have postprandial dysglycaemia[67,68], which reflects peripheral, primarily skeletal muscle, IR. In lean women with PCOS hyperinsulinaemia is often evident postprandially but not in the fasted state[69]. IR and compensatory hyperinsulinaemia have also been proposed as a cause of reactive hypoglycaemia in women with PCOS. A cross-sectional study of sixty-four lean young women with PCOS reported the rate of reactive hypoglycaema to be 50 %[70]. A survey of clinical practice (*n* 88) also indicated a high prevalence of hypoglycaemia; self-reported in 71 % of women with PCOS[36]. A recent study also indicated a higher prevalence of reactive hypoglycaemia in PCOS (*n* 88) compared with age- and BMI-matched controls (*n* 34), though a much lower prevalence of 17 %[71]. Robust studies investigating hypoglycaemia are sparse.

## Impaired glucose tolerance and type 2 diabetes mellitus

The prevalence of IGT and T2DM in women with PCOS is substantially higher compared with age- and weight-matched healthy women; a meta-analysis of cross-sectional and cohort studies reported an OR of 2·54 (95 % CI 1·44, 4·47) for OGT and 4·00 (95 % CI 1·97, 8·10) for T2DM[72]. Differences in the diagnostic criteria for IGT and PCOS were evident, and there were inadequate data to allow for PCOS phenotype sub-analysis; additionally, differences in ethnicity and BMI have resulted in variable risk estimates between PCOS studies. Studies in the USA have indicated a prevalence of 23–35 % for IGT and 4–10 % for T2DM[67,68], whereas in non-US studies prevalence rates in women with PCOS are elevated although not all to the same extent, with prevalence rates of 16 % for IGT and 2·5 % for T2DM described in an Italian cohort[73]. In the US studies, even lean women with PCOS had increased rates of IGT and T2DM compared with healthy controls[67], the onset of IGT occurring in their thirties and forties which is earlier than the normal population[74]. A more recent longitudinal study following women for at least 10 years reported that the age-standardised prevalence of diabetes at the end of follow-up was 39·3 %, significantly higher than in healthy women (prevalence of 5·8 %)[75].

The Royal College of Obstetrics and Gynaecology 2014 guidelines[76] state that women presenting with PCOS with a BMI $\geq$ 25 kg/m$^2$ and lean women with PCOS, but who have additional risk factors such as advanced age (> 40 years), personal history of gestational diabetes or family history of T2DM, should have a 2-h post-75 g oral glucose tolerance test.

## Metabolic syndrome and non-alcoholic liver disease in polycystic ovary syndrome

PCOS commonly includes many metabolic syndrome components, such as abdominal obesity, dyslipidaemia, hyperglycaemia and hypertension, which predispose women with PCOS to the development of T2DM and CVD. There is a higher prevalence of the metabolic syndrome in women with PCOS compared with women in the general population[77], estimated to be between 34 and 46 %, based on studies of Caucasian women with PCOS from the USA using the National Cholesterol Education Program Adult Treatment Panel III criteria[78–80]. Women diagnosed with PCOS using National Institutes of Health criteria are more likely to have a higher prevalence of the metabolic syndrome compared with controls, compared with women diagnosed with PCOS using the ESHRE/ASRM guidelines[72,81]. Studies that clearly indicate the prevalence of the metabolic syndrome in differing PCOS phenotypes are lacking.

IR and obesity are strong predictors of non-alcoholic fatty liver disease (NAFLD)[82,83]; thus it is not surprising that there is a higher prevalence of NAFLD in women with PCOS compared with age- and BMI-matched controls; prevalence rates ranging from 27 to 62 %[84–87]. This variation can be attributed to differences in laboratory diagnostic criteria (for example, liver function tests, liver imaging studies, liver biopsy) and differences in study populations relating to ethnicity, age, BMI and PCOS diagnostic criteria[88]. Vassilatou[88] has highlighted the increasing evidence that hyperandrogenism is related to NAFLD in PCOS and suggested that hyperandrogensim should be considered as an additional link in the synergy with obesity and IR for the development of NAFLD in PCOS. However,


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sub-analyses with the PCOS phenotypes were not reported and would seem prudent for further evaluation.

Of clinical importance, the prevalence of non-alcoholic steatohepatitis (NASH) in women with PCOS is particularly high[89]. Bariatric surgery has shown beneficial outcomes on NASH[90]. With the exception of weight loss through lifestyle modification and encouraging exercise in obese women, few evidence-based effective treatments specifically target NAFLD/NASH in women with PCOS[91].

## Dietary influences of the metabolic aspects of polycystic ovary syndrome

With no cure for PCOS, clinical management focuses on treating the presenting symptoms. Lifestyle management is advocated as the primary therapy in overweight and obese women with PCOS since even a modest weight loss of just 5–10 %, without medical intervention, improves many of the symptoms associated with PCOS[30,92,93].

The optimal method of achieving sustainable weight loss is under constant debate; studies demonstrating the clinical benefits of weight loss in women with PCOS incorporate a variety of methods to reduce energy intake and achieve a negative energy balance[94]. Behaviour-change strategies have long been recognised as being instrumental in managing chronic conditions, particularly weight management. However, lifestyle modification programmes are often reported to have low compliance and high dropout rates[95]. Furthermore, many overweight or obese women with PCOS are not following a diet that would promote weight loss[96]. Bariatric surgery, on the other hand, in women with PCOS has resulted in substantial weight loss and marked improvement of multiple biochemical abnormalities[97,98]. A recent meta-analysis of thirteen studies (2130 women) reported a reduction in the incidence of PCOS preoperatively from 45·6 %, to 6·8 % at 12-month follow-up[99].

Dietary modification to improve IR may produce benefits greater than those achieved by modest weight loss and would also be suitable for lean IR women with PCOS. Modifying the carbohydrate content and fatty acid content of the diet have both been proposed as methods to improve insulin sensitivity. The concept of a low-glycaemic index (GI) diet aims to reduce the glycaemic load (GL) and hence insulin response to ingested foods and drinks. Greater improvement in insulin sensitivity and regular menses were reported after weight loss with a low dietary GI approach ($n$ 29) compared with weight loss through conventional healthy eating ($n$ 20)[100]. In this study, women were followed for 12 months or until they achieved 7 % weight loss. Barr *et al.*[101] also demonstrated an improvement in insulin sensitivity after following an isoenergetic diet with a reduction in dietary GI in twenty-one women with PCOS over a 12-week intervention period. Furthermore, one study[102] reported a reduction in IR after a 12-week hypoenergetic diet, high in protein (30 % protein: $n$ 23), with a low GL compared with a conventional hypoenergetic healthy eating intervention (15 % protein: $n$ 26). A low-GL diet in lean women with PCOS ($n$ 41) resulted in a reduction in waist circumference[36]. These small studies suggest a benefit for promoting lower GI dietary advice to lean women with PCOS, many of whom are also insulin resistant. However, exploring the mechanisms behind the intervention and assessing IR with the euglycaemic clamp technique and evaluating the effect of modifying dietary GI in a subgroup of IR women with PCOS is prudent to improving the evidence base for dietary recommendations.

Inflammation is implicated in the pathogenesis of PCOS; dietary constituents such as SFA can influence inflammatory mediators, such as TNF-α and IL-1β and disrupt insulin signalling[103,104]. Conversely, MUFA and PUFA do not interrupt metabolism and inflammation to the same extent. SFA intake has been shown to influence glucose metabolism through the altering of insulin signalling and cell membrane function, with a diet high in SFA associated with a decrease in insulin sensitivity when compared with a high-MUFA diet[103]. Of interest, it has been reported that 68 % of women with PCOS had a total fat intake greater than a 35 % contribution to total energy, with SFA intake accounting for 12 % of total energy, substantially exceeding the reference nutrient intake (<10 % energy)[105,106]. Thus, a reduction of total fat intake should focus on lowering SFA intake concurrently with the encouragement of the maintenance of MUFA and PUFA in women with PCOS. There is some evidence to show that by reducing the consumption of saturated fat[107] and reducing the GI and GL of the diet[107], insulin sensitivity can be improved in insulin-resistant populations; however, there remains a paucity of studies in women with PCOS[108].

Advanced glycation end products (AGEs) are elevated in PCOS[109]; these pro-inflammatory molecules trigger a state of intracellular oxidative stress and inflammation after binding to their cell membrane receptors (RAGE). They are highly reactive molecules formed by non-enzymic reactions of carbonyl group of carbohydrates with free amino groups of proteins, nucleic acids or lipids. AGEs within the body are from endogenous production or ingested in high amounts from cooked fast food[110]. A small study[111] (twenty-three PCOS participants) has shown that a 2-month intervention of a low dietary intake of AGEs is associated with improved levels of serum testosterone, oxidative stress and HOMA-IR in PCOS. The role of dietary AGEs as mediators of metabolic and reproductive alterations in PCOS is an exciting and emerging area of dietary modification for PCOS.

An area yet to be explored in PCOS is the role of the gut microbiota[112], which could influence glucose homeostasis and insulin sensitivity by fermenting indigestible dietary components to produce SCFA. These SCFA have been shown to beneficially modulate adipose tissue, skeletal muscle and liver tissue function. Furthermore, research, including well-designed robust studies, is needed to evaluate the mechanisms behind the improvements observed with dietary modification and inherent with all weight-reduction interventions to improve adherence to interventions and weight maintenance.

## Conclusion

PCOS is a diverse condition that manifests itself in a variety of symptoms linked to ovulatory dysfunction and the overproduction of androgens. Women with PCOS are at a greater risk of metabolic dysfunction and consequently need regular


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monitoring for risk of diabetes and CVD. Whilst treatment is mostly aimed at addressing the presenting symptoms, dietary adaptations to improve metabolic symptoms will be beneficial for long-term health. Young women with PCOS often present to healthcare professionals due to infrequent menses or infertility; it is at this stage that they can be educated about the increased long-term risk of metabolic complications of the syndrome in order to make timely and appropriate lifestyle modifications.

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