### human reproduction update

# Comparison of dietary and physical activity behaviors in women with and without polycystic ovary syndrome: a systematic review and meta-analysis of 39 471 women

# Maryam Kazemi (1,2,\*, Joy Y. Kim<sup>1</sup>, Cynthia Wan<sup>1</sup>, Julia D. Xiong<sup>1</sup>, Julia Michalak<sup>1</sup>, Isabella B. Xavier<sup>1</sup>, Kiran Ganga<sup>1</sup>, Chau Thien Tay<sup>3</sup>, Jessica A. Grieger (1,5, Stephen A. Parry<sup>6</sup>, Lisa J. Moran (1,3,4, and Marla E. Lujan (1,3,4)

<sup>1</sup>Division of Nutritional Sciences, Human Metabolic Research Unit, Cornell University, Ithaca, NY, USA <sup>2</sup>Hilda and J. Lester Gabrilove Division of Endocrinology, Diabetes, and Bone Disease, Department of Medicine, Icahn School of Medicine at Mount Sinai, New York, NY, USA <sup>3</sup>Monash Centre for Health Research and Implementation (MCHRI), School of Public Health and Preventive Medicine, Monash University, Clayton, Australia <sup>4</sup>Robinson Research Institute, University of Adelaide, North Adelaide, SA, Australia <sup>5</sup>Adelaide Medical School, University of Adelaide, Adelaide, SA, Australia <sup>6</sup>Cornell Statistical Consulting Unit, Cornell University, Ithaca, NY, USA

\*Correspondence address. Division of Nutritional Sciences, Human Metabolic Research Unit, Cornell University, 216 Savage Hall, Ithaca, NY 14853, USA. E-mail: marla.lujan@cornell.edu https://orcid.org/0000-0002-7203-5814 (M.E.L.); Hilda and J. Lester Gabrilove Division of Endocrinology, Diabetes, and Bone Disease, Department of Medicine, Icahn School of Medicine at Mount Sinai, 18-92 Floor 18 Annenberg Building, 1468 Madison Avenue, New York, NY 10029, USA. E-mail: maryam.kazemi@cornell.edu, maryam.kazemi@mssm.edu https://orcid.org/0000-0001-6001-7208 (M.K.)

Submitted on November 05, 2021; resubmitted on April 08, 2022; editorial decision on May 04, 2022

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**BACKGROUND:** Lifestyle (dietary and/or physical activity [PA]) modification is recommended as first-line therapy to manage polycystic ovary syndrome (PCOS). Current recommendations are based on healthy lifestyle practices for the general public since evidence for unique lifestyle approaches in PCOS is limited and low quality.

**OBJECTIVE AND RATIONALE:** We aimed to synthesize evidence on dietary and PA behaviors between women with PCOS and those without PCOS. Primary outcomes were overall diet quality, total energy intake and total PA, and secondary outcomes included macronutrients, micronutrients, food groups, foods, glycemic indices, sedentary time and sitting levels. We conducted this work to identify any unique lifestyle behaviors in women with PCOS that could underlie the propensity of weight gain and obesity in PCOS and be targeted for precision nutrition and PA interventions. These findings could be used to inform future practice recommendations and research that more effectively address complications (weight gain, obesity, diabetes, infertility, cardiovascular disease and mental health) in this high-risk population.

**SEARCH METHODS:** Databases of MEDLINE, Web of Science, Scopus and CINAHL were searched until 15 February 2022 to identify observational studies documenting dietary and PA behaviors between women with PCOS and without PCOS (Controls). Studies on children, adolescents (<18 years), pregnant or menopausal-aged women (>50 years) were excluded. Data were pooled by random-effects models and expressed as (standardized) mean differences (MD) and 95% Cls. The risk of bias was assessed by the Newcastle-Ottawa scale (NOS).

**OUTCOMES:** Fifty-four studies (N = 39 471 participants; [n = 8736 PCOS; 30735 Controls]) were eligible (96%; [52/54] NOS scores  $\geq$  7). Women with PCOS had higher cholesterol (MD: 12.78, 95% CI: 1.48 to 24.08 mg/day; P = 0.03;  $l^2 = 19\%$ ), lower magnesium (MD: -21.46, 95% CI: -41.03 to -1.91 mg/day; P = 0.03;  $l^2 = 76\%$ ), and a tendency for lower zinc (MD: -1.08, 95% CI: -2.19 to 0.03 mg/day; P = 0.05;  $l^2 = 96\%$ ) intake, despite lower alcohol consumption (MD: -0.95, 95% CI: -1.67 to -0.22 g/day; P = 0.02;  $l^2 = 0\%$ ) versus Controls. Also, women with PCOS had lower total PA (standardized mean difference: -0.38, 95% CI: -0.72 to -0.03; P = 0.03;  $l^2 = 98\%$ ). Conversely, energy, macronutrients (carbohydrate, fat, protein, fiber), micronutrients (folic acid, iron, calcium, so-dium), glycemic index and glycemic load were similar (all:  $P \ge 0.06$ ). Most eligible studies reported lower total adherence to healthy eating patterns or poorer consumption of major food groups (grains, fruits, vegetables, proteins, seeds, nuts, dairy) in women with PCOS, as described narratively since variable study methodology did not permit meta-analyses.

**WIDER IMPLICATIONS:** Collective evidence supports that women with PCOS have a lower overall diet quality, poorer dietary intakes (higher cholesterol, lower magnesium and zinc) and lower total PA, despite lower alcohol consumption versus those without PCOS. Considerable heterogeneity among studies reinforces the need for research to address any relative contributions of other factors (e.g. genetic, metabolic or sociodemographic) to the observed differences. These clarifications may contribute to future evidence-based guideline recommendations on monitoring and managing PCOS in the era of precision lifestyle medicine.

Key words: polycystic ovary syndrome / obesity / lifestyle / nutrition / exercise / reproduction / PCOS

## Introduction

Characterized by hyperandrogenism, ovulatory dysfunction and/or polycystic ovarian morphology, polycystic ovary syndrome (PCOS) is a highly heritable, prevalent and complex endocrine disorder affecting up to 18% of reproductive-aged women globally (Carmina and Lobo, 1999; March et al., 2010; Dapas and Dunaif, 2022). Besides reproductive manifestations of increased risk of infertility (Teede et al., 2018) and pregnancy complications (Palomba et al., 2015), women with PCOS often exhibit cardio-metabolic aberrations, including insulin resistance (IR) and compensatory hyperinsulinemia, dyslipidemia and visceral adiposity and are at risk for developing metabolic syndrome, type 2 diabetes and sleep disturbance (Carmina and Lobo, 1999; Wild et al., 2010; Sam and Ehrmann, 2019; Kazemi et al., 2019d, 2020b). Women with PCOS are also at risk for psychosocial comorbidities, including depression, poor self-esteem, anxiety, body image issues, demoralization, social isolation and disordered eating or eating disorders (binge-eating, laxative use, purging, diet pills) (Teede et al., 2010; Naessén et al., 2019; Pirotta et al., 2019; Tay et al., 2019a; Kazemi et al., 2019c).

IR and hyperinsulinemia are key pathophysiological factors in PCOS (Diamanti-Kandarakis and Dunaif, 2012) and have been linked with aggravated hyperandrogenism and reproductive complications. Furthermore, up to 80% of women with PCOS present with overweight or obesity, which further exacerbates inherent IR and compensatory hyperinsulinemia, cardiometabolic and reproductive sequelae (Diamanti-Kandarakis and Dunaif, 2012). Weight management can, therefore, improve PCOS outcomes, in part, through reducing extrinsic IR (Teede *et al.*, 2018). The link between diet and physical activity (PA) as modifiable environmental factors and PCOS complications has biological plausibility (Kazemi *et al.*, 2020a). We and others have shown that adherence to a healthy diet and active lifestyle in women with PCOS improves metabolic, reproductive and psychological outcomes either independent of or in conjunction with, weight loss (Harrison *et al.*, 2011; Kazemi *et al.*, 2018a; Lim *et al.*, 2019; Kazemi et al., 2020a,e). Thus, the International Evidence-based Guideline for the Assessment and Management of PCOS advocates maintaining a healthy weight and preventing weight gain through lifestyle interventions in this clinical population (Teede et al., 2018).

Evidence obtained by us and others supports a propensity for obesity (Barr et al., 2011; Legro et al., 2013; Lin et al., 2019; Kazemi et al., 2021c) and weight gain in women with PCOS during their reproductive years (Teede et al., 2013; Kazemi et al., 2019c, 2021c) and pregnancy (Palomba et al., 2015), that is differentially more pronounced in patients with unhealthy lifestyle behaviors (Awoke et al., 2021). However, whether poorer lifestyle behaviors per se contribute to adiposity and associated PCOS complications have been debated. Specifically, the notion that women with PCOS have a low diet quality, excessive energy intake or engage in shorter PA sessions, is conflicting. Some studies have reported lower overall diet quality (Huijgen et al., 2015; Hosseini et al., 2017; Noormohammadi et al., 2021), increased energy intake (Ahmadi et al., 2013; Eslamian et al., 2017) and decreased PA levels (Moran et al., 2013; Eslamian et al., 2017) in women with versus those without PCOS. Conversely, others have shown higher diet quality (Moran et al., 2017), lower energy intake (Tsai et al., 2013; De Giuseppe et al., 2019) and higher PA levels (Melekoglu et al., 2020) in PCOS cohorts. There is even evidence of similar dietary and PA behaviors between the groups (Wright et al., 2004a; Douglas et al., 2006; Álvarez-Blasco et al., 2011; Cutler et al., 2019). Together, little can be concluded on any difference in dietary or PA behaviors of women with and without PCOS.

An improved understanding of suboptimal dietary and PA behaviors in women with PCOS is crucial for targeted interventions to mediate favorable changes in lifestyle behaviors and body weight. To address this knowledge gap, we conducted a systematic review and metaanalysis to comprehensively characterize and contrast dietary and PA behaviors between women with PCOS and those without PCOS. Our objective was to test the hypothesis that reproductive-aged women with PCOS would exhibit worse dietary and PA behaviors versus their counterparts without PCOS. Our primary outcomes were overall diet quality, energy intake, and total PA levels. As secondary aims, we evaluated whether other dietary and PA factors (micronutrients, macronutrients, food group intake, exercise intensity, leisure activity, sedentary behaviors) differed between groups.

## **Methods**

This systematic review was conducted according to The Cochrane Handbook of Systematic Reviews (Cochrane Handbook for Systematic Reviews of Interventions 2019), and results were reported based on the Meta-analysis Of Observational Studies in Epidemiology (MOOSE) (Stroup *et al.*, 2000). The study protocol was registered at PROSPERO (registration ID: CRD42021252178).

### Review question (PEO–Population [P], Exposure [E], Outcome [O])

The PEO criteria are detailed in Supplementary Table SI. Our study question was: in reproductive-aged women (P), do women with PCOS versus their counterparts without PCOS (E) exhibit worse dietary and PA behaviors (O)?

### **Primary and secondary outcomes**

A completed list of study outcomes is presented in Supplementary Table SI and is summarized herein. Our primary outcome was 2-fold for the diet component: overall diet quality and total energy intake; for the PA component, we evaluated total PA levels. Secondary a priori outcomes for diet components included: macronutrients (carbohydrate, fat, protein, fiber, alcohol); micronutrients (folic acid, vitamin D, iron, calcium, magnesium, zinc, sodium); glycemic index [GI]; glycemic load [GL]); and food groups (grains, fruit, vegetable, proteins, seeds and nuts, dairy, added sugar). Regarding PA, our secondary a priori outcomes included exercise intensity (mild, moderate, vigorous), leisure activity levels, and sedentary or sitting levels. Our *a priori* primary and secondary outcomes were chosen based on evidence from us and others on the clinical relevance of select dietary and PA factors to the pathophysiology and magnitude of signs and symptoms of PCOS, including adiposity (Hahn et al., 2006; Chiu et al., 2018; Kazemi et al., 2018a, 2019a,b,e; Chilibeck et al., 2020; Kazemi et al., 2020a,c,e, 2021a). Additionally, we recorded other post hoc dietary and PA outcomes (e.g. micronutrients and food intakes, engagement in various sports) to assess lifestyle behaviors comprehensively.

### Data sources and search strategy

A search for studies published from inception through 26 February 2021 was conducted using MEDLINE, Web of Science, Scopus, and CINAHL databases based on the PEO framework. Subject headings and key terms used in the search strategy for MEDLINE are detailed in Supplementary Table SII. No restrictions (e.g. language, publication year) were imposed. Manual searches of reference lists from included studies supplemented the electronic database searches. Animal studies were excluded. The search was continuously updated up to 15 February 2022.

### Inclusion and exclusion criteria

Studies were included if they met the PEO criteria (Supplementary Table SII). Observational studies on reproductive-aged (18–50 years) women in which dietary and PA behaviors were compared between cases (with PCOS) and control groups (without PCOS) were included.

Exclusion criteria included non-peer-reviewed studies; studies without designs of interest (reviews, interventions, case reports, books, reports, conference proceedings, commentaries, letters); duplicated reports from identical studies; non-human models; studies wherein outcomes of interest were not compared between groups; studies on children or adolescents (<18 years), pregnant women, or menopausal-aged women (>50 years); or studies wherein data were irretrievable after contacting their corresponding authors.

Three investigators (C.W., J.D.X. and J.Y.K.) completed the screening processes for inclusion and exclusion of studies independently using the Covidence platform (Covidence.org, Alfred Health, Australia). All disagreements were resolved by a fourth investigator (M.K.).

### **Data extraction**

The following data were extracted: first author's name, study publication year and country of conduct; participants' characteristics, including the sample sizes of PCOS and control groups and total sample size, participants' age and BMI, and racial/ethnic composition (i.e. ancestry) of PCOS and control groups; study design, setting and type of data analysis/collection (prospective/retrospective); dietary and PA assessment tool; reported dietary and PA outcomes; and variables used for matching and/or adjusting of underlying differences (confounders) between groups.

Additional information was also evaluated, including the presence of other (non-PCOS) underlying conditions; tobacco use; medication or supplement use (e.g. metformin, hormonal contraception, multivitamins), or herbs use; PCOS diagnostic criteria applied; and past knowledge of having PCOS where data were available. Where any missing or unclear data was reported, up to two attempts were made to contact the study corresponding author via email to request data or clarify methods. Six investigators (C.T., J.A.G., I.B.X., K.G., J.M. and M.K.) independently completed data extraction. All data extraction was reviewed by M.K.

### **Quality assessment**

The Newcastle-Ottawa scale (NOS) was used to evaluate the methodological quality of studies (Wells *et al.*, 2011), as described in Supplementary Table SIII and our previous work (Kakoly *et al.*, 2018), independently by six investigators (C.W., J.D.X., J.Y.K., I.B.X., K.G. and J.M.). A seventh investigator (M.K.) reviewed all quality assessments and resolved all discrepancies.

### Data synthesis and analysis

The effect sizes for each outcome measure were expressed as the weighted mean difference (MD) and 95% CI between cases (women with PCOS) and controls (women without PCOS). Studies were weighted based on the inverse of the variance for the evaluated measure with a random-effects model. Mean differences and SDs of outcome measures were collected to estimate pooled effects for all measures. Where multiple measurement units were used for reporting on specific outcomes (i.e. total carbohydrate, protein, fat [saturated fatty acids (SFAs), monounsaturated fatty acids (MUFAs), polyunsaturated fatty acids (PUFAs)], PA), we pooled data using standardized mean difference (SMD) to improve statistical power and reported the outcomes in their original measurement units to aid in clinical interpretability.

Chi-square tests were used to evaluate heterogeneity, and Cochran Q and  $l^2$  statistics were reported. The  $l^2$  value describing the percentage variation between studies was calculated as  $100\% \times ([Q - df])/Q$ , Q being the  $\chi^2$  value, and df corresponding to degrees of freedom. Low, moderate and high heterogeneity were defined using the  $l^2$  tests (Higgins *et al.*, 2003) and cutoffs of 25%, 50% and 75%, respectively. Tau-square was estimated using the restricted maximum likelihood (REML) method and used to evaluate between-study variance (Higgins *et al.*, 2003).

Sources of heterogeneity were explored using subgroup and sensitivity analyses. We performed *a priori* subgroup analyses to detect any impact of: age (categorical subgroups: <30 or  $\geq$ 30 years); BMI (<30 or  $\geq$ 30 kg/m<sup>2</sup>); PCOS diagnostic criteria (categorical: 1990 NIH (Zawadski and Dunaif, 1992), 2003 Rotterdam (Rotterdam ESHRE/ ASRM-sponsored PCOS Consensus Workshop Group 2004) or 2006 Androgen Excess and PCOS Society [AE-PCOS] criteria (Azziz *et al.*, 2006), self-reported, unspecified); dietary assessment tool used (24-h dietary recall, food record, food frequency questionnaire, researcher 913

devised surveys); and groups' country of origin (categorical) on study outcomes where sufficient data were available ( $\geq 2$  studies per subgroup). We considered subgroup analyses based on the group's racial/ethnic composition and the tool used to assess PA; however, such analyses were not possible because all but five studies (Douglas et al., 2006; Huijgen et al., 2015; Cutler et al., 2019; Lin et al., 2021; Neubronner et al., 2021) did not report on these data or included predominantly (>60%) White women, and all PA studies used survey data except a single study (Lin et al., 2019) that used accelerometer data.

Further, we performed sensitivity analyses by removing each study and recalculating the overall effect size to determine whether an individual study exerted undue influence (i.e. any alteration in the direction or statistical significance of the overall effect estimate) (Patsopoulos et al., 2008; Iyengar and Greenhouse, 2009; Cochrane Handbook for Systematic Reviews of Interventions 2019). We completed sensitivity analyses to determine the robustness of the observed overall effect estimates and, thus, any assumptions made.

Publication bias was assessed by visual inspection of funnel plots and formal testing by Begg's rank correlation tests and Egger's regression asymmetry (Begg and Mazumdar, 1994; Egger *et al.*, 1997). Each funnel plot represents all studies included for each measure; therefore, where data was presented as both SMD and 95% CI for all studies, and MD and 95% CI for certain groups of studies, we provided corresponding funnel plots with SMD data only. M.K. performed all analyses using R version 4.1.0 and RStudio version 1.4.1717 using the meta, metaviz and metacom packages (R Foundation for Statistical Computing, Austria) (Balduzzi *et al.*, 2019). Results were considered significant at P < 0.05.

# Results

### Literature search

The systematic search resulted in 16370 records (Fig. 1), of which 54 studies (61 publications) were deemed eligible and included. The selected studies comprised a total of 39471 participants and 116 experimental arms (n = 8736 PCOS [59 arms]; 30735 Controls [57 arms]). Reasons for excluding studies at each stage of the literature screening are reported in Fig. 1.

### **Study characteristics**

The general characteristics of the studies are presented in Table I and summarized herein. Studies were published between 2006 and 2022 and were conducted in Iran (Khademi *et al.*, 2010; Pourghassem Gargari *et al.*, 2011; Rajaeieh *et al.*, 2014; Sedighi *et al.*, 2014; Pourghassem Gargari *et al.*, 2015; Shishehgar *et al.*, 2016a,b; Eslamian *et al.*, 2017; Hosseini *et al.*, 2017; Kazemi Jaliseh *et al.*, 2017; Rajaeieh *et al.*, 2018; Zaeemzadeh *et al.*, 2018; Alipour *et al.*, 2019; Shahdadian *et al.*, 2019; Shishehgar *et al.*, 2019; Shahdadian *et al.*, 2019; Shishehgar *et al.*, 2019; Panjeshahin *et al.*, 2020; Shahrokhi and Naeini, 2020; Badri-Fariman *et al.*, 2021; Noormohammadi *et al.*, 2006; Colombo *et al.*, 2009; Altieri *et al.*, 2013; Barrea *et al.*, 2019), Spain (Álvarez-Blasco *et al.*, 2011; Cutillas-Tolín *et al.*, 2021; Novarro-Lafuente *et al.*, 2022), Australia (Thomson *et al.*, 2009; Moran *et al.*,

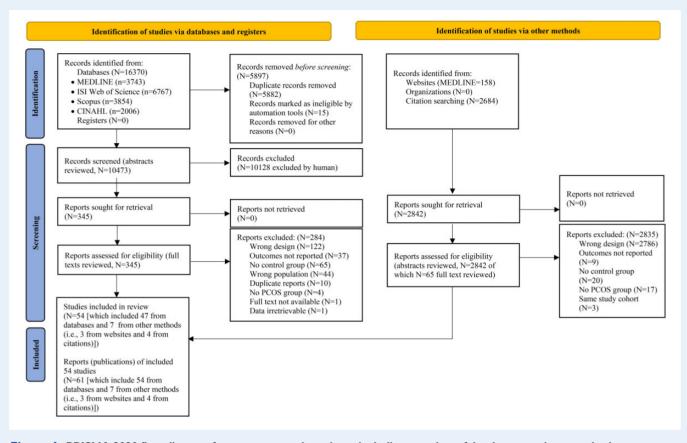


Figure 1. PRISMA 2020 flow diagram for new systematic reviews, including searches of databases, registers and other sources.

2013; Banting et al., 2014; Moran et al., 2015; Copp et al., 2020; Tay et al., 2020), Brazil (Dantas et al., 2015; Cunha et al., 2019), Canada (Cutler et al., 2019), USA (Wright et al., 2004a; Douglas et al., 2006; Lin et al., 2019, 2021), Turkey (Melekoglu et al., 2020), Croatia (Misir et al., 2016), India (Ganie et al., 2019), United Kingdom (Hart et al., 2016), Netherlands (Huijgen et al., 2015; Wang et al., 2021a,b, Poland (Jurewicz et al., 2021; Pokorska-Niewiada et al., 2021; Szczuko et al., 2021), Sweden (Larsson et al., 2016), Austria (Lerchbaum et al., 2021), Taiwan (Tsai et al., 2013) and China (Zhang et al., 2020; Liang et al., 2021; Lu et al., 2021; Wang et al., 2022).

Most (50/54; 92%) studies were conducted in medical centers, whereas five (six publications) were conducted in community settings (Khademi et al., 2010; Moran et al., 2013; Banting et al., 2014; Moran et al., 2015; Zaimzadeh et al., 2018; Tay et al., 2020). Similarly, most (44/54; 82%) studies had prospective data collection and 10 (12 publications) had retrospective data collection (Pourghassem Gargari et al., 2011; Altieri et al., 2013; Pourghassem Gargari et al., 2015; Hart et al., 2016; Panjeshahin et al., 2020; Badri-Fariman et al., 2021; Lerchbaum et al., 2021; Noormohammadi et al., 2021; Wang et al., 2021a,b; Navarro-Lafuente et al., 2022; Wang et al., 2022).

Of the 54 studies, 49 (91%) had a cross-sectional (Colombo et al., 2009; Thompson et al., 2010; Moran et al., 2013; Banting et al., 2014; Rajaeieh et al., 2014; Moran et al., 2015; Misir et al., 2016; Thara and Divakar, 2017; Rajaeieh et al., 2018; Barrea et al., 2019; Cutler et al., 2019; Shishehgar et al., 2019; Melekoglu et al., 2020; Tay et al., 2020;

Zhang et al., 2020; Lerchbaum et al., 2021; Lin et al., 2021; Neubronner et al., 2021; Pokorska-Niewiada et al., 2021; Wang et al., 2021a,b; 2022) or case-control/cohort design (Wright et al., 2004a; Orio et al., 2006; Khademi et al., 2010; Álvarez-Blasco et al., 2011; Pourghassem Gargari et al., 2011; Altieri et al., 2013; Sedighi et al., 2014; Huijgen et al., 2015; Pourghassem Gargari et al., 2015; Larsson et al., 2016; Shishehgar et al., 2016a; Eslamian et al., 2017; Hosseini et al., 2017; Kazemi Jaliseh et al., 2017; Zaeemzadeh et al., 2018; Alipour et al., 2019; Cunha et al., 2019; Ganie et al., 2019; Lin et al., 2019; Shahdadian et al., 2019; Panjeshahin et al., 2020; Shahrokhi and Naeini, 2020; Badri-Fariman et al., 2021; Cutillas-Tolín et al., 2021; Jurewicz et al., 2021; Liang et al., 2021; Lu et al., 2021; Noormohammadi et al., 2021; Soodi et al., 2021; Szczuko et al., 2021; Zirak Sharkesh et al., 2021; Navarro-Lafuente et al., 2022), 2 had a cohort design (Douglas et al., 2006; Copp et al., 2020) and 3 were observational without specifying their design (Tsai et al., 2013; Dantas et al., 2015; Hart et al., 2016).

Mean age and BMI of participants ranged from 21.0 to 48.2 years and from 19.9 to 35.5 kg/m<sup>2</sup>, respectively, across PCOS and control arms. Most studies (38/54, 70%) used the Rotterdam criteria for PCOS (Orio et al., 2006; Thomson et al., 2009; Khademi et al., 2010; Pourghassem Gargari et al., 2011; Altieri et al., 2013; Tsai et al., 2013; Sedighi et al., 2014; Dantas et al., 2015; Huijgen et al., 2015; Pourghassem Gargari et al., 2015; Larsson et al., 2016; Misir et al., 2016; Eslamian et al., 2017; Thara and Divakar, 2017; Zaeemzadeh

Author, yrs. (reference), and country	Participants' characteristics (n, mean age [yrs.], mean BMI [kg/m <sup>2</sup> ]), PCOS definition, and racial/ethnic composition	Study design, setting, data analysis	Dietary/PA assessment tool	Reported outcomes of interest
Alipour et <i>al.</i> , 2019, Iran	Total, 90 (PCOS, 45; Control, 45) PCOS, age: 26.64, BMI: 26.37 Control, age: 27.56, BMI: 25.29 PCOS definition, Rotterdam Ethnic composition, NR	<ul> <li>Design: Case-control</li> <li>Setting: AMC</li> <li>Analysis: Prospective</li> </ul>	3-d 24-h food recall (2 weekdays and 1 weekend d) <b>PA:</b> NR	<ul> <li>Diet:</li> <li><i>a priori</i> diet outcomes: ↔E, ↑total CHO, ↑starch ↔total F, ↔total P, ↔total fiber, ↑GI, ↔GL, ↔total sugar</li> <li>Other diet outcomes: NR</li> </ul>
				<ul> <li>PA:</li> <li><i>a priori</i> PA outcomes: NR</li> <li>Other PA outcomes: NR</li> </ul>
Altieri <i>et al.</i> , 2013, Italy	Total, 200 (PCOS, 100; Control, 100) PCOS, age: 27.7, BMI: 34.7 Control, age: 28.4, BMI: 34.8 PCOS definition, Rotterdam Ethnic composition, NR	Design: Case-control Setting: AMC Analysis: Retrospective	Diet: 7-d food diary (7 days of a single wk) PA: NR	<ul> <li>Diet:</li> <li><i>a priori</i> diet outcomes: ↔E, ↔total CHO (%E and g), mono- and ↔oligosaccharides (total and oligorich sweets), total F (↓%E but ↔g), ↔Chol, ↔SFA, ↔total P (%E and g), ↑total fiber, ↔total fruit, ↔total vegetable, ↔low and high saturated fat red meat, ↔fish, ↔legume, ↔low fat milk, ↔yogurt [low-fat or whole fat], ↔cheese, ↔carbonated SSB</li> <li>Other diet outcomes: ↔glycosylated end products, ↑starchy sweet with high Gl, ↔starchy food (LGI or HGI), ↔ice cream, ↔chocolate, ↔egg, ↑oil, ↓cooking fat, ↔wine, ↔beer</li> <li>PA:</li> <li>a priori PA outcomes: NR</li> <li>Other PA outcomes: NR</li> </ul>
Álvarez-Blasco et al., 2011, Spain	Total, 81 (PCOS, 22; Control, 59) PCOS, age: 26.3, BMI: 35.2 Control, age: 32.2, BMI: 34.8 PCOS definition, NIH Ethnic composition, NR	<b>Design:</b> Case-control <b>Setting:</b> AMC <b>Analysis:</b> Prospective	<b>Diet:</b> Semi-quantitative FFQ (modified, Harvard Service FFQ for Spanish population) <b>PA:</b> Self-reported question- naire (unspecified)	<ul> <li>Diet:</li> <li><i>a priori</i> diet outcomes: ↔E, ↔total CHO (%E and g), ↔total F (%E and g), ↔Chol, ↔SFA (%E and g), ↔MUFA (%E and g), ↔Turans F, ↔total P (%E and g), ↔total fiber, ↔alcohol, ↔Vit D, ↔Ca, ↔Mg, ↔Na</li> <li>Other diet outcomes: ↔n-3, ↔n-6, ↔Vit C, ↔Vit E, ↔K, ↔caffeine</li> </ul>

### Table I General characteristics of the 54 included studies in a comparison of dietary and physical activity behaviors in women with and without PCOS.

Continued

Author, yrs. (reference), and country	Participants' characteristics (n, mean age [yrs.], mean BMI [kg/m <sup>2</sup> ]), PCOS definition, and racial/ethnic composition	Study design, setting, data analysis	Dietary/PA assessment tool	Reported outcomes of interest
				<ul> <li>PA:</li> <li><i>a priori</i> PA outcomes: ↔PA (%women exercising regularly in hrs/wk)</li> <li>Other PA outcomes: NR</li> </ul>
Badri-Fariman et <i>d</i> ., 2021, Iran	Total, 240 (PCOS, 120; Control, 120) PCOS, age: NR, BMI: 29.55 Control, age: NR, BMI: 28.88 PCOS definition, Rotterdam Ethnic composition, PCOS [48.3% Fars, 29.2% Turk, 27.5% Other]: Controls [39.2% Fars, 41.7% Turk, 19.2% Other]	Design: Case-control Setting: AMC Analysis: Retrospective	Diet: 168-item FFQ (Mirmiran et al., 2010) (semi- quantitative, validated) PA: MET-based question- naire (Momenan et al., 2012) (validated, consisted of 9 dif- ferent MET levels and 3 cate- gories of PA)	<ul> <li>Diet:</li> <li>a priori diet outcomes: ↑Quasi-Western dietary pattern (↑PCOS risk), ↔E, ↓total CHO, ↓ChOl, ↑SFA ↑MUFA, ↔PUFA, ↔trans F, ↓total P, ↓fiber (total, insoluble, and crude), ↔soluble fiber, ↓Vit D, ↓Fe, ↓Mg, ↓Ca, ↓Zn, ↔Na, ↔whole grains, ↔refined grains, ↓fruits and vegetables, ↑red and organ meats, ↑processed meats, ↓fish and poultry, ↓legumes and soy, ↑solid oils and animal F, ↓nuts, ↓dairy products, ↔total sugar</li> <li>Other diet outcomes: ↓Vit A, ↓Vit B1, ↓Vit B2, ↓Vit B3, ↓Vit B5, ↓Vit B6, Vit B8, ↓Vit B12, ↓Vit C, ↔Vit E, ↓dihydrofolate, ↓tetrahydrofolate, alpha-to-copherol, ↓Ph, ↓Cu, ↓K, ↔Se, ↔Mn, ↔Chromium, ↓egg, ↑caffeine, ↑ tea and coffee, ↑SSB, ↔French fries and potato chips, ↔pickles and salinity, ↔liquid ois, ↓tomato, ↑salt, ↑sugar, ↑sweets, ↑desserts, ↑industrial juice and soft drinks ↓docsahexaenoic acid, ↔linoleic acid, ⇒eicosapentaenoic acid, ↓oliocie acid, ↓beta-cryptoxanthin, ↓beta-carotene, ↓alpha-carotene, ↓lutein, ↓lycopene, ↓glucose, ↓galactose, ↓fructose, ↓lactose, ↔maltose, ↑sucrose, ↑fluorine</li> </ul>
				PA: • <i>a priori</i> PA outcomes: ↓PA (MET-hr, ↑PCOS risk ↓MET-hr (rest or sleep)
				Other PA outcomes: NR

Author, yrs. (reference), and country	Participants' characteristics (n, mean age [yrs.], mean BMI [kg/m <sup>2</sup> ]), PCOS definition, and racial/ethnic composition	Study design, setting, data analysis	Dietary/PA assessment tool	Reported outcomes of interest
<mark>Banting et <i>al.</i>, 2014,</mark> Australia	Total, 217 (PCOS, 153; Control, 64) PCOS, age: 31.99, BMI: 31.32 Control, age: 31.50, BMI: 24.15 PCOS definition, Self-reported based on Rotterdam Ethnic composition, NR	<b>Design:</b> Cross-sectional <b>Setting:</b> Community setting <b>Analysis:</b> Prospective	Diet: NR PA: Questionnaire (self- reported trans-theoretical model, developed based on the National Physical Activity Guidelines for Australians) (Prochaska and DiClemente, 1983)	Diet: • a priori diet outcomes: NR • Other diet outcomes: NR PA: • a priori PA outcomes: NR • Other PA outcomes: JPA (%women physically active)
Barrea <i>et al.</i> , 2019, Italy	Total, 224 (PCOS, 112; Control 112) PCOS, age: 24.21, BMI: 30.95 Control, age: 24.07, BMI: 30.76 PCOS definition, Rotterdam Ethnic composition, White	Design: Cross-sectional Setting: AMC Analysis: Prospective	Diet: 14-item PREDIMED study questionnaire (Martínez-González et al., 2012) used for MED assess- ment and 7-d food diary for dietary intake assessment (nu- tritionist-administered by face-to-face interviews) PA: Questionnaire (self- reported habitual aerobic ex- ercise engagement for a mini- mum of 30 min/d [yes/no]) (Barrea et al., 2018)	<ul> <li>Diet:</li> <li><i>a priori</i> diet outcomes: ↓Overall adherence to MED based on PREDIMED score, ↔E, ↔total CHO, ↑mono-saccharides, ↓complex CHO, ↑total F, ↑SFA, ↓MUFA, PUFA (↑total, ↑n-6, ↓n-3), ↔total P, ↓total fber, ↔total fruit ≥3 servings/wk, ↔total vegetable ≥2 servings/wk, ↔%red or processed meat &lt; 1 serving/d, ↔%poultry more than red meat, ↓%fish/seafood ≥3 servings/wk, ↓%tree nuts ≥3/wk, total sugar, ↔%carbonated &lt;1/sSB</li> <li>Other diet outcomes: ↑%Low adherence to MED, ↔%average adherence to MED, ↔%high adherence to MED, ↓%use of extra virgin oil s main culinary lipid, ↔%carb virgin oil &gt; 4 tablespoons, ↔%butter, cream, margarine &lt;1 serving/d, ↔wine glasses ≥7/wk, ↔%commercial sweets and confectionary ≤2/wk ↔%use of sofrito sauce ≥2 servings/wk, ↓unsaturater fat</li> </ul>
				<ul> <li>PA:</li> <li><i>a priori</i> PA outcomes: NR</li> <li>Other PA outcomes: ↔%Moderate PA,</li> <li>↔%sedentary behaviors</li> </ul>

Author, yrs. (reference), and country	Participants' characteristics (n, mean age [yrs.], mean BMI [kg/m <sup>2</sup> ]), PCOS definition, and racial/ethnic composition	Study design, setting, data analysis	Dietary/PA assessment tool	Reported outcomes of interest
Colombo <i>et al.</i> , 2009, Italy	<b>Total</b> , 18 (PCOS, 8; functional hypothalamic amenorrhea, 10) <b>PCOS</b> , age: 21.0, BMI: 24.3 <b>Control</b> , age: 26.0, BMI: 19.9 <b>PCOS definition</b> , AEPCOS <b>Ethnic composition</b> , White	Design: Cross-sectional Setting: AMC Analysis: Prospective	<b>Diet:</b> 188-item FFQ (European Prospective Investigation into Cancer and Nutrition) (Pisani <i>et al.</i> , 1997; Pala <i>et al.</i> , 2003) and 7-d food diary <b>PA:</b> NR	<ul> <li>Diet:</li> <li><i>a priori</i> diet outcomes: ↔E (kcal and kcal/FFM kg/d), ↔total CHO (g and %E and g/FFMkg/d), ↔starch, ↔total F (g and %E and g/FFMkg/d), ↔Cho (mg and mg/FFMkg/d), ↑SFA (g and g/FFMkg/d), ↔MUFA (g and g/FFMkg/d), ↔PUFA (g and g/FFMkg/d), ↔total fiber, ↔alcohol, ↔GI, ↔GL</li> <li>Other diet outcomes: ↑Animal fat, ↔soluble sugar, ↔water</li> </ul>
				<ul> <li>PA:</li> <li><i>a priori</i> PA outcomes: NR</li> <li>Other PA outcomes: NR</li> </ul>
Copp <i>et al.</i> , 2020, Australia	Total, 7170 (PCOS, 222; Control, 6948) PCOS, age: 21.4, BMI: 27.1 Control, age: 21.4, BMI: 24.1 PCOS definition, Self-reported Ethnic composition, NR	<b>Design:</b> Cross-sectional analysis of ALSWH Study <b>Setting:</b> AMC <b>Analysis:</b> Prospective	Diet: Survey to assess vege- table intake (non-validated, self-reported) PA: Active Australia Survey (Brown <i>et al.</i> , 2008; Fjeldsoe <i>et al.</i> , 2013) (self-reported)	<ul> <li>Diet:</li> <li><i>a priori</i> diet outcomes: ↔Total vegetable</li> <li>Other diet outcomes: NR</li> <li>PA:</li> <li><i>a priori</i> PA outcomes: ↔Total PA time or intensity</li> <li>Other PA outcomes: NR</li> </ul>
Cunha et <i>al.</i> , 2019, Brazil	Total, 74 (PCOS, 39; Control, 34) PCOS, age: 25.17, BMI: 24.43 Control, age: 25.67, BMI: 23.95 PCOS definition, Rotterdam Ethnic composition, NR	Design: Case-control Setting: AMC Analysis: Prospective	<b>Diet:</b> 7-d food report <b>PA:</b> NR	<ul> <li>Diet:</li> <li><i>a priori</i> diet outcomes: ↔E (kcal and kcal/kg), ↔total CHO (g and %E and g/kg), ↔total F (g and %E and g/kg), ↔Chol (mg and mg/kg), ↔SFA (g and %E and g/kg), ↔total P (g and %E and g/kg), ↔total fiber (g and g/kg)</li> <li>Other diet outcomes: NR</li> </ul>
				PA: • a priori PA outcomes: NR • Other PA outcomes: NR

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Author, yrs. (reference), and country	Participants' characteristics (n, mean age [yrs.], mean BMI [kg/m <sup>2</sup> ]), PCOS definition, and racial/ethnic composition	Study design, setting, data analysis	Dietary/PA assessment tool	Reported outcomes of interest
Cutillas-Tolín et <i>al.</i> , 2021, Spain	Total, 276 (PCOS, 121; Control, 155) PCOS, NR Control, NR PCOS definition, Rotterdam Ethnic composition, NR	Design: Case-control Setting: AMC Analysis: Prospective	Diet: 117-item FFQ (semi- quantitative, validated) PA: NR	<ul> <li>Diet:</li> <li><i>a priori</i> diet outcomes: ↑AHEI-2010 associated with ↓risk of PCOS HA-OA but ↔Other dietary patterns (AHEI, rMED, aMED, DASH) and PCOS risk</li> <li>Other diet outcomes: NR</li> <li>PA:</li> <li><i>a priori</i> PA outcomes: NR</li> <li>Other PA outcomes: NR</li> </ul>
Cutler <i>et al.</i> , 2019, Canada	Total, 137 (PCOS, 87; Control, 50) PCOS, age: 30.7, BMI: 29.0 Control, age: 35.7, BMI: 24.1 PCOS definition, Rotterdam Ethnic composition, 40% White, 42% East Asian, 15% South Asian, 2% Aboriginal, 1% South American	<b>Design:</b> Cross-sectional <b>Setting:</b> AMC <b>Analysis:</b> Prospective	Diet: 3-d food record (2 weekdays, 1 weekend d) PA: 3-d PA record (2 week- days, 1 weekend d) and pe- dometer (SM-2000 Step Pedometer by Heart Rate Monitors USA) for steps/d	<b>Diet:</b> • <i>a priori</i> <b>diet outcomes:</b> $\leftrightarrow E$ , $\leftrightarrow$ total CHO, $\leftrightarrow$ tota F, $\leftrightarrow$ Chol, $\leftrightarrow$ total P, $\downarrow$ total fiber, $\leftrightarrow$ Vit B9, $\leftrightarrow$ Vit D, $\downarrow$ Fe (only crude but $\leftrightarrow$ in adjusted models for E), $\leftrightarrow$ Ca $\downarrow$ Mg, $\leftrightarrow$ Zn, $\leftrightarrow$ Na, $\leftrightarrow$ GL • <b>Other diet outcomes:</b> $\downarrow$ Vit A, $\leftrightarrow$ Vit C <b>PA:</b> • <i>a priori</i> PA outcomes: NR • <b>Other PA outcomes:</b> $\leftrightarrow$ steps
Dantas et <i>al.</i> , 2015, Brazil	<b>Total</b> , 27 (PCOS, 15; Control, 12) <b>PCOS</b> , age: 24.8, BMI: 32.8 <b>Control</b> , age: 29.6, BMI: 30.3 <b>PCOS definition</b> . Rotterdam	Design: NR Setting: AMC Analysis: Prospective	<b>Diet:</b> NR <b>PA:</b> Accelerometer (Actigraph accelerometer) with Freedson activity cut	Diet: • a priori diet outcomes: NR • Other diet outcomes: NR

Author, yrs. (reference), and country	Participants' characteristics (n, mean age [yrs.], mean BMI [kg/m <sup>2</sup> ]), PCOS definition, and racial/ethnic composition	Study design, setting, data analysis	Dietary/PA assessment tool	Reported outcomes of interest
	Ethnic composition, NR		points used to define PA in- tensity (Freedson <i>et al.</i> , 1998)	PA: ● <i>a priori</i> PA outcomes: NR ● Other PA outcomes: ↔Total steps/d
Douglas et <i>al.</i> , 2006, USA	<b>Total</b> , 57 (PCOS, 30; Control, 27) <b>PCOS</b> , age: 28.9, BMI 29.7 <b>Control</b> , age: 28.9, BMI 29.1 <b>PCOS definition</b> , NIH <b>Ethnic composition</b> , PCOS (83.3% White, 13.3% Black, 3.3% Other); Controls (85.1% White, 11.1% Black, 3.7% Other)	<b>Design:</b> Baseline analysis of a cohort study <b>Setting:</b> AMC <b>Analysis:</b> Prospective	<b>Diet:</b> 4-d food record (2 weekdays and 2 weekends, self-reported) and multiple- choice food questionnaire (unspecified, self-reported) <b>PA:</b> NR	<ul> <li>Diet:</li> <li><i>a priori</i> diet outcomes: ↔E, ↔total CHO, ↔total F, ↔Chol, ↔SFA, ↔MUFA, ↔PUFA, ↔trans F, ↔total P, ↔total fiber, ↔soluble fiber, ↔Mg, ↔Na</li> <li>Other diet outcomes: ↔cola beverages, ↔fried potatoes, ↔cooked potatoes, ↔white rice, ↔pasta, ↑white bread, ↔total servings of high GI foods (carbonated cola beverages, white bread, fried potatoes, cooked potatoes, white rice, and pasta)</li> </ul>
				<ul> <li>PA:</li> <li><i>a priori</i> PA outcomes: NR</li> <li>Other PA outcomes: ↔Exercise sessions (frequency)</li> </ul>
slamian <i>et al.</i> , 2017, Iran	Total, 753 (PCOS, 281; Control, 472) PCOS, age: 28.8, BMI: 31.2 Control, age: 29.4, BMI: 25.9 PCOS definition, Rotterdam Ethnic composition, NR	Design: Case-control Setting: Medical lefts (out- patient clinics) Analysis: Prospective	<b>Diet:</b> Semi-quantitative FFQ for Iranians (usual dietary in- take in past 1 yr., validated) <b>PA:</b> Questionnaire (unspeci- fied and validated, consisted of 9 different MET categories ranging from sleep to high intensity)	<ul> <li>Diet:</li> <li><i>a priori</i> diet outcomes: ↑E, ↑total CHO, ↔starch ↑total F, ↑SFA, ↔total P, ↓total fiber, ↑GI, ↑GL, grains (↓whole, ↑refined), ↔total sugar</li> <li>Other diet outcomes: NR</li> <li>PA:</li> <li><i>a priori</i> PA outcomes: ↓Total PA time</li> <li>Other PA outcomes: NR</li> </ul>
anie et <i>al.</i> , 2019, India	Total, 464 (PCOS, 144 [82 vege- tarian and 62 non-vegetarian]; Control, 320 [179 vegetarian and 141 non-vegetarian]) PCOS, age: 26.06, BMI: 24.81 Control, age: 26.55, BMI: 23.97 PCOS definition, Rotterdam Ethnic composition, NR	<b>Design:</b> Case-control <b>Setting:</b> Medical lefts <b>Analysis:</b> Prospective	<b>Diet:</b> FFQ and 72-hr dietary recalls (dietitians administered) <b>PA:</b> NR	<ul> <li>Diet:</li> <li><i>a priori diet outcomes:</i> ↑E in vegetarian PCOS versus vegetarian control but ↔E in non-vegetarian PCOS versus non-vegetarian control, ↔total CHO (g and % of E in both vegetarian PCOS versus vegetarian control and non-vegetarian PCOS versus non-vegetarian control), ↔total F (g and % of E in both vegetarian PCOS versus vegetarian control and non-vegetarian PCOS versus vegetarian PCOS versus vegetarian PCOS versus vegetarian PCOS versus vegetarian PCOS versus non-vegetarian pCOS versus vegetarian control and non-vegetarian PCOS versus non-vegetarian control), ↔total P (g and % of E in both vegetarian PCOS versus non-vegetarian control), ↔total P (g and % of E in both vegetarian pcos versus non-vegetarian control), ↔total P (g and % of E in both vegetarian pcos versus non-vegetarian control), ↔total P (g and % of E in both vegetarian pcos versus non-vegetarian control), ↔total P (g and % of E in both vegetarian pcos versus non-vegetarian control), ↔total P (g and % of E in both vegetarian pcos versus non-vegetarian control), ↔total P (g and % of E in both vegetarian pcos versus non-vegetarian control), ↔total P (g and % of E in both vegetarian pcos versus non-vegetarian control), ↔total P (g and % of E in both vegetarian pcos versus non-vegetarian pcos versus non-veg</li></ul>

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Hart et al., 2016, UK       Total, 68 (PCOS, 38; Control, 30) PCOS, age: 30.8, BMI: 24.5 Control, age: 29.3, BMI: 24.5 Control, age: 29.5, BMI: 26.6 Control, age: 29.5, BMI: 26	Study design, setting, Dietary/PA assessment Reported outcomes of interest data analysis tool
<ul> <li>Hart et al., 2016, UK</li> <li>Total, 68 (PCOS, 38; Control, 30) PCOS, age: 308, BMI: 24.5 Control, age: 29.3, BMI: 23.5 PCOS definition. Clinically established diagnosis (unspecified) Ethnic composition, 94% White, 3% Black, 3% Asian</li> <li>Hosseini et al., 2017, Iran</li> <li>Total, 297 (PCOS, 99; Control, 199)</li> <li>PCOS age: 29.0, BMI: 26.6 Control, age: 29.5, BMI: 26.6</li> <li>Hocsseini et al., 2017, Iran</li> <li>PCOS definition, AEPCOS Ethnic composition, Iranian</li> <li>Design: Case-control Analysis: Prospective</li> <li>Diet: 1: 168-Item FFQ (Instance over the past yr. daly, weekly, or monthy, interview-adminis- tered and validated) (Mirmian et al., 2010)</li> <li>PA: IPAQ (Craig et al., 2003)</li> <li>Diet: Btink: composition, Iranian</li> </ul>	both vegetarian PCOS <i>versus</i> vegetarian control and non-vegetarian PCOS <i>versus</i> non-vegetarian control) • <b>Other diet outcomes:</b> NR
<ul> <li>PCOS, age: 30.8, BMI: 24.5 Control, age: 29.3, BMI: 23.5</li> <li>PCOS definition, Clinically established diagnosis (unspecified) Ethnic composition, 94% White, 3% Black, 3% Asian</li> <li>Hosseini et al., 2017, Iran</li> <li>Total, 297 (PCOS, 99; Control, 198)</li> <li>PCOS, age: 29.0, BMI: 26.6 Control, age: 29.5, BMI: 26.6 Control, age: 29.5, BMI: 26.6</li> <li>Control, age: 29.5, BMI: 26.6</li> <li>Control, age: 29.0, BMI: 26.6</li> <li>Control, age: 29.5, BMI: 26.6</li> <li>Control, age: 29.5, BMI: 26.6</li> <li>Control, age: 29.0, BMI: 26.6</li> <li>Control, age</li></ul>	PA: • a priori PA outcomes: NR • Other PA outcomes: NR
198)       Setting: AMC       (assessed food intake over the past yr. daily, weekly, or monthly, interview-administered and validated)       • a priori diet outcomess         Control, age: 29.5, BMI: 26.6       Control, age: 29.5, BMI: 26.0       • monthly, interview-administered and validated)       • Na, grains (↓whole, ↑reft         PCOS definition, AEPCOS       Ethnic composition, Iranian       • PCOS definition, according to the past yr. daily, weekly, or monthly, interview-administered and validated)       • (Mirmiran et al., 2010)       • Analysis: Prospective         PA:       • PA:       • PA:       • Other diet outcomess       • calories from solid fat an ratio, +>greens and beans	Setting: AMC       PA: 7-d activity diary and pe- dometer (YamaxDigi-walker SW-200, worn for 7 consecu- tive d)       • a priori diet outcomes: ↔E, ↓total CHO, ↑total F, ↑SFA, ↑MUFA, ↔PUFA, ↔total P, ↔total fiber, ↔GI, ↔GL, ↓total sugar         • Other diet outcomes: NR
	Setting: AMC       (assessed food intake over the past yr. daily, weekly, or monthly, interview-administered and validated) (Mirmiran et al., 2010)       • a priori diet outcomes: ↓HEI-2010 score, ↑E, ↑total CHO, ↑total F, ↓PUFA, ↓total P, ↓total fiber, ↔NA, grains (↓whole, ↑refined), fruit (↑total, ↔Whole), vegetable (↔total, ↔starchy) protein food (↔total, red meat, processed meat, poultry, chicken, fish, ↓seafood, ↓plant protein [pulse, ↔legume]), ↓dairy (total, low fat, whole fat, fermented, processed milk, yogurt, cheese, kefir), ↔added sugar (total sugar, carbonated or non-carbonated SSB)         Other diet outcomes: ↔empty calories, ↔calories from solid fat and added sugar, ↔PUFA:SFA ratio, ↔greens and beans
· · · ·	<ul> <li><i>a priori</i> PA outcomes: ↔Total PA time or intensity</li> <li>Other PA outcomes: NR</li> </ul>

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Author, yrs. (reference), and country	Participants' characteristics (n, mean age [yrs.], mean BMI [kg/m <sup>2</sup> ]), PCOS definition, and racial/ethnic composition	Study design, setting, data analysis	Dietary/PA assessment tool	Reported outcomes of interest
Huijgen et al., 2015, Netherlands	<b>Total</b> , 1017 (PCOS, 218 [HA = 112, non-HA = 106]; Control, 799) <b>PCOS</b> , age: 28.5, BMI: 25.6 <b>Control</b> , age: 33.1, BMI: 24.5 <b>PCOS definition</b> , Rotterdam <b>Ethnic composition</b> , 56% Dutch; 44% Non-Dutch (Other)	Setting: AMC Analysis: Prospective	Diet: Questionnaire of 6 food groups to calculate Preconception Dietary Risk score (unspecified; self- reported) PA: NR	<ul> <li><i>a priori</i> diet outcomes: ↑Diet inadequacy by Preconception Dietary Risk score (all PCOS cohorts versus control)</li> <li>Other diet outcomes: ↔%inadequate whole wheat intake (&lt;4–5 slices of bread/d, HA phenotype versus non-HA phenotype versus control);</li> <li>↔%inadequate intake of MUFA or PUFA (unspecifie threshold, HA phenotype versus non-HA phenotype versus control);</li> <li>↔%inadequate intake of MUFA or PUFA (unspecifie threshold, HA phenotype versus non-HA phenotype versus control);</li> <li>↔%inadequate intake of MUFA or PUFA (unspecifie threshold, HA phenotype versus non-HA phenotype versus control);</li> <li>↔%inadequate vegetable intake (&lt;200 g/d, HA phenotype versus non-HA phenotype versus control); ↑%inadequate meat intake (&lt;3–4 serings/wk, HA phenotype versus control); ↑%inadequat fish intake (&lt;2 servings/wk, HA phenotype versus control); ↑%inadequate fish intake (&lt;2 servings/wk, HA phenotype versus control); ↑%inadequate intake (&lt;3 priori PA outcomes: NR</li> <li>Other PA outcomes: NR</li> </ul>
urewicz et <i>al.</i> , 2021, Poland	Total, 357 (PCOS, 199; Control, 158) PCOS, age: 26.6, BMI, 25.9 Control, age: 31.2, BMI, 25.0 PCOS definition, AEPCOS Ethnic composition, NR	Design: Case-control Setting: AMC Analysis: Prospective	<b>Diet:</b> Questionnaire (unspe- cified; self-reported) <b>PA:</b> NR	Diet: • <i>a priori</i> diet outcomes: ↔Alcohol • Other diet outcomes: NRPA: • <i>a priori</i> PA outcomes: NR • Other PA outcomes: NR
Kazemi Jaliseh e <i>t al.</i> , 2017, Iran	<b>Total</b> , 1702 (PCOS, 178; Control, 1524)	Design: Case-control Setting: AMC	Diet: NR	Diet: • a priori diet outcomes: NR

### Table I Continued

Author, yrs. (reference), and country	Participants' characteristics (n, mean age [yrs.], mean BMI [kg/m <sup>2</sup> ]), PCOS definition, and racial/ethnic composition	Study design, setting, data analysis	Dietary/PA assessment tool	Reported outcomes of interest
	PCOS, age: 26.4, BMI: 26.1 Control, age: 28.9, BMI: 25.4 PCOS definition, NIH Ethnic composition, NR	Analysis: Prospective	<b>PA:</b> Lipid Research Clinic questionnaire (Ainsworth <i>et al.</i> , 1993) (self-reported, validated)	<ul> <li>Other diet outcomes: NR</li> <li>PA:         <ul> <li><i>a priori</i> PA outcomes: ↔Exercise level (light, moderate, strenuous)</li> <li>Other PA outcomes: NR</li> </ul> </li> </ul>
Khademi <i>et al.</i> , 2010, Iran	Total, 294 (PCOS, 26; Control, 268) PCOS, age: 31, BMI: 23.8 Control, age: 33, BMI: 22.02 PCOS definition, Rotterdam Ethnic composition, NR	<b>Design:</b> Case-control <b>Setting:</b> Female employees of a company who had regular physical activities <b>Analysis:</b> Prospective	<b>Diet:</b> NR <b>PA:</b> Questionnaire (unspeci- fied; interviewer- administered)	<ul> <li>Diet: <ul> <li><i>a priori</i> diet outcomes: NR</li> <li>Other diet outcomes: NR</li> </ul> </li> <li>PA: <ul> <li><i>a priori</i> PA outcomes: ↔total PA time, ↔regular walking</li> <li>Other PA outcomes: NR</li> </ul> </li> </ul>
Larsson <i>et al.</i> , 2016, Sweden	Total, 102 (PCOS, 72; Control, 30) PCOS, age: 30.2, BMI: 28.5 Control, age: 27.8, BMI: 24.6 PCOS definition, Rotterdam (modified) Ethnic composition, NR	<b>Design:</b> Case-control <b>Setting:</b> AMC <b>Analysis:</b> Prospective	<b>Diet:</b> FFQ (verified in Swedish adolescents, inter- view-administered by dieti- tians) (Sjöberg et al., 2003) <b>PA:</b> NR	<ul> <li>Diet:</li> <li><i>a priori</i> diet outcomes: ↔E, ↑total CHO (g and AMDR), ↔total F (g and AMDR), ↔SFA, ↔total P, ↔total fiber, ↓alcohol, ↔whole grains</li> <li>Other diet outcomes: ↔Total EAT Score, ↔unsaturated fatty acidsPA:</li> <li><i>a priori</i> PA outcomes: NR</li> <li>Other PA outcomes: NR</li> </ul>
Lerchbaum et <i>al.</i> , 2021, Austria	Total, 330 (PCOS, 180; Control, 150) PCOS, age: 26.0, BMI: 27.6 Control, age: 35.8, BMI: 25.2 PCOS definition, Rotterdam Ethnic composition, NR	<b>Design</b> : Baseline characteris- tic (cross-sectional analysis) of a randomized clinical trial <b>Setting</b> : AMC <b>Analysis</b> : Retrospective	Diet: Questionnaire (unspecified) PA: NR	Diet: • a priori diet outcomes: ↔Vit D • Other diet outcomes: NR <b>PA:</b> • a priori PA outcomes: NR • Other PA outcomes: NR

Author, yrs. (reference), and country	Participants' characteristics (n, mean age [yrs.], mean BMI [kg/m <sup>2</sup> ]), PCOS definition, and racial/ethnic composition	Study design, setting, data analysis	Dietary/PA assessment tool	Reported outcomes of interest
Liang <i>et al.</i> , 2021, China	<ul> <li>Total, 40 (PCOS, 20 [lean 10, overweight 10]; Control, 20 [lean 10, overweight 10])</li> <li>PCOS, lean age: 24.13, BMI: 20.46; overweight age: 28.94, BMI: 27.34</li> <li>Control, lean age: 25.08, BMI: 20.43; overweight age: 30.12, BMI: 26.05</li> <li>PCOS definition, Rotterdam</li> <li>Ethnic composition, Han from Southern China</li> </ul>	Design: Case-control Setting: AMC Analysis: Prospective	<b>Diet:</b> Questionnaire (3-d 24-hr recalls; inter- viewer-administered) <b>PA:</b> NR	<b>Diet:</b> • <i>a priori</i> diet outcomes: $\leftrightarrow E$ , $\leftrightarrow$ total CHO, $\leftrightarrow$ total F, $\downarrow$ total P, $\downarrow$ total fiber, $\downarrow$ Vit D, $\leftrightarrow$ Ca, $\leftrightarrow$ Mg, $\leftrightarrow$ Na • Other diet outcomes: $\leftrightarrow$ Vit A, $\downarrow$ Vit E, $\leftrightarrow$ iodine, $\leftrightarrow$ Se, $\leftrightarrow$ Cu, $\leftrightarrow$ Mn, $\leftrightarrow$ K <b>PA:</b> • <i>a priori</i> PA outcomes: NR • Other PA outcomes: NR
Lin et al., 2019, USA	Total, 124 (PCOS, 80; Control, 44) PCOS, age: 26.8, BMI: 31.5 Control, age: 29.5, BMI: 28.0 PCOS definition, Rotterdam (with diagnostic thresholds of International Evidence-based Guideline for the Assessment and Management of PCOS) Ethnic composition, 62% White, 12% Black, 9% Asian, 17% Other	Design: Case-control Setting: AMCs Analysis: Prospective	Diet: FFQ (web-based self- reported FFQ [VioScreen] with ~1200 food images and branching questions, validated) PA: Accelerometry (Actigraph triaxial accelerom- eter) and self-reported PA (Women's Health Initiative Physical Activity Questionnaire) (Meyer et al., 2009)	<ul> <li>Diet:</li> <li><i>a priori</i> diet outcomes: ↔HEI-2015 score, ↔E, ↔total CHO (g and AMDR), ↔total F (g and AMDR), ↔SFA, ↔MUFA, ↔PUFA, ↔trans F, ↔total P, ↔total fiber, ↔soluble fiber, ↔insoluble fiber, ↔alcohol, ↔Vit B9, ↔Vit D, ↔Fe, ↔Ca, ↔Mg, ↔Zn, ↔Na, ↔GI, ↔GL, grains (↔whole, ↔refined), ↔total or whole fruit, ↔total or starchy vegetable, protein food (↔total, ↔seafood, ↔plant protein [pulse, legume]), ↔total dairy, ↔total sugar</li> <li>Other diet outcomes: ↔Vit A, ↔Vit B1, ↔Vit B2, ↔Vit B3, ↔Vit B5, ↔Vit B6, ↔Vit B12, ↔Vit C, ↔Vit E, ↔Vit K, ↔Cu, ↔Mn, ↔Ph, ↔Se, ↔caffeine, ↔fatty acidsPA:</li> <li>a priori PA outcomes: NR</li> </ul>
Lin et al., 2021, USA	Total, 569 (PCOS, 40; Control, 529) PCOS, age: 24.7, BMI: 25.5 Control, age: 25.4, BMI: 24.6 PCOS definition, NIH Ethnic composition, 46.8% Black	Design: Cross-sectional Setting: AMC Analysis: Prospective	<b>Diet:</b> CARDIA diet history questionnaire (past 28 d; in- terviewer-administered) <b>PA:</b> CARDIA PA question- naire (past yr.; self-reported)	Diet: • <i>a priori</i> diet outcomes: ↔AHEI-2010 score, ↔E, total CHO (g and AMDR), ↔total F (g and AMDR), ↔SFA, ↔MUFA, ↔PUFA, ↔trans F, ↔total fiber, ↔alcohol, ↔Vit B9, ↔Vit D, ↔Fe, ↔Ca, ↔Mg, ↔Zn, ↔Na, grains (↔whole, ↔refined), ↔whole fruit, ↔total or starchy vegetable, ↔total protein food, ↔plant protein (pulse, legume), ↔total dairy, ↔carbonated or non-carbonated SSB

Author, yrs. (reference), and country	Participants' characteristics (n, mean age [yrs.], mean BMI [kg/m <sup>2</sup> ]), PCOS definition, and racial/ethnic composition	Study design, setting, data analysis	Dietary/PA assessment tool	Reported outcomes of interest
				<ul> <li>Other diet outcomes: ↔ Vit A, ↔ Vit B1, ↔ Vit B2, ↔ Vit B3, ↔ Vit B5, ↔ Vit B6, ↔ Vit B12, ↔ Vit C ↔ Vit E, ↔ Vit K, ↔ Cu, ↔ Mn, ↔ Ph, ↔ Se, ↔ caffein ↔ fatty acids PA:</li> <li><i>a priori</i> PA outcomes: ↔ Total PA time, duratio intensity), exercise level (↔ moderate, ↔ vigorous)</li> <li>Other PA outcomes: NR</li> </ul>
Lu et al., 2021, China	Total, 650 (PCOS, 325; Control, 325) PCOS, age: 29.5, BMI: 21.8 Control, age: 30.2, BMI: 22.1 PCOS definition, Rotterdam Ethnic composition, NR	Design: Case-control Setting: AMC Analysis: Prospective	Diet: 102-item FFQ from the 2002 China National Nutrition and Health Survey (Li <i>et al.</i> , 2005) (validated) <b>PA:</b> Structured questionnaire (unspecified, interview- administered)	<ul> <li>Diet:</li> <li><i>a priori</i> diet outcomes: ↔E, ↔alcohol, ↔total 3 PUFA, ↓long chain n-3 PUFA</li> <li>Other diet outcomes: ↔fish oil supplements, ↔alpha-linoleic acid, ↓eicosapentaenoic acid, ↓docosahexaenoic acid</li> </ul>
				<ul> <li>PA:</li> <li><i>a priori</i> PA outcomes: ↓Total PA time (frequency/wk)</li> <li>Other PA outcomes: NR</li> </ul>
Melekoglu et <i>al.</i> , 2020, Turkey	Total, 130 (PCOS, 65; Control, 65) PCOS, age: 26.45, BMI: 29.7 Control, age: 26.52, BMI: 22.6 PCOS definition, Rotterdam Ethnic composition, NR	Design: Cross-sectional Setting: AMC Analysis: Prospective	<b>Diet:</b> 3-d integrated food and PA record <b>PA:</b> 3-d integrated food and PA record	<b>Diet:</b> • <i>a priori</i> diet outcomes: $\downarrow E$ , $\leftrightarrow$ total F (g and AMDR), $\uparrow$ SFA, $\leftrightarrow$ MUFA, $\leftrightarrow$ PUFA, $\leftrightarrow$ trans F, $\leftrightarrow$ total P, $\downarrow$ total fiber, soluble fiber, insoluble fiber, $\downarrow$ Vit B9, $\downarrow$ Fe, $\leftrightarrow$ Ca, $\downarrow$ Mg, $\leftrightarrow$ Zn, $\leftrightarrow$ Na, $\leftrightarrow$ Gl, $\downarrow$ GL, vegetable ( $\leftrightarrow$ total or starchy vegetable), $\leftrightarrow$ total dairy • Other diet outcomes: $\uparrow$ Vit B12

Author, yrs. (reference), and country	Participants' characteristics (n, mean age [yrs.], mean BMI [kg/m <sup>2</sup> ]), PCOS definition, and racial/ethnic composition	Study design, setting, data analysis	Dietary/PA assessment tool	Reported outcomes of interest
				<ul> <li>PA:</li> <li><i>a priori</i> PA outcomes: ↑PA (calculated as TEE/ BMR (Third report of the national cholesterol educa- tion program (NCEP) expert panel on detection, eval- uation, and treatment of high blood cholesterol in adults, 2002)</li> <li>Other PA outcomes: NR</li> </ul>
Misir e <i>t al.</i> , 2016, Croatia	Total, 28 (PCOS, 12; Control, 16) PCOS, age: NR, BMI: 27.4 Control, age: NR, BMI: 23.1 PCOS definition, Rotterdam Ethnic composition, Croatian	<b>Design:</b> Cross-sectional <b>Setting:</b> AMC <b>Analysis:</b> Prospective	<b>Diet:</b> 24-hr diet recall <b>PA:</b> Survey on the Basic data, Dietary Habits, and PA (unspecified)	<ul> <li>Diet:</li> <li><i>a priori</i> diet outcomes: ↔Total CHO (g and AMDR), ↔total F (g and AMDR), ↔SFA, ↔MUFA, ↔PUFA, ↑total P, ↑animal P, ↔total fiber</li> <li>Other diet outcomes: ↑Vit B12, ↔linoleic acidPA:</li> <li><i>a priori</i> PA outcomes: NR</li> <li>Other PA outcomes: ↔Work index, ↔sport index, ↓free time index</li> </ul>
1oran et al., 2013, 2015, Australia	Total, 7569 (PCOS, 414; Control, 7155) for MED dietary pattern and PA data 7466 (PCOS, 409; Control, 7057) for Dietary Guideline Index and all Other dietary factors PCOS, age: 33.5, BMI: 29.0 for die- tary pattern and PA data age: 33.5, BMI: 29.3 for all Other di- etary factors Control, age: 33.7, BMI: 25.4 for dietary pattern and PA data age: 33.7, BMI: 25.6 for all Other di- etary factors PCOS definition, Self-reported Ethnic composition, NR	Design: Cross-sectional analysis of ALSWH Study Setting: Community setting Analysis: Prospective	Diet: FFQ (Dietary Questionnaire for Epidemiological Studies Version 2, self-reported 100- item) PA: Self-reported (mailed survey, unspecified)	<ul> <li>Diet:</li> <li><i>a priori</i> diet outcomes: îdiet quality by Dietary Guideline Index, dietary glycemic indices, MED pattern (crude or adjusted), îmeat/fish/poultry and take-away dietary pattern (crude only), and ↔unhealthy non-com foods (crude or adjusted; all patterns indicate associa- tions with PCOS status), îE, ↔total CHO (g and AMDR), ↔mono- and oligosaccharides, ↔Other poly saccharides, ↔total F (g and AMDR), ↔Chol, ↓SFA, ↔MUFA, ↔PUFA, îtotal fiber, ↔alcohol, îVit B9, îFe, îCa, îMg, îZn, îNa, ↓GI, îGL</li> <li>Other diet outcomes: ↔Vit A, ↔Vit BI, ↔Vit B2, îVit B3, îVit B12, ↔Vit C, îVit EPA:</li> <li><i>a priori</i> PA outcomes: ↔Total PA time, îsedentary/sitting time</li> <li>Other PA outcomes: NR</li> </ul>
Javarro-Lafuente et al., 022, Spain	<b>Total</b> , 276 (PCOS, 121; Control, 155)	Design: Case-control Setting: AMC	Diet: 101-food item semi-	Diet:

Author, yrs. (reference), and country	Participants' characteristics (n, mean age [yrs.], mean BMI [kg/m <sup>2</sup> ]), PCOS definition, and racial/ethnic composition	Study design, setting, data analysis	Dietary/PA assessment tool	Reported outcomes of interest
	PCOS, age: 27.3, BMI: 25.6 Control, age: 30.6, BMI: 23.3 PCOS definition, Rotterdam Ethnic composition, PCOS (96.7% Caucasian, 3.3% South American); Control (97.4% Caucasian, 1.9% South American, 0.6% Other)	Analysis: Retrospective	quantitative FFQ (Vioque et al., 2013) <b>PA:</b> IPAQ-SF (Baecke et al., 1982)	<ul> <li><i>a priori diet outcomes:</i> ↔E, ↔total CHO, ↔total F, ↑Chol, ↔SFA, ↔MUFA, ↔PUFA, ↔trans F, ↔total P, ↔total fiber, ↓alcohol</li> <li>Other diet outcomes: ↔Omega 3, ↔omega 6, ↔omega 6/omega 3, ↔a carotene, ↔β carotene, ↔lycopene, ↔cryptoxanthin, ↔lutein, ↔zeaxanthin, ↓caffeine</li> </ul>
				<ul> <li>PA:</li> <li>a priori PA outcomes: ↔PA (hr/wk of moderate-vigorous exercise)</li> <li>Other PA outcomes: NR</li> </ul>
Neubronner et <i>al.</i> , 2021, Singapore	<b>Total</b> , 389 (PCOS, 134 [Normal BMI, 61; High BMI, 73]; Control, 255 [Normal BMI, 152; High BMI, 103]) <b>PCOS</b> , age: 29.84, BMI: 25.14 <b>Control</b> , age: 32.24, BMI: 23.08 <b>PCOS definition</b> , Rotterdam <b>Ethnic composition</b> , Chinese 71%, Malay 9%, Indian 8%, Other 12%	Design: Cross-sectional Setting: AMC Analysis: Prospective	Diet: NR PA: NR	<ul> <li>Diet:</li> <li><i>a priori</i> diet outcomes: NR</li> <li>Other diet outcomes: ↔Alcohol intake (%PCOS [obese, lean] versus control [obese, lean]), ↔coffee intake (%PCOS [obese, lean] versus control [obese, lean])</li> <li>PA:</li> <li><i>a priori</i> PA outcomes: ↔Total PA time, ↔regular walking</li> <li>Other PA outcomes: NR</li> </ul>
Noormohammadi et <i>al.</i> , 2021, Iran	Total, 891 (PCOS, 303; Control, 588) PCOS, age: 29.1, BMI: 33.7 Control, age: 28.8, BMI: 24.2 PCOS definition, Rotterdam Ethnic composition, NR	Design: Case-control Setting: AMC Analysis: Retrospective	<b>Diet:</b> 168-item FFQ (semi- quantitative, validated) <b>PA:</b> PA scale (self-reported, validated in Danish men and women)	<ul> <li>Diet:</li> <li><i>a priori</i> diet outcomes: ↓Fertility Diet score and ↑PCOS risk, ↑E, ↑total CHO (g and AMDR), ↔total F (g and AMDR), ↑total P, ↓total fiber, ↓vegetable protein, ↑animal protein, ↑GL</li> <li>Other diet outcomes: ↓Ratio of MUFA to trans I</li> </ul>
				<ul> <li>PA:</li> <li><i>a priori</i> PA outcomes: Total PA (time or intensity)</li> <li>Other PA outcomes: NR</li> </ul>
Orio et al., 2006, Italy	<b>Total</b> , 90 (PCOS, 45; Control, 45) <b>PCOS</b> , age: 21.3, BMI: 29.4	Design: Case-control Setting: AMC	Diet: NR	Diet: • a priori diet outcomes: NR

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Author, yrs. (reference), and country	Participants' characteristics (n, mean age [yrs.], mean BMI [kg/m <sup>2</sup> ]), PCOS definition, and racial/ethnic composition	Study design, setting, data analysis	Dietary/PA assessment tool	Reported outcomes of interest
	Control, age: 21.6, BMI: 29.0 PCOS definition, Rotterdam Ethnic composition, Italian	Analysis: Prospective	<b>PA:</b> Cardiopulmonary test on bicycle ergometer	<ul> <li>Other diet outcomes: NR</li> <li>PA:         <ul> <li><i>a priori</i> PA outcomes: NR</li> <li>Other PA outcomes: ↔PA score</li> </ul> </li> </ul>
Panjeshahin <i>et al.</i> , 2020, Iran	Total, 216 (PCOS, 108; Control, 108) PCOS, age: 28.95, BMI: 27.10 Control, age: 30.45, BMI: 26.63 PCOS definition, Rotterdam Ethnic composition, Iranian	Design: Case-control Setting: AMC Analysis: Retrospective	<b>Diet:</b> 178-item FFQ (semi- quantitative, modified version of a 168-item FFQ used in the Tehran Lipid and Glucose Study) <b>PA:</b> IPAQ-SH (Baecke <i>et al.</i> , 1982)	<ul> <li>Diet:</li> <li><i>a priori</i> diet outcomes: ↑High GI-high fat dietary pattern and ↑PCOS risk, ↑anti-inflammatory dietary patterns and ↓PCOS risk, ↔Iow GI-low fat dietary pattern and PCOS risk], ↑E</li> <li>Other diet outcomes:PA:</li> <li><i>a priori</i> PA outcomes: ↓Total PA time and intensity</li> <li>Other diet outcomes: NR</li> </ul>
Pokorska-Niewiada <i>et al.</i> , 2021, Poland	<b>Total</b> , 63 (PCOS, 47 [with insulin resistance, 28; without insulin resis- tance, 19]; Control, 16) <b>PCOS</b> , age: 28.3, BMI: 29.95 <b>Control</b> , age: 29.0, BMI: 23.3 <b>PCOS definition</b> , Rotterdam <b>Ethnic composition</b> , NR	Design: Cross-sectional Setting: AMC Analysis: Prospective	<b>Diet:</b> 4-d dietary food record (2 weekdays and 2 weekend d) <b>PA:</b> NR	<ul> <li>Diet:</li> <li><i>a priori</i> diet outcomes: ↔Fe, ↔Mg, ↔Zn, ↔Na (for matched groups based on insulin resistance)</li> <li>Other diet outcomes: ↔K, ↔Cu (for matched groups based on insulin resistance)</li> <li>PA:</li> <li><i>a priori</i> PA outcomes: NR</li> <li>Other PA outcomes: NR</li> </ul>
Pourghassem Gargari <i>et al.</i> , 2011, 2015, Iran	Total, 60 (PCOS, 30; Control, 30) PCOS, age: 25.8, BMI: 25.0 Control, age: 26.1, BMI: 23.7 PCOS definition, Rotterdam Ethnic composition, NR	Design: Case-control Setting: AMC Analysis: Retrospective	<b>Diet:</b> 24-hr recall (2 workday and I weekend) and FFQ (weekly, monthly, and usual dietary intake in past I yr.; unspecified)	<b>Diet:</b> • <i>a priori</i> <b>diet outcomes:</b> $\downarrow$ E, $\downarrow$ total CHO (g and %E), $\downarrow$ total F (g, but $\leftrightarrow$ %E), $\downarrow$ SFA, $\leftrightarrow$ MUFA, $\leftrightarrow$ PUFA, $\downarrow$ total P (g, but $\leftrightarrow$ %E), $\leftrightarrow$ total fiber, $\leftrightarrow$ Vit B9, $\downarrow$ Vit D,

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Author, yrs. (reference), and country	Participants' characteristics (n, mean age [yrs.], mean BMI [kg/m <sup>2</sup> ]), PCOS definition, and racial/ethnic composition	Study design, setting, data analysis	Dietary/PA assessment tool	Reported outcomes of interest
			PA: NR	<ul> <li>↓Ca, ↓Mg, ↔Zn, ↔Na, ↓total fruit, ↓nuts, ↓total dair and milk, ↔total sugar</li> <li>Other diet outcomes: ↔Chromium, ↔Vit B12.</li> <li>↑onion</li> </ul>
				PA: • a priori PA outcomes: NR • Other PA outcomes: NR
Rajaeieh et <i>al.</i> , 2014, 2018, Iran	Total, 400 (PCOS, 40; Control, 360) PCOS, age: 26.8, BMI: 23.99 Control, age: 29.8, BMI: 24.13 PCOS definition, Clinically established diagnosis (sonography diagnostic assessment with checking women clinical manifestations) Ethnic composition, Iranian	Design: Cross-sectional Setting: AMC Analysis: Prospective	<b>Diet:</b> 168-item FFQ (developed for the Tehran Lipid and Glucose Study) <b>PA:</b> IPAQ-SH (Baecke <i>et al.</i> , 1982)	<ul> <li>Diet:</li> <li><i>a priori</i> diet outcomes: ↔E, dairy (↔total and low fat and whole fat milk, ↔total, high-fat, and low-f yogurt, ↔cheese)</li> <li>Other diet outcomes: ↔Cocoa milk, ↔skim milk, or other kinds of milk (except for skim, low-fat, whole fat, or coca milk), ↔dough, ↔curd, ↔ice creamPA:</li> <li><i>a priori</i> PA outcomes: ↔PA (% of women with mild, moderate, or vigorous PA)</li> <li>Other PA outcomes: NR</li> </ul>
Sedighi <i>et al.</i> , 2014, Iran	Total, 130 (PCOS, 65; Control, 65) PCOS, age: 28.85, BMI: 24.02 Control, age: 29.57, BMI: 23.47 PCOS definition, Rotterdam Ethnic composition, NR	Design: Case-control Setting: AMC Analysis: Prospective	<b>Diet:</b> 28-item researcher-de- vised questionnaire (0–112 points wherein higher scores showed adherence to a more appropriate diet) <b>PA:</b> IPAQ	<ul> <li>Diet:</li> <li><i>a priori</i> diet outcomes: ↓Diet score</li> <li>Other diet outcomes: NRPA:</li> <li><i>a priori</i> PA outcomes: ↓Total PA time and intensity</li> <li>Other PA outcomes: NR</li> </ul>
Shahdadian et <i>al.</i> , 2019, Iran	Total, 570 (PCOS, 225; Control, 345) PCOS, age: 29.51, BMI: 24.87 Control, age: 28.56, BMI: 24.35 PCOS definition, Rotterdam Ethnic composition, Iranian	Design: Case-control Setting: AMC and private medical left Analysis: Prospective	<b>Diet:</b> 168-item semi-quanti- tative FFQ (verified in Iranian population) (Asghari et al., 2012) <b>PA:</b> IPAQ-SH (Baecke et al., 1982)	<ul> <li>Diet:</li> <li><i>a priori</i> diet outcomes: ↔E, ↑total CHO, ↑total ↑total P</li> <li>Other diet outcomes: NRPA:</li> </ul>

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Author, yrs. (reference), and country	Participants' characteristics (n, mean age [yrs.], mean BMI [kg/m <sup>2</sup> ]), PCOS definition, and racial/ethnic composition	Study design, setting, data analysis	Dietary/PA assessment tool	Reported outcomes of interest
				<ul> <li><i>a priori</i> PA outcomes: Total PA time and intensity</li> <li>Other PA outcomes: NR</li> </ul>
ihahrokhi and Naeini, 1020, Iran	Total, 150 (PCOS, 60; Control, 90) PCOS, age: 32.2, BMI: 26.4 Control, age: 32.4, BMI: 24.7 PCOS definition, Clinically established diagnosis (unspecified) Ethnic composition, NR	Design: Case-control Setting: AMC Analysis: Prospective	<b>Diet:</b> 168-item FFQ (self- reported in the past yr., on daily, weekly, or monthly basis) <b>PA:</b> IPAQ-SH (Baecke <i>et al.</i> , 1982)	<ul> <li>Diet:</li> <li><i>a priori</i> diet outcomes: ↑E, ↓Zn</li> <li>Other diet outcomes: ↓Vit C, ↓Vit E, ↓Se, ↓beta carotenePA:</li> <li><i>a priori</i> PA outcomes: ↔Total PA time or intensity</li> <li>Other PA outcomes: NR</li> </ul>
ihishehgar et <i>al.</i> , 2016a, ran	Total, 282 (PCOS, 142; Control, 140) PCOS, age: 28.56, BMI: 26.56 Control, age: 28.95, BMI: 26.04 PCOS definition, AEPCOS Ethnic composition, Iranian	<b>Design:</b> Case-control <b>Setting:</b> AMC <b>Analysis:</b> Prospective	<b>Diet:</b> 147-item FFQ (interview-administered; participants reported the frequency of consumption of each food item per d, wk or mo during previous yr.) <b>PA:</b> IPAQ	<ul> <li>Diet:</li> <li><i>a priori</i> diet outcomes: ↔E, ↔total CHO (g and %E), ↔total F (g and %E), ↔SFA (g and %E), ↔MUF (g and %E), PUFA (↔g but ↑% E), trans F NR, ↔total P (g and %E), ↔total fiber, ↑Na, ↔GI, ↔GL, ↔fruit (total, whole fruit, fruit juice), vegetable (↓total, ↔starchy vegetable), protein food (↔red meat, ↔chicken, plant protein [↓legume]), dairy (↔low fat, ↔high fat), ↔carbonated SSB</li> <li>Other diet outcomes: ↑Egg, ↔oil, ↔fast foods, ↑high Gl foods, ↔medium and low Gl foods</li> </ul>
				<ul> <li>PA:</li> <li><i>a priori</i> PA outcomes: ↔ Total PA time,</li> <li>↔ exercise level (mild, moderate, vigorous), ↑sitting time</li> <li>Other PA outcomes: NR</li> </ul>
nishehgar et <i>al.</i> , 2019, an	Total, 73 (PCOS, 33; Control, 40) PCOS, age: 29.7, BMI: 31 Control, age: 30.8, BMI: 30.9 PCOS definition, Rotterdam Ethnic composition, NR	Design: Baseline characteris- tic (cross-sectional analysis) of an interventional study Setting: AMC Analysis: Prospective	<b>Diet:</b> 3-d dietary food record (2 working days and 1 week- end d) <b>PA:</b> IPAQ-SH (Baecke et al., 1982)	<ul> <li>Diet:</li> <li><i>a priori</i> diet outcomes: ↔E, ↔total CHO (g an %E), ↔total F (%E), ↔total P (%E), ↔total fiber, ↔GL</li> <li>Other diet outcomes: NRPA:</li> </ul>

Author, yrs. (reference), and country	Participants' characteristics (n, mean age [yrs.], mean BMI [kg/m <sup>2</sup> ]), PCOS definition, and racial/ethnic composition	Study design, setting, data analysis	Dietary/PA assessment tool	Reported outcomes of interest
				<ul> <li><i>a priori</i> PA outcomes: ↔ Total PA time or intensity</li> <li>Other PA outcomes: NR</li> </ul>
Soodi <i>et al.</i> , 2021 and Zirak Sharkesh et <i>al.</i> , 2021, Iran	Total, 494 (PCOS, 203; Control, 291) PCOS, age: 28.98, BMI: 25.74 Control, age: 30.15, BMI: 23.65 PCOS definition, Rotterdam Ethnic composition, Iranian	Design: Case-control Setting: AMC Analysis: Prospective	Diet: 147-item FFQ (vali- dated for Iranian population) (Esfahani et al., 2010) (self-reported) PA: IPAQ-SH (Baecke et al., 1982)	<ul> <li>Diet:</li> <li><i>a priori</i> diet outcomes: ↓Total DDS, ↔grain DDS, ↔fruit DDS, ↓vegetables DDS, ↓meat/meat product DDS, ↔dairy DDS, ↔E, ↑total CHO, ↔tota F, ↑Chol, ↔SFA, ↓MUFA, ↔PUFA, ↔total P, ↓total ber, ↔Vit B9, ↓Vit D, ↔Fe, ↔Ca, ↔Mg, ↔Zn, ↔N grains (↔whole, ↑refined), ↔fruit, ↔vegetables, ↔protein food (↔red and processed meat, plant protein [(↔legume]), ↓dairy</li> <li>Other diet outcomes: ↔ Vit B6, ↓Vit B12, ↔V C, ↔Vit E, ↔K, ↔ Ph ↔ chromium, ↔caffeine, ↔fructose, ↔glucose, ↓omega 3PA:</li> <li><i>a priori</i> PA outcomes: ↓Total PA time (MET min/d)</li> <li>Other PA outcomes: NR</li> </ul>
Szczuko et <i>al.</i> , 2021, Poland	Total, 55 (PCOS, 40; Control, 15) PCOS, age: 32.52, BMI: 29.65 Control, age: 30.23, BMI: 22.22 PCOS definition, Rotterdam Ethnic composition, White	Design: Case-control Setting: AMC Analysis: Prospective	<b>Diet:</b> 3-d food diary (inter- view-administered) <b>PA:</b> NR	<ul> <li>Diet:</li> <li><i>a priori</i> diet outcomes: ↓Vit B9</li> <li>Other diet outcomes: ↔Vit B1, ↔Vit B2, ↔Vi B3, ↔Vit B6, ↔Vit B12, ↓Vit CPA:</li> <li><i>a priori</i> PA outcomes: NR</li> <li>Other PA outcomes: NR</li> </ul>
Tay et al., 2020, Australia	Total, 7847 (PCOS, 796; Control, 7051) PCOS, age: 24.8, BMI: 29.2 Control, age: 24.6, BMI: 25.3 PCOS definition, Self-reported Ethnic composition, NR	Design: Cross-sectional analysis of the ALSWH study Study Setting: Community setting Analysis: Prospective	Diet: NR PA: Active Australia Survey (Brown et al., 2008; Fjeldsoe et al., 2013) (self-reported)	<ul> <li>Diet:</li> <li><i>a priori</i> diet outcomes: NR</li> <li>Other diet outcomes:  →Alcohol drinking patter (% of non-, low-risk, and high-risk drinkers) PA:</li> <li><i>a priori</i> PA outcomes: ↓Total PA time, ↑sedentary/sitting time</li> <li>Other PA outcomes: ↓%Meeting Australian PA guidelines for weight maintenance (≥500 MET.min/wk), ↓%meeting Australian PA guidelines for weight</li> </ul>

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Author, yrs. (reference), and country	Participants' characteristics (n, mean age [yrs.], mean BMI [kg/m <sup>2</sup> ]), PCOS definition, and racial/ethnic composition	Study design, setting, data analysis	Dietary/PA assessment tool	Reported outcomes of interest
				loss ( $\geq$ 833 MET.min/wk), $\leftrightarrow$ high ( $\geq$ 8 hrs/d) sitting time
hara and Divakar, 2017, ndia	Total, 80 (PCOS, 40; Control, 40) PCOS, age: range 26–30, BMI: NR Control, age: range 26–30, BMI: NR PCOS definition, Rotterdam Ethnic composition, NR	Design: Cross-sectional Setting: AMC Analysis: Prospective	<b>Diet:</b> 24-hr recall (structured questionnaire, unspecified) <b>PA:</b> NR	<ul> <li>Diet:</li> <li><i>a priori</i> diet outcomes: ↑E, ↑total CHO, ↑total ↑total P, ↓Fe, ↔Ca</li> <li>Other diet outcomes: ↓Vit A</li> <li>PA:</li> <li><i>a priori</i> PA outcomes: NR</li> </ul>
Fhomson et al., 2009,	<b>Total</b> , 26 (PCOS, 10; Control, 16)	Design: Cross-sectional	Diet: NR	Other PA outcomes: NR     Diet:
ustralia	PCOS, age: 33.6, BMI: 34.1 Control, age: 36.8, BMI: 35.5 PCOS definition, Rotterdam Ethnic composition, NR	Setting: AMC Analysis: Prospective	<b>PA:</b> IPAQ-SF (Baecke <i>et al.</i> , 1982) and questionnaires (Philippaerts <i>et al.</i> , 1999) (self-reported and recorded PA over the previous yr., unspecified).	<ul> <li><i>a priori</i> diet outcomes: NR</li> <li>Other diet outcomes: NRPA:</li> <li><i>a priori</i> PA outcomes: ↔Exercise level (light, moderate, strenuous), ↔leisure activity level (walki gardening)</li> <li>Other PA outcomes: ↔Total PA score, ↔tim</li> </ul>
				to exhaustion (exercise tolerance)
<sup>-</sup> sai <i>et al.</i> , 2013, Taiwan, COC	Total, 206 (PCOS, 45; Control, 161) PCOS, age: 32.7, BMI: 23.0 Control, age: 34.7, BMI: 21.3 PCOS definition, Rotterdam Ethnic composition, NR	Design: NR Setting: AMC Analysis: Prospective	<b>Diet:</b> 3-d dietary record (2 weekdays and 1 weekend d; records were verified by a registered dietitian via tele- phone interview) <b>PA:</b> NR	<ul> <li>Diet:</li> <li><i>a priori</i> diet outcomes: ↓E, ↓total CHO, ↔F (g but ↑%E), ↔total P</li> <li>Other diet outcomes: NRPA:</li> <li><i>a priori</i> PA outcomes: NR</li> <li>Other PA outcomes: NR</li> </ul>
Vang et <i>al.</i> , 2021a,b, Netherlands	Total, 491 (PCOS, 170; Control, 321) PCOS, age: 28; BMI: 36 Control, age: 30.8, BMI: 36 PCOS definition, Rotterdam Ethnic composition, PCOS (90% Western European); Control (89.4% Western European)	Design: Cross-sectional Setting: AMC Analysis: Retrospective	Diet: FFQ (van den Brink, 2005) PA: Short questionnaire to Assess Health Enhancing PA (Wendel-Vos et al., 2003), Pedometer (Yamax Digi- Walker SW 200, Develing	<ul> <li>Diet:</li> <li><i>a priori</i> diet outcomes: ↔E, ↔alcohol, ↓total wetable, ↔total fruit</li> <li>Other diet outcomes: ↔Sugary drink, ↔savorsnack ↔sweet snack</li> </ul>

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Author, yrs.	Participants' characteristics	Study design, setting,	Dietary/PA assessment	Reported outcomes of interest
(reference), and country	(n, mean age [yrs.], mean BMI [kg/m <sup>2</sup> ]), PCOS definition, and racial/ethnic composition	data analysis	tool	
			International <sup>®</sup> , Bunschoten, The Netherlands)	<ul> <li><i>a priori</i> PA outcomes: ↔Total PA (moderate to vigorous min/wk)</li> <li>Other PA outcomes: ↔Leisure time (moderate to vigorous min/wk), ↔commuting time (moderate to vigorous min/wk), ↔steps</li> </ul>
Wang et <i>al.</i> , 2022, China	Total, 527 (PCOS, 202; Control, 325) PCOS, age: 30.15, BMI: NR Control, age: 31.77, BMI: NR PCOS definition, Rotterdam Ethnic composition, NR	Design: Cross-sectional Setting: AMC Analysis: Retrospective	<b>Diet:</b> Chinese Health and Nutrition Survey question- naire (Zhang <i>et al.</i> , 2014) <b>PA</b> : NR	<ul> <li>Diet:</li> <li><i>a priori</i> diet outcomes: ↑DII, ↓Mediterranean diet (↑PCOS risk), ↑meat-egg and ↑shellfish-shrimp-dairy diets (positive associations with PCOS risk), ↔E, ↔total CHO, ↑Chol, ↑total F, ↑PUFA, ↔MUFA, ↔trans F, ↔total P, ↓Vit B9, ↑Vit D, ↔Fe, ↔Mg, ↔Zn, ↓total fiber</li> <li>Other diet outcomes: ↓Vit A, ↓Vit C, ↔Vit E, ↔Vit B1, ↔Vit B2, ↔Vit B3, ↔Vit B6, ↑Vit B12, ↓beta-carotene, ↔daidzein, ↑SePA:</li> <li><i>a priori</i> PA outcomes: ↔Total PA time (MET-hrs/wk)</li> <li>Other PA outcomes: NR</li> </ul>
Wright et al., 2004a, USA	<b>Total</b> , 163 (PCOS, 84; Control, 79) <b>PCOS</b> , age: 46.7, BMI: 32.1 <b>Control</b> , age: 48.2, BMI: 29.0 <b>PCOS definition</b> , NIH (OA+[HA/↑LH/FSH]) <b>Ethnic composition</b> , PCOS [83% White, 17% Non- White]; Controls [90% White, 10% Non- White]	Design: Case-control Setting: AMC Analysis: Prospective	<b>Diet:</b> 109-item diet history questionnaire (Block et al., 1986) (self-administered and modified version of the Block questionnaire <b>PA:</b> Paffenbarger PA Questionnaire (self-reported or interviewer-administered)	<ul> <li>Diet:</li> <li><i>a priori</i> diet outcomes: ↔E, ↔total CHO, ↔tota F, ↔Chol, ↔MUFA, ↔PUFA, ↔total P</li> <li>Other diet outcomes: NRPA:</li> <li><i>a priori</i> PA outcomes: Exercise level (↔light, ↔moderate, ↔strenuous), ↔sedentary/sitting time</li> <li>Other PA outcomes: ↔Sleeping/reclining time</li> </ul>
Zaeemzadeh et <i>al.</i> , 2018, Iran	<b>Total</b> , 182 (PCOS, 151 [sub- groups: frank: 41; non-PCO, 37; ovulatory: 33; mild: 40]; Control, 31) <b>PCOS,</b> subgroups, frank, age: 28.1; BMI: 25.5; non-PCO: 29.7; BMI: 25.1; ovulatory, 27; BMI: 25.3; mild, age: 27; BMI: 25.0	Design: Case-control Setting: AMC Analysis: Prospective	<b>Diet:</b> 168-food item FFQ (validated for Iranian population) <b>PA:</b> NR	<ul> <li>Diet:</li> <li><i>a priori diet outcomes:</i> ↑E (all PCOS subgroups versus control), ↑total CHO (all PCOS subgroup versu control), ↓total F (all PCOS subgroup versus control), ↓MUFA (only frank PCOS subgroup versus control), ↓PUFA (only frank PCOS subgroup versus control), ↓ruran F only frank PCOS subgroup versus control), ↔total P</li> </ul>

Continued

Author, yrs. (reference), and country	Participants' characteristics (n, mean age [yrs.], mean BMI [kg/m <sup>2</sup> ]), PCOS definition, and racial/ethnic composition	Study design, setting, data analysis	Dietary/PA assessment tool	Reported outcomes of interest
	Control, age: 29.8, BMI: 25.0 PCOS definition, Rotterdam Ethnic composition, Iranian			<ul> <li>(all PCOS subgroup versus control), ↓total fiber (all PCOS subgroup versus control), ↔soluble fiber, ↓Zn (all PCOS subgroup versus control)</li> <li>Other diet outcomes: ↔Chromium (only non-PCO PCOS subgroup versus control), ↓Se (all PCOS subgroup versus control), ↔carotenoid (all PCOS subgroup versus control)</li> <li>PA:         <ul> <li>a priori PA outcomes: ↔Total PA time</li> <li>Other PA outcomes: NR</li> </ul> </li> </ul>
Zhang et al., 2020, China	Total, 2496 (PCOS, 2217 [sub- groups: 1979 PCOS with OA; 238 PCOS with normal anovulation]; Control, 279) PCOS, subgroups: PCOS with OA, age: 31.11, BMI: 24.90 PCOS with normal ovulation, age: 31.56, BMI: 25.31) Control, age: 29.81, BMI: 22.93 PCOS definition, Rotterdam Ethnic composition, NR	<b>Design:</b> Baseline analysis of a cohort study <b>Setting:</b> AMC <b>Analysis:</b> Prospective	<b>Diet:</b> Questionnaire (interviewer-administered; unspecified) <b>PA:</b> Questionnaire (interviewer-administered; unspecified)	<ul> <li>Diet:</li> <li><i>a priori</i> diet outcomes: ↔%Alcohol consumption (either PCOS with or without OA subgroup versus control)</li> <li>Other diet outcomes: ↑%Tea drinking, I cup/d for at least 6 mo (either PCOS with or without OA subgroup versus control)PA:</li> <li><i>a priori</i> PA outcomes: ↔Total PA time in either PCOS with or without OA subgroup versus control</li> <li>Other PA outcomes: NR</li> </ul>

<sup>1</sup>denote increases in evaluated outcome measures in PCOS compared to control group; ↓denote decreases in evaluated outcome measures in PCOS compared to control group; AEPCOS, Androgen Excess and Polycystic Ovary Syndrome; AHEI, Alternative Healthy Eating Index; ALSWH, Australian Longitudinal Study on Women's Health; AMC, academic medical left; AMDR, Acceptable Macronutrient Distribution Range; aMED, alternate Mediterranean Dietary Score; BMR, basal metabolic rate; CARDIA, Coronary Artery Risk Development in Young Adults; CHO, carbohydrate; Chol, cholesterol; Cu, copper; d, day; DASH, Dietary Approaches to Stop Hypertension; DDS, Dietary Diversity Score; DII, dietary inflammatory index; E, energy; EAT, Eating Attitudes Test; F, fat; Fe, iron; FFM, fat free mass; FFQ, food frequency questionnaire; g, gran; Gl, glycemic Index; GL, glycemic load; h, hour; HA, hyperandrogenic; HA-OA, hyperandrogenism + oligo/amenorrhea; HEI, Healthy Eating Index; HeI, high glycemic index; h(s), hour(s); IPAQ. International Physical Activity Questionnaire; IPAQ-SH, International Physical Activity Questionnaire; ME, massi, Kilocalories; LGI, low glycemic index; LH/FSH, LH/FSH, LH/FSH, acids; NIH, National Institutes of Health; NR, not-reported; P, protein; PA, physical activity; Ph, phosphorus; PREDIMED, Prevención con Dieta Mediterranea; PUFA, solyunsaturated fatty acids; SSB, sugar-sweetened beverages; TEE, total energy expenditure; Vir, vitamin; wk, week; yrs., years; Zn, zinc.

et al., 2018; Alipour et al., 2019; Barrea et al., 2019; Cunha et al., 2019; Cutler et al., 2019; Ganie et al., 2019; Lin et al., 2019; Shahdadian et al., 2019; Shishehgar et al., 2019; Melekoglu et al., 2020; Panjeshahin et al., 2020; Zhang et al., 2020; Badri-Fariman et al., 2021; Cutillas-Tolín et al., 2021; Lerchbaum et al., 2021; Liang et al., 2021; Lu et al., 2021; Neubronner et al., 2021; Noormohammadi et al., 2021; Pokorska-Niewiada et al., 2021; Soodi et al., 2021; Szczuko et al., 2021; Zirak Sharkesh et al., 2021; Wang et al., 2021a,b; Navarro-Lafuente et al., 2022; Wang et al., 2022), whereas five used the National Institutes of Health (NIH) (Wright et al., 2004b; Douglas et al., 2006; Álvarez-Blasco et al., 2011; Kazemi Jaliseh et al., 2017; Lin et al., 2021), and four used the Androgen Excess and Polycystic Ovary Syndrome (AEPCOS) (Colombo et al., 2009; Shishehgar et al., 2016a,b; Hosseini et al., 2017; Jurewicz et al., 2021) criteria. Three did not specify clinical criteria (Rajaeieh et al., 2014; Hart et al., 2016; Rajaeieh et al., 2018; Shahrokhi and Naeini, 2020) and four used selfreported PCOS history (Moran et al., 2013; Banting et al., 2014; Moran et al., 2015; Copp et al., 2020; Tay et al., 2020).

### Quality assessment

Supplementary Table SIII shows the NOS quality assessment scores of each study. Most (42/54, 78%) studies had high quality (NOS score  $\geq$  8), and 12 (24%) were considered poor quality by seven stars (Orio *et al.*, 2006; Colombo *et al.*, 2009; Khademi *et al.*, 2010; Moran *et al.*, 2013; Banting *et al.*, 2014; Rajaeieh *et al.*, 2011; Hart *et al.*, 2016; Thara and Divakar, 2017; Rajaeieh *et al.*, 2018; Copp *et al.*, 2020; Szczuko *et al.*, 2021) and six stars (Shahrokhi and Naeini, 2020; Tay *et al.*, 2020), respectively, because comparability of their groups were not confirmed by a satisfactory record.

### Systematic review

Some outcome measures (diet quality, PA characteristics, food group) were compared qualitatively between groups herein, as pooling analyses were not possible.

Alpour et al., 2019 Alleri et al., 2013 Alleri et al., 2013 Alleri et al., 2013 Alleri et al., 2013 Alleri et al., 2014 Avarez-Bisco et al., 2011 122 2245, 200, 810, 592 22374, 061, 592 225, 016, 592 22374, 067, 110 2245, 272, 4 953, 183, 164, 251, 31% Avarez-Bisco et al., 2019 122 2245, 290, 81, 122 254, 827, 24 953, 183, 164, 251, 31% Colombo et al., 2019 39, 1654, 5667, 10, 100, 114, 491, 2 Colombo et al., 2019 39, 1654, 5667, 10, 100, 114, 491, 2 Colombo et al., 2019 Colombo et al., 2019 Colombo et al., 2019 Colombo et al., 2016 Colombo et al., 2017 Colombo et al., 2017 Colombo et al., 2017 Colombo et al., 2020 Colombo et al., 2020 Colombo et al., 2020 Colombo et al., 2020 Colombo et al., 2021 Colombo e	Author and Year	Total	Mean	PCOS SD	Total	( Mean	Control SD	Mean Difference	MD	95%-Cl Weight
Allier et al. 20131002220.0457.01002223.0405.03.00122.68; 116.89]3.1%Avarez-Biasco et al., 20191122245.3290.81122254.8272.460.0130.25; 342.25]2.4%Colombo et al., 200982471.2676.1102010.1492.14451.10 $-97.94$ , 102.01.411.0%Curlat et al., 2019391651.4566.734.1487.9491.2451.01 $-97.94$ , 102.01.411.0%Curlat et al., 2010301783.0333.0501815.0374.0-32.00 $-157.07$ , 93.073.0%Estamian et al., 20172812215.0721.0472248.0661.0726.00 $-726.00$ $-627.67$ , 624.33]3.1%Ganie et al., 201621805.5308.01411895.42.08111 $-143.96$ , 84.183.1%Hart et al., 2017999200.0822.00198.236.0746.0-250.00 $-259.00$	Alipour et al., 2019	45	1919.0	359.0	45	1880 0	337.0	#	39.00	[-104 86 <sup>-</sup> 182 86] 3 0%
Avarez-Biasco et al., 2011       22 2374.0       681.0       59       268.0       702.0       6.00 $[330.25, 342.25]$ 2.4%         Barrea et al., 2019       112 2245.3       290.8       112 2245.3       290.8       112 2245.3       290.8       112 2245.3       290.8       112 2245.3       290.8       112 2245.3       290.8       112 2245.3       290.8       112 2245.3       290.8       112 2245.3       290.8       112 2245.3       290.8       112 2245.3       290.8       112 2245.3       290.8       112 2245.3       290.8       112 2245.3       290.8       112 2245.3       290.7       1163.54       1.79.14, 406.22       2.7%         Cutter et al., 2019       0.00       1783.9       379.3       27       1781.5       444.8       2.40       [213.01, 210.2       28%         Estamian et al., 2017       291.8       2183.25       30.8       141       198.4       20.8       10.1       1.74.78       85.40       3.1%         Ganie et al., 2016       201.90       072.0       92.050.0       779.0       -40.00       1.286.69       306.69       2.3%         Larsson et al. 2016       2020       0.2218.0       879.6       2.525.7       1.74.78       85.41.83       3.1%										· · · · · · · · · · · · · · · · · · ·
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								Favors Control Favors PCOS		

Figure 2. Forest plot for energy intake in women with and without PCOS with effect estimate expressed as kcal/day. MD, mean difference.

### Primary outcomes

Thirteen studies (14 publications) (Sedighi et al., 2014; Huijgen et al., 2015; Moran et al., 2015; Hosseini et al., 2017; Barrea et al., 2019; Lin et al., 2019; Panjeshahin et al., 2020; Badri-Fariman et al., 2021; Cutillas-Tolín et al., 2021; Lin et al., 2021; Noormohammadi et al., 2021; Soodi et al., 2021; Zirak Sharkesh et al., 2021; Wang et al., 2022) evaluated diet quality between PCOS and control groups using various indices; thus pooled analyses were impossible. Details of the study groups and characteristics are elaborated in Table I. Most (9/13; 69%) studies reported lower diet quality in PCOS versus Control groups as assessed by lower adherence to the Prevención con Dieta Mediterránea (PREDIMED) score (Barrea et al., 2019), Healthy Eating Index (HEI-2015) (Hosseini et al., 2017), researcher-devised questionnaires (Sedighi et al., 2014), diet diversity score (Soodi et al., 2021; Zirak Sharkesh et al., 2021), higher diet inadequacy using the Preconception Dietary Risk (Huijgen et al., 2015) score, or inflammatory potential of a diet using the dietary inflammatory index (Wang et al., 2022). Across these 13 studies, four showed that lower adherence to the alternative HEI-2010 index (AHEI-2010) (Cutillas-Tolín et al., 2021), lower adherence to the Fertility Diet Score (Chavarro et al., 2007; Noormohammadi et al., 2021), lower adherence to the Mediterranean diet (Wang et al., 2022) and higher adherence to a Western dietary pattern (Badri-Fariman et al., 2021) and high GI and high-fat dietary pattern (Cutillas-Tolín et al., 2021) were associated with an increased likelihood of PCOS. Conversely, a higher adherence to an anti-inflammatory dietary pattern was linked to a lower PCOS risk (Panjeshahin et al., 2020). One study (two publications) (Moran et al., 2013, 2015) showed increased diet quality in women with PCOS, as evidenced by the Mediterranean Diet, dietary glycemic indices, and Dietary Guidelines Index (DGI), while two remaining studies reported comparable HEI-2015 (Lin et al., 2019) and AHEI-2010 (Lin et al., 2021) scores between groups (Table I).

### Secondary outcomes

Few studies reported on food groups and used variable measurement indices (e.g. grams, serving numbers or sizes [broadly defined], component scores of dietary indices), making any pooled analyses impossible. Details of these studies are presented in Table I. Overall, women with PCOS exhibited poorer or comparable intakes of major food groups (grains, fruits, vegetables, proteins, seeds and nuts and dairy). Namely, of seven studies reporting on grain intakes, three (Eslamian et al., 2017; Hosseini et al., 2017; Zirak Sharkesh et al., 2021) showed higher refined grains and/or lower whole grains consumption in PCOS versus Control groups, and three (Lin et al., 2019; Badri-Fariman et al., 2021; Lin et al., 2021; Soodi et al., 2021) showed comparable intakes. Of nine studies that reported on total fruit intake, two reported lower (Pourghassem Gargari et al., 2011; Badri-Fariman et al., 2021) and another higher intakes (Hosseini et al., 2017) in PCOS versus Control groups, whereas the remaining six (Altieri et al., 2013; Shishehgar et al., 2016a; Barrea et al., 2019; Lin et al., 2019; Soodi et al., 2021; Wang et al., 2021a) reported comparable higher intake. Of eight studies reporting on total vegetable intakes, three (Shishehgar et al., 2016a; Badri-Fariman et al., 2021; Wang et al., 2021a) showed lower vegetable intakes, and five (Altieri et al., 2013; Hosseini et al., 2017; Barrea et al., 2019; Copp et al., 2020; Zirak Sharkesh et al., 2021) showed similar intakes. Of nine studies reporting on protein food group intake, five showed lower seafood and/or fish intake (Hosseini et al., 2017; Barrea et al., 2019; Badri-Fariman et al., 2021), lower plant protein intake (pulses and/or legumes) (Shishehgar et al., 2016a; Hosseini et al., 2017; Barrea et al., 2019) or increased animal protein intake (Misir

0.00		1223	PCOS	1221100		Control		100000		
Author and Year	Total	Mean	SD	Total	Mean	SD	Standardized Mean Differ	ence SMD	95%-CI	Weight
Copp et al., 2020	222	1445.6	1423.4	6948	1410.3	1361.9	1 ÷	0.03	[-0.11; 0.16]	6.9%
Eslamian et al., 2017	281	48.6	5.1	472	59.8	7.5	-	-1.67	[-1.84; -1.50]	6.8%
Hosseini et al. 2017	99	59.0	42.0	198	56.0	38.0		0.08	[-0.17; 0.32]	6.7%
Lin et al., 2019	48	1294.9	763.0	34	1279.0	818.6	÷	0.02		6.2%
Moran et al., 2013	409	814.0	875.0	7057	820.0	895.0		-0.01	[-0.11; 0.09]	6.9%
Noormohammadi et al., 2021	303	47.3	5.9	588	58.1	7.1		-1.61	[-1.76; -1.45]	6.9%
Panjeshahin et al., 2020	108	987.0	201.2	108	1426.0	760.7		-0.79	[-1.06; -0.51]	6.6%
Sedighi et al., 2014	65	809.9	629.2	65	1916.8	1708.9		-0.85	[-1.21; -0.50]	6.4%
Shahdadian et al., 2019	225	787.1	797.4	345	1829.4	1870.1		-0.68	[-0.85; -0.50]	6.8%
Shahrokhi et al., 2020	60	717.2	491.8	90	606.1	346.1	· + · · ·	0.27	[-0.06; 0.60]	6.5%
Shishehgar et al., 2016	142	548.0	633.3	140	539.0	723.1		0.01	[-0.22; 0.25]	6.7%
Shishehgar et al., 2019	28	167.5	105.8	34	147.9	106.1		0.18	[-0.32; 0.68]	5.9%
Tay et al., 2020	796	899.0	1165.2	7051	999.0	1183.7	-+		[-0.16; -0.01]	7.0%
Wang et al. 2022	202	8.0	11.2	325	9.0	11.1	1	-0.09	[-0.27; 0.09]	6.8%
Zirak Sharkesh et al. 2021	203	1639.0	573.0	291	1996.7	1258.0		-0.35	[-0.53; -0.17]	6.8%
<b>Overall effect</b> Heterogeneity: $I^2 = 98\%$ , $\tau^2 =$	<b>3191</b> 0.3733	$\gamma^2 = 68$		<b>23746</b>				-0.38	[-0.72; -0.03]	100.0%
Test for overall effect: $t_{14} = -2$							-15 -1 -05 0 05 1	1.5		
							Favors Control Favors PC			



# Α

Author and Year	Total		PCOS SD	Total	Mean	ontrol SD	Standardized Mean Difference	e SMD	95%-CI	Weight
Alipour et al., 2019	45	301.4	98.2	45	269.6	57.8	<u> </u>	0.39	[-0.03; 0.81]	3.5%
Altieri et al., 2013	100	270.8	71.1	100	262.2	67.2	<u></u>	0.12	[-0.15; 0.40]	3.6%
Álvarez-Blasco et al., 2011	22	281.0	102.0	59	275.0	97.0	-	0.06	[-0.43; 0.55]	3.4%
Barrea et al., 2019	112	308.0	42.0		310.5		i i i i i i i i i i i i i i i i i i i		[-0.32; 0.20]	3.6%
Colombo et al., 2009			116.6		256.6		1		[-0.25; 1.69]	2.7%
Cunha et al., 2019		203.5			182.9				[-0.21; 0.71]	3.4%
Cutler et al., 2019	87	46.2		50					[-0.79; -0.08]	3.6%
Douglas et al., 2006		220.3			235.7				[-0.78; 0.26]	3.4%
Eslamian et al., 2017		418.1	39.5		323.5			2.25		3.7%
Ganie et al., 2019 (non-vegetarian sub-		301.9			304.9		÷ —		[-0.36; 0.24]	3.6%
Ganie et al., 2019 (vegetarian sub-coho		318.2			313.8				[-0.20; 0.33]	3.6%
Hart et al., 2016	38	42.0		30					[-1.32; -0.32]	3.4%
Liang et al., 2020 (lean sub-cohort)		184.5			188.5				[-0.95; 0.80]	2.8%
Liang et al., 2020 (lean sub conort)		188.7			222.5			-0.50		2.8%
Lin et al., 2019			106.8		273.0		and a second sec		[-0.44; 0.30]	3.5%
Lin et al., 2021			108.2		262.5		<u> </u>		[-0.34; 0.30]	3.6%
		216.7			249.4				[-0.83; -0.14]	3.6%
Melekoglu et al., 2020 Misir et al., 2016			100.2		162.1	79.3			[-0.25; 1.28]	3.0%
Misir et al., 2016	409						T min		[-0.06; 0.14]	3.7%
Moran et al., 2013		40.5					indiana			3.6%
Navarro-Lafuente et al., 2022		173.4			176.3		I and		[-0.32; 0.16]	
Noormohammadi et al., 2021			121.0		277.0				[1.00; 1.30]	3.7%
Pourghassem Gargari et al., 2011 and 2		171.6			222.6		<u> </u>		[-3.95; -2.40]	3.0%
Shahdadian et al., 2019		353.2			339.8				[0.07; 0.41]	3.7%
Shishehgar et al., 2016		344.3			355.3				[-0.37; 0.10]	3.7%
Shishehgar et al., 2019		307.5			322.6		- <u></u>		[-0.85; 0.15]	3.4%
Soodi et al., 2021		344.1			326.1				[0.01; 0.37]	3.7%
Tsai et al., 2013		191.0			222.0		<u>=1</u>		[-1.01; -0.33]	3.6%
Wright et al., 2004		205.1			211.2		Ŧ		[-0.39; 0.22]	3.6%
Zaeemzadeh et al., 2018	151	342.6	87.5	31	234.2	55.1	-	1.30	[0.89; 1.71]	3.5%
Overall effect Heterogeneity: $l^2 = 97\%$ , $\tau^2 = 0.6432$ , $\chi^2_2$ Test for overall effect: $t_{28} = 0.12$ ( $p = 0.9$	<b>2864</b> <sub>8</sub> = 832.32 (p < 1)	0.001)	)	10844			-2 0 2 Favors Control Favors PCOS	0.02	[-0.30; 0.34]	100.0%
В										
	PC	cos		Co	ntrol					
Author and Year	fotal Mean	SD	Total	Mean	SD		Mean Difference	MD	95%-CI	Weigh
Alipour et al., 2019	45 62.6	30.4	45	62.9	23.6		- <u>-</u>	0.28 [-	-11.52; 10.96]	24.79
Eslamian et al., 2017	281 140.3	33.5	472	135.7	316			•	[-0.24; 9.44]	27.69
Hart et al., 2016				114.0		10	· · ·		[-45.00; -7.00]	19.99
Pourghassem Gargari et al., 2011	30 33.1	8.7	30	17.5	9.8		1	5.61 [	10.92; 20.30]	27.79
Overall effect Heterogeneity: $I^2 = 89\%$ , $\tau^2 = 221.15$ Test for overall effect: $t_3 = 0.04$ (p =	<b>394</b> 549, $\chi_3^2 = 26.3$ 0.97)	3 (p <	<b>577</b> 0.001)			-40 Favors	-20 0 20 40 s Control Favors PCOS	0.34 [-	26.16; 26.84]	100.0%
						Favor	s Control Favors PCOS			

**Figure 4.** Forest plots for carbohydrate intake in women with and without PCOS. (A) Total carbohydrate intake. (B) Added sugar expressed in g/day. MD, mean difference; SMD, standardized mean difference.

et al., 2016) or red, organ, and processed meat (Badri-Fariman et al., 2021), whereas three (Lin et al., 2019, 2021; Soodi et al., 2021) showed comparable intakes of animal or plant proteins. Of three studies reporting on nuts and seeds intakes, two showed a lower proportion of PCOS cohorts who consumed mixed nuts (Badri-Fariman et al., 2021) or tree nuts (Barrea et al., 2019), while another showed similar scores for this food group (Lin et al., 2019). Regarding dairy consumption, eight studies were available, of which three (Pourghassem Gargari et al., 2011; Hosseini et al., 2017; Badri-Fariman

et *al.*, 2021) showed lower dairy (total, low fat, whole fat, fermented, processed milk, and/or yogurt, cheese and kefir) intake in PCOS versus Controls, and five showed comparable intakes (Altieri *et al.*, 2013; Rajaeieh *et al.*, 2014; 2018; Lin *et al.*, 2019, 2021; Soodi *et al.*, 2021).

Further, studies also reported on a wide array of other foods and nutrients (e.g. chocolate, coffee, caffeine, tea, wine, beer, ice cream, eggs, sweets with a high GI, vitamins, minerals essential/unsaturated fats or their ratio). These studies reported either lower, higher, or similar intakes between groups, making any conclusions challenging (Table I) (Álvarez-Blasco et al., 2011; Altieri et al., 2013; Larsson et al., 2016; Hosseini et al., 2017; Thara and Divakar, 2017; Zaeemzadeh et al., 2018; Barrea et al., 2019; Cutler et al., 2019; Tay et al., 2020; Lin et al., 2021; Neubronner et al., 2021; Noormohammadi et al., 2021). Likewise, PA characteristics measured by various indices (e.g. percentage of women active/sedentary, step counts and sport, work, or free time) showed poorer, similar or more favorable PA levels in women with PCOS (Álvarez-Blasco et al., 2011; Banting et al., 2014; Huijgen et al., 2015; Misir et al., 2016; Barrea et al., 2019; Cutler et al., 2019; Wang et al., 2022) (Table I).

### **Meta-analyses**

### Primary outcomes

**Total energy intake.** PCOS groups demonstrated comparable total energy intake versus Controls (MD: 101.01, 95% CI: -4.10 to 206.13 kcal/day; P=0.06; Fig. 2; N=35) (Wright et al., 2004a; Douglas et al., 2006; Colombo et al., 2009; Álvarez-Blasco et al., 2011; Pourghassem Gargari et al., 2011; Altieri et al., 2013; Moran et al., 2013; Tsai et al., 2013; Pourghassem Gargari et al., 2015; Hart et al., 2016; Larsson et al., 2016; Shishehgar et al., 2016b; Eslamian et al., 2017; Hosseini et al., 2017; Zaeemzadeh et al., 2018; Alipour et al., 2019; Barrea et al., 2019; Cunha et al., 2019; Ganie et al., 2019; Lin et al., 2020; Panjeshahin et al., 2020; Shahrokhi and Naeini, 2020; Liang et al., 2021; Lin et al., 2021; Lu et al., 2021; Noormohammadi et al., 2021; Soodi et al., 2021; Wang et al., 2021a; Navarro-Lafuente et al., 2022; Wang et al., 2022). Studies were highly heterogeneous ( $I^2 = 95\%$ ; P < 0.001).

Subgroup analyses based on age, BMI, PCOS criteria, dietary assessment tool or country did not explain heterogeneity, except studies that used the food frequency questionnaire (FFQ), wherein women with PCOS showed higher energy intakes (MD: 278.06, 95% CI: 60.72 to 495.40 kcal/day; P = 0.02) and in studies that were conducted in Iran (MD: -297.75; 95% CI: 17.28 to 578.22 kcal/day; P < 0.01; Fig. 2 Supplementary Table SIV). Of note, subgroup analyses were not possible for certain a priori confounders (e.g. self-reported history of PCOS, questionnaire for the assessment of diet quality, or other countries) and, therefore, are not shown in Supplementary Table SIV. Sensitivity analyses showed that excluding two studies (Pourghassem Gargari et al., 2011, 2015; Liang et al., 2021) from the overall effect estimate resulted in significant differences between the groups (all  $P \le 0.04$ ), without changing the direction of effect estimate: ((overweight subgroup in Liang et al. (2021) (MD: 109.77, 95% CI: 3.40 to 216.12 kcal/ day) and (Pourghassem Gargari et al., 2011, 2015) (MD: 116.64, 95% CI: -12.82 to 220.45 kcal/day)). We observed no evidence of publication bias (funnel plot, Supplementary Fig. S1; P = 0.15, Begg's test; P = 0.79, Egger's test).

**Total PA.** Pooling data for total PA across 15 studies (Moran *et al.*, 2013; Sedighi *et al.*, 2014; Shishehgar *et al.*, 2016b; Eslamian *et al.*, 2017; Hosseini *et al.*, 2017; Lin *et al.*, 2019; Shahdadian *et al.*, 2019; Shishehgar *et al.*, 2019; Copp *et al.*, 2020; Panjeshahin *et al.*, 2020; Shahrokhi and Naeini, 2020; Tay *et al.*, 2020; Noormohammadi *et al.*, 2021; Zirak Sharkesh *et al.*, 2021; Wang *et al.*, 2022) showed comparable levels between groups (SMD: -0.38; 95% CI: -0.72 to -0.03; P=0.03; Fig. 3). Studies were highly heterogeneous  $l^2 = 98\%$ ;  $P \le 0.001$ ). Four studies (Eslamian *et al.*, 2017; Hosseini *et al.*, 2017;

Noormohammadi et al., 2021; Wang et al., 2022) reported similar PA in metabolic equivalent (MET)-hour/week (MD: -5.88, 95% Cl: -16.69 to 4.92; P = 0.18; Supplementary Fig. S2A), and the remaining II (Moran et al., 2013; Sedighi et al., 2014; Shishehgar et al., 2016b; Lin et al., 2019; Shahdadian et al., 2019; Shishehgar et al., 2019; Copp et al., 2020; Panjeshahin et al., 2020; Shahrokhi and Naeini, 2020; Tay et al., 2020; Zirak Sharkesh et al., 2021) reported comparable PA in MET-min/week (MD: -241.89, 95% Cl: -524.90 to 41.10; P = 0.09; Supplementary Fig. S2B).

Subgroup analyses based on age, BMI, or country did not explain heterogeneity; however, PA was lower in PCOS versus Controls in analyses of subgroups that used Rotterdam criteria (SMD: -0.79; 95% CI: -1.45 to -0.14; P=0.03; Supplementary Table SIV). Sensitivity analyses for SMD findings on PA showed that excluding certain studies resulted in no significant differences between groups: (Eslamian *et al.*, 2017) (SMD: -0.28; 95% CI: 0.59 to 0.02); (Noormohammadi *et al.*, 2021) (SMD: -0.29; 95% CI: -0.60 to 0.02); (Panjeshahin *et al.*, 2020) (SMD: -0.35; 95% CI: -0.71 to 0.02); (Sedighi *et al.*, 2014) (SMD: -0.35; 95% CI: -0.71 to 0.02); (Shahdadian *et al.*, 2019) (SMD: -0.35; 95% CI: -0.72 to 0.02; All P=0.02), albeit the direction of effect estimates was consistent with a lower PA in the PCOS versus Control group. We observed no evidence of publication bias (funnel plot, Supplementary Fig. S3; P=0.66, Begg's test; P=0.47, Egger's test).

#### Secondary outcomes

**Total carbohydrate and added sugar.** Meta-analysis assessing total carbohydrate intake revealed comparable intakes in women with PCOS versus Controls (SMD: 0.02; 95% Cl: -0.30 to 0.34; P = 0.91; Fig. 4; N = 27) (Wright *et al.*, 2004a; Douglas *et al.*, 2006; Colombo *et al.*, 2009; Álvarez-Blasco *et al.*, 2011; Pourghassem Gargari *et al.*, 2013; Moran *et al.*, 2013; Tsai *et al.*, 2013; Pourghassem Gargari *et al.*, 2015; Hart *et al.*, 2016; Misir *et al.*, 2016; Shishehgar *et al.*, 2016; Eslamian *et al.*, 2017; Zaeemzadeh *et al.*, 2018; Alipour *et al.*, 2019; Barrea *et al.*, 2019; Cunha *et al.*, 2019; Cutler *et al.*, 2019; Ganie *et al.*, 2019; Lin *et al.*, 2020; Liang *et al.*, 2021; Lin *et al.*, 2021; Noormohammadi *et al.*, 2021; Soodi *et al.*, 2021; Navarro-Lafuente *et al.*, 2022).

Carbohydrate intakes were comparable in studies that reported in g/day (MD: 11.85, 95% CI: -6.09 to 29.79; P = 0.19; Supplementary Fig. S4A; N = 24) (Wright et al., 2004a; Douglas et al., 2006; Colombo et al., 2009; Álvarez-Blasco et al., 2011; Pourghassem Gargari et al., 2011; Altieri et al., 2013; Tsai et al., 2013; Pourghassem Gargari et al., 2015; Misir et al., 2016; Shishehgar et al., 2016b; Eslamian et al., 2017; Zaeemzadeh et al., 2018; Alipour et al., 2019; Barrea et al., 2019; Cunha et al., 2019; Ganie et al., 2019; Lin et al., 2019; Shahdadian et al., 2019; Shishehgar et al., 2019; Melekoglu et al., 2020; Liang et al., 2021; Lin et al., 2021; Noormohammadi et al., 2021; Soodi et al., 2021; Navarro-Lafuente et al., 2022) or % energy intake/day (MD: -2.28, 95% Cl: -8.82 to 4.27; P=0.27; Supplementary Fig. S4B; N = 3) (Moran et al., 2013; Hart et al., 2016; Cutler et al., 2019), respectively. Added sugar intakes were similar between groups (MD: 0.34, 95% CI: -26.16 to 26.84 g/day; P = 0.97; Fig. 4B; N=4) (Pourghassem Gargari et al., 2011; Hart et al., 2016; Eslamian et al., 2017; Alipour et al., 2019). High heterogeneity was

•		P	cos		Co	ntrol					
Author and Year	Total	Mean	SD	Total	Mean	SD	Standardized Mean Difference	SMD	95%	-CI	Weigh
Alipour et al., 2019	45	58.2	18.5	45	57.3	12.3	*	0.05	[-0.36; 0	).47]	3.39
Altieri et al., 2013	100	89.7	22.6	100	94.1	21.4		-0.20	[-0.48; 0	[80.0	3.49
Álvarez-Blasco et al., 2011	22	95.0	25.0	59	99.0	35.0		-0.12	[-0.61; 0	0.37]	3.19
Barrea et al., 2019	112	73.9	13.6	112	70.1	10.7	-	0.32	[ 0.05; 0	0.58]	3.49
Colombo et al., 2009	8	91.7	22.5	10	71.7	26.1		0.77	[-0.20; 1	.75]	2.39
Cunha et al., 2019	39	57.1	25.3	34	55.5	29.8	-	0.06	[-0.40: 0	0.521	3.29
Cutler et al., 2019	87	36.0	5.0	50	34.0	6.0	line.	0.37	[ 0.02; 0	0.721	3.39
Douglas et al., 2006	30	69.2	25.0	27	61.5	21.1			[-0.20; 0		3.19
Eslamian et al., 2017	281			472	83.0			1.89	[ 1.72: 2		3.59
Ganie et al., 2019 (non-vegetarian sub-cohort)	62	49.1	12.3	141	46.1	9.5			[-0.02; 0		3.49
Ganie et al., 2019 (vegetarian sub-cohort)	82	43.1		179	42.0				[-0.20; 0		3.49
Hart et al., 2016	38	39.0	6.0	30	35.0	5.0	T-m-	0.71	[0.21: 1		3.19
arsson et al., 2016	51	31.0	_	29	33.0				[-0.77: 0		3.29
Liang et al., 2020 (lean sub-cohort)	10		20.3	10					[-1.30; 0		2.59
Liang et al., 2021 (overweight sub-cohort)	10		18.3	10					[-1.16; 0		2.59
Lin et al., 2019	80		42.0	44	83.0		-		[-0.22; 0		3.39
Lin et al., 2021	40		43.2	529	94.2		-		[-0.34; 0		3.49
Melekoglu et al., 2020	65	69.6		65	67.3		~		[-0.24; 0		3.49
Misir et al., 2016	12	86.4		16	81.9				[-0.66; 0		2.79
Moran et al., 2013	409	36.6		7057		4.9	- F		[-0.18; 0		3.69
Navarro-Lafuente et al., 2022	121		10.4	155	70.2		The second se		[-0.08; 0		3.59
Noormohammadi et al., 2021		111.0		588	76.0		Гв		[ 1.06; 1		3.59
Pourghassem Gargari et al., 2021	30	50.7		30	65.7				[-3.84; -2		2.79
Shahdadian et al., 2019	225	85.2		345	79.3		100		[ 0.09; 0		3.59
Shishehgar et al., 2016	142		30.8	140			and the second s		[-0.17; 0		3.59
Shishehgar et al., 2019	28	31.7		34			Line.		[-0.14; 0		3.19
Soodi et al., 2021	203	92.5		291	87.0		Take		[-0.02; 0		3.59
Tsai et al., 2021	203		17.0	161	51.0		line .		[-0.17; 0		3.49
Wang et al. 2022		30.6					E				
	202			325	27.5		2		[0.05; 0		3.59
Wright et al., 2004	84	75.5		79	73.8		- 1		[-0.26; 0		3.49
Zaeemzadeh et al., 2018	151	69.6	38.5	31	103.4	36.6	-	-0.88	[-1.28; -0	).48J	3.39
<b>Overall effect</b> Heterogeneity: $l^2$ = 96%, $\tau^2$ = 0.4438, $\chi^2_{30}$ = 675	3117			11198			A 1	0.11	[-0.16; 0	.37]	100.09

# В

		P	cos		Co	ntrol					
Author and Year	Total	Mean	SD	Total	Mean	SD	Standardized Mean Difference	e SMD	95%	-CI	Weight
Altieri et al., 2013	100	30.1	10.0	100	31.9	10.3	10	-0.18	[-0.45; 0	.10]	5.2%
Álvarez-Blasco et al., 2011	22	28.0	7.0	59	30.0	12.0	*	-0.18	[-0.67; 0	.31]	5.0%
Barrea et al., 2019	112	24.6	7.5	112	17.4	10.7	+	0.77	[ 0.50; 1	.04]	5.2%
Colombo et al., 2009	8	30.8	8.9	10	21.4	9.9	100	0.94	[-0.05; 1	.94]	4.3%
Cunha et al., 2019	39	16.1	7.8	34	17.3	10.3	*	-0.13	[-0.59; 0	.33]	5.0%
Douglas et al., 2006	30	24.3	10.7	27	21.5	8.4	<u></u>	0.29	[-0.24; 0	.81]	5.0%
Eslamian et al., 2017	281	12.3	5.6	472	9.1	3.9	10	0.69	[ 0.54; 0	.85]	5.2%
Hart et al., 2016	38	13.7	3.0	30	11.6	3.2	-	0.67	[0.18; 1	.16]	5.0%
Larsson et al., 2016	51	12.0	3.0	29	12.0	3.0	*	0.00	[-0.46; 0	.46]	5.0%
Lin et al., 2019	80	29.0	15.6	44	28.0	16.5	<u>iii</u>	0.06	[-0.31; 0	.43]	5.1%
Lin et al., 2021	40	35.3	18.0	529	35.4	17.7	<u></u>	-0.01	[-0.33; 0	.32]	5.1%
Melekoglu et al., 2020	65	10.8	3.0	65	8.7	2.3	55	0.78	[0.42; 1	.14]	5.1%
Misir et al., 2016	12	28.0	20.4	16	22.9	17.3		0.26	[-0.49; 1	.02]	4.7%
Moran et al., 2013	409	15.1	3.1	7057	15.4	3.1	i di	-0.10	[-0.20; 0	.00]	5.2%
Navarro-Lafuente et al., 2022	121	21.4	5.0	155	20.5	5.7		0.17	[-0.07; 0	.40]	5.2%
Pourghassem Gargari et al., 2011	30	23.6	1.0	30	32.3	1.9		-5.62	[-6.78; -4	.47]	4.1%
Shishehgar et al., 2016	142	26.6	9.9	140	27.3	8.3	<b>D</b>	-0.08	[-0.31; 0	.16]	5.2%
Wright et al., 2004	84	23.4	12.7	79	23.0	9.7	<u></u>	0.03	[-0.27; 0	.34]	5.1%
Zaeemzadeh et al., 2018	151	20.5	9.1	31	25.2	11.6	100	-0.49	[-0.88; -0	.10]	5.1%
Zirak Sharkesh et al. 2021	203	26.5	12.5	291	26.8	11.4	<u>.</u>	-0.02	[-0.20; 0	.16]	5.2%
Overall effect	2018			9310			4	-0.06	[-0.64; 0	.52]	100.0%
Heterogeneity: $l^2 = 92\%$ , $\tau^2 = 1.23$	570, χ <sup>2</sup>	= 233.9	90 (p ·	< 0.001	)				0.5000000000000000000000000000000000000		
Test for overall effect: $t_{19} = -0.21$ (	p = 0.8	3)					-6 -4 -2 0 2 4 6				
							Favors Control Favors PCOS				

# С

		Р	cos		Co	ntrol				
Author and Year	Total	Mean	SD	Total	Mean	SD	Standardized Mean Difference	SMD	95%-CI	Weight
Álvarez-Blasco et al., 2011	22	43.0	14.0	59	46.0	18.0		-0.17	[-0.66; 0.32]	5.6%
Barrea et al., 2019	112	38.2	4.6	112	43.7	5.9		-1.04	[-1.32; -0.76]	6.4%
Colombo et al., 2009	8	43.6	10.7	10	34.9	13.4		0.67	[-0.29; 1.64]	3.6%
Douglas et al., 2006	30	26.7	10.1	27	23.2	9.0	+	0.36	[-0.16; 0.88]	5.4%
Hart et al., 2016	38	13.2	2.2	30	11.5	2.3		0.75	[0.25; 1.24]	5.6%
Lin et al., 2019	80	35.0	17.8	44	32.0	13.2		0.18	[-0.19; 0.55]	6.1%
Lin et al., 2021	40	34.6	16.8	529	34.5	17.3		0.01	[-0.32; 0.33]	6.2%
Melekoglu et al., 2020	65	12.8	3.4	65	12.1	3.3		0.21	[-0.14; 0.55]	6.2%
Misir et al., 2016	12	28.7	22.3	16	30.8	28.4		-0.08	[-0.83; 0.67]	4.5%
Moran et al., 2013	409	13.1	2.3	7057	13.1	2.1	ė.	0.00	[-0.10; 0.10]	6.8%
Navarro-Lafuente et al., 2022	121	32.7	6.7	155	32.4	7.0	*	0.04	[-0.19; 0.28]	6.5%
Pourghassem Gargari et al., 2011	30	16.1	0.9	30	19.3	2.2	- <u></u>	-1.83	[-2.44; -1.22]	5.1%
Shishehgar et al., 2016	142	27.5	10.2	140	26.0	9.4		0.15	[-0.09; 0.38]	6.5%
Wang et al. 2022	202	12.3	7.6	325	10.9	6.1	-	0.21	[0.04; 0.39]	6.7%
Wright et al., 2004	84	28.1	14.4	79	27.3	10.9		0.06	[-0.24; 0.37]	6.3%
Zaeemzadeh et al., 2018	151	24.8	10.2	31	30.9	12.6		-0.57	[-0.96; -0.18]	6.0%
Zirak Sharkesh et al. 2021	203	32.5	14.1	291	30.5	12.3	1	0.15	[-0.03; 0.33]	6.7%
Overall effect	1749			9000			4	-0.06	[-0.36; 0.24]	100.0%
Heterogeneity: $I^2 = 87\%$ , $\tau^2 = 0.27$	711, x1F	= 121.5	50 (p ·	< 0.001	)				850 E 5	
Test for overall effect: $t_{16} = -0.39$	(p = 0.7)	70)					-2 -1 0 1 2			
							Favors Control Favors PCOS			

**Figure 5.** Forest plots for fat intake in women with and without PCOS. (**A**, total fat; **B**, saturated fatty acids [SFA]; **C**, monounsaturated fatty acids [MUFA]; **D**, polyunsaturated fatty acids [PUFA]; **E**, cholesterol) with cholesterol expressed as mg/day. SMD, standard mean difference.

		P	COS		Con	trol				
Author and Year	Total	Mean	SD	Total	Mean	SD	Standardized Mean Diff	ference SMD	95%-CI	Weig
Álvarez-Blasco et al., 2011	22	16.0	5.0	59	16.0	9.0		0.00	[-0.49; 0.49]	5.6
Barrea et al., 2019	112	11.2	6.9	112	9.0	4.7		0.37	[0.10; 0.63]	6.4
Colombo et al., 2009	8	12.0	3.6	10	11.0	7.7		0.15	[-0.78; 1.08]	3.8
Douglas et al., 2006	30	12.5	3.8	27	11.7	4.6			[-0.33; 0.71]	5.5
Hart et al., 2016	38	8.2	5.7	30	6.5	1.7	1000		[-0.10; 0.86]	5.6
Lin et al., 2019	80		8.9	44	17.0	6.6	1		[-0.25; 0.49]	6.1
Lin et al., 2021	40	16.6	7.3	529	17.5	9.0			[-0.42; 0.22]	6.2
	65		3.8	65						
Melekoglu et al., 2020					9.5	2.7	1		[-0.19; 0.50]	6.2
Misir et al., 2016	12		9.6	16	22.2 1				[-1.02; 0.48]	4.5
Moran et al., 2013	409	5.2		7057		1.6			[-0.04; 0.16]	6.8
Navarro-Lafuente et al., 2022			3.6	155	12.3	2.9	<u></u>		[-0.05; 0.42]	6.5
Pourghassem Gargari et al., 2	2011 30	11.1	0.8	30	14.1	1.3		-2.67	[-3.38; -1.96]	4.7
Shishehgar et al., 2016	142	20.4	9.7	140	18.9	8.4		0.16	[-0.07; 0.40]	6.5
Wang et al. 2022	202	4.8	2.2	325	4.5	2.3		0.10	[-0.08; 0.27]	6.6
Wright et al., 2004	84	18.2	10.3	79	18.0	8.4	-	0.02	[-0.28; 0.33]	6.3
Zaeemzadeh et al., 2018	151		7.6	31	18.8	8.3			[-0.79; -0.01]	6.0
Zirak Sharkesh et al. 2021	203		8.0	291	21.2 1		175		[-0.52; -0.16]	6.6
<b>Overall effect</b> Heterogeneity: $l^2 = 83\%$ , $\tau^2 =$ Test for overall effect: $t_{16} = -0$	<b>1749</b> 0.2904, χ <sup>2</sup> 0.51 ( <i>p</i> = 0.0	= 91.63	(p <	9000 0.001)			-3 -2 -1 0 1 Favors Control Favors	2 3	[-0.40; 0.25]	100.0
Heterogeneity: $l^2 = 83\%$ , $\tau^2 =$ Test for overall effect: $t_{16} = -0$	$0.2904, \chi^2_1$	= 91.63	(p <					2 3	[-0.40; 0.25]	100.0
Heterogeneity: $l^2 = 83\%$ , $\tau^2 =$ Test for overall effect: $t_{16} = -0$	$0.2904, \chi^2_1$	91.63 2)	5		Contro an SI			2 3	[-0.40; 0.25] 95%-CI	
Heterogeneity: / <sup>2</sup> = 83%, r <sup>2</sup> = Test for overall effect: t <sub>16</sub> = -0	0.2904, $\chi^2_{11}$ 0.51 ( $p$ = 0.0	91.63 2) PCOS	S ) Tot	0.001) al Mea	an SI	D	Favors Control Favors	2 3 PCOS	95%-CI	Weig
Heterogeneity: $l^2 = 83\%$ , $t^2 = Test for overall effect: t_{16} = -0Author and YearAltieri et al., 2013$	0.2904, $\chi_1^2$ 5.51 ( $p$ = 0.0 Total Me 100 253	= 91.63 32) PCOS an SI 6.0 87.1	<b>5</b> <b>7 Tot</b>	0.001) al Mea	an SI	<b>0</b>	Favors Control Favors	2 3 PCOS MD -12.00	<b>95%-Ci</b> [-34.51; 10.51]	<b>Weig</b> 13.1
Heterogeneity: $t^2 = 83\%$ , $t^2 = 1$ Test for overall effect: $t_{16} = -0$ Author and Year Altieri et al., 2013 Álvarez-Blasco et al., 2011	0.2904, $\chi_1^2$ 5.51 ( $p$ = 0.0 <b>Total Me</b> 100 253 22 329	PCOS an SI 0.0 87.1 0.0 89.1	5 7 Tot 0 10 0 5	al Mea	an SI .0 75.0 .0 128.0	0	Favors Control Favors	2 3 PCOS MD -12.00 -1.00	<b>95%-Ci</b> [-34.51; 10.51] [-50.50; 48.50]	Weig 13.1 4.1
Heterogeneity: $l^2 = 83\%$ , $t^2 = 7$ Test for overall effect: $t_{16} = -0$ Author and Year Altieri et al., 2013 Álvarez-Blasco et al., 2011 Colombo et al., 2009	0.2904, $\chi_1^2$ 5.51 ( $p$ = 0.0 <b>Total Me</b> 100 253 22 329 8 347	PCOS an SI 0.0 87.1 0.0 89.1 5 108.1	5 <b>) Tot</b> 0 10 0 5 7 1	al Mea 00 265 59 330 10 291	an SI .0 75.0 .0 128.0 .6 115.	0	Favors Control Favors	2 3 PCOS MD -12.00 -1.00 55.90 [	<b>95%-CI</b> [-34.51; 10.51] -47.84; 159.64]	Weig 13.1 4.1 1.1
Heterogeneity: $l^2 = 83\%$ , $t^2 = 7$ Test for overall effect: $t_{16} = -0$ Author and Year Altieri et al., 2013 Álvarez-Blasco et al., 2011 Colombo et al., 2019 Cunha et al., 2019	0.2904, χ <sup>2</sup> <sub>1</sub> .51 (ρ = 0.1 <b>Total Me</b> 100 253 22 325 8 347 39 195	PCOS an SI 0.0 87.1 0.0 89.1 5 108.1 5 137.1	5 ) Tot 0 10 0 5 7 1 3 3	al Mea 00 265 59 330 10 291 34 209	an SI 0.0 75.0 0.0 128.0 0.6 115.1 0.5 153.0	0 0 1 6	Favors Control Favors	MD -12.00 -1.00 55.90 [ -9.96	<b>95%-Cl</b> [-34.51; 10.51] [-50.50; 48.50] -47.84; 159.64] [-77.21; 57.29]	Weig 13.1 4.1 1.1 2.4
Heterogeneity: $t^2 = 83\%$ , $t^2 = 7$ Test for overall effect: $t_{16} = -0$ Author and Year Altieri et al., 2013 Álvarez-Blacco et al., 2011 Colombo et al., 2009 Cunha et al., 2019 Cutler et al., 2019	0.2904, $\chi_1^2$ .51 ( $\rho$ = 0.0 <b>Total Me</b> 100 253 22 326 8 347 39 199 87 284	PCOS an SI 0.0 87.1 0.0 89.1 5 108.3 5 137.3 1 185.3	5 5 5 5 7 1 3 3 5 5 5	al Mea 00 265 59 330 10 291 34 209 50 260	an SI 0.0 75.0 0.0 128.0 0.6 115.1 0.5 153.0 0.8 144.1	0 0 1 6 1	Favors Control Favors	MD -12.00 -1.00 55.90 [ -9.96 23.30	<b>95%-Cl</b> [-34.51; 10.51] [-50.50; 48.50] -47.84; 159.64] [-77.21; 57.29] -32.52; 79.12]	Weig 13.1 4.1 1.1 2.4 3.3
Heterogeneity: $l^2 = 83\%$ , $t^2 = 7$ Test for overall effect: $t_{16} = -0$ Author and Year Altieri et al., 2013 Aivarez-Blasco et al., 2011 Colombo et al., 2009 Cunha et al., 2019 Cutler et al., 2019 Douglas et al., 2006	0.2904, $\chi_1^2$ .51 ( $p$ = 0.0 <b>Total Me</b> 100 255 22 325 8 347 39 198 87 284 30 245	PCOS an SI 5 108. <sup>1</sup> 5 108. <sup>1</sup> 5 137. <sup>1</sup> 1 185. <sup>3</sup> 6 113. <sup>1</sup>	<b>3</b> <b>7</b> <b>1</b> <b>3</b> <b>3</b> <b>5</b> <b>5</b> <b>5</b> <b>2</b> <b>2</b>	al Mea 00 265 59 330 10 291 34 209 50 260 27 245	an SI .0 75.0 .0 128.0 .6 115.1 .5 153.0 .8 144.1 .9 132.1	0 0 1 6 1 3	Favors Control Favors	MD -12.00 -1.00 -5.90 [ -9.96 23.30 -0.30	<b>95%-CI</b> [-34.51; 10.51] [-50.50; 48.50] [-77.21; 57.29] [-32.52; 79.12] [-64.57; 63.97]	Weig 13.1 4.1 1.1 2.4 3.3 2.6
Heterogeneity: $l^2 = 83\%$ , $t^2 = 7$ Test for overall effect: $t_{16} = -0$ Author and Year Altieri et al., 2013 Álvarez-Blasco et al., 2011 Colombo et al., 2009 Cunha et al., 2019 Cutler et al., 2019 Douglas et al., 2006	0.2904, $\chi_1^2$ .51 ( $p$ = 0.0 <b>Total Me</b> 100 255 22 325 8 347 39 198 87 284 30 245	PCOS an SI 0.0 87.1 0.0 89.1 5 108.3 5 137.3 1 185.3	<b>3</b> <b>7</b> <b>1</b> <b>3</b> <b>3</b> <b>5</b> <b>5</b> <b>5</b> <b>2</b> <b>2</b>	al Mea 00 265 59 330 10 291 34 209 50 260 27 245	an SI 0.0 75.0 0.0 128.0 0.6 115.1 0.5 153.0 0.8 144.1	0 0 1 6 1 3	Favors Control Favors	MD -12.00 -1.00 -5.90 [ -9.96 23.30 -0.30	<b>95%-Cl</b> [-34.51; 10.51] [-50.50; 48.50] -47.84; 159.64] [-77.21; 57.29] -32.52; 79.12]	Weig 13.1 4.1 1.1 2.4 3.3 2.6
Heterogeneity: $t^2 = 83\%$ , $t^2 = 7$ Test for overall effect: $t_{16} = -0$ Author and Year Altieri et al., 2013 Álvarez-Blacco et al., 2011 Colombo et al., 2009 Cunha et al., 2019 Cutler et al., 2019	0.2904, $\chi_1^2$ .51 ( $p$ = 0.0 <b>Total Me</b> 100 253 22 325 8 347 39 196 87 284 30 244 80 303	PCOS an SI 0.0 87.0 0.5 108.1 5.5 108.1 5.5 137.1 0.1 73.0 6 113.1 0.0 173.0 2 178.1	<b>3</b> <b>5 Tot</b> <b>1</b> <b>1</b> <b>1</b> <b>1</b> <b>1</b> <b>1</b> <b>1</b> <b>1</b> <b>1</b> <b>1</b>	al Mea 00 265 59 330 10 291 34 209 50 260 27 245 14 271	an SI .0 75.0 .0 128.0 .6 115.1 .5 153.0 .8 144.1 .9 132.1	0 0 1 6 1 3 3	Favors Control Favors	MD -12.00 -1.00 -5.90 [ -9.96 23.30 -0.30 32.00	<b>95%-CI</b> [-34.51; 10.51] [-50.50; 48.50] [-77.21; 57.29] [-32.52; 79.12] [-64.57; 63.97]	Weig 13.1 4.1 1.2.4 3.3 2.6 3.3 3.1
Heterogeneity: $l^2 = 83\%$ , $t^2 = 1$ Test for overall effect: $t_{16} = -0$ Author and Year Altieri et al., 2013 Álvarez-Blasco et al., 2011 Colombo et al., 2009 Cunha et al., 2019 Douglas et al., 2019 Douglas et al., 2019	0.2904, $\chi_1^2$ .51 ( $p$ = 0.0 <b>Total Me</b> 100 253 22 325 8 347 39 196 87 284 30 244 80 303	PCOS an SI 0.0 87.1 0.0 89.1 5 108.1 5 137.1 1 185.3 6 113.1 0 173.1 2 178.1	<b>3</b> <b>5 Tot</b> <b>1</b> <b>1</b> <b>1</b> <b>1</b> <b>1</b> <b>1</b> <b>1</b> <b>1</b> <b>1</b> <b>1</b>	al Mea 00 265 59 330 10 291 34 209 50 260 27 245 14 271	an SI .0 75.0 .0 128.0 .6 115. .5 153.0 .8 144. .9 132.3 .0 140.3 .2 207.4	0 0 1 6 1 3 3 4	Favors Control Favors	MD -12.00 -12.00 -1.00 55.90 [ -9.96 23.30 -0.30 32.00 3.00	<b>95%-Cl</b> -34.51; 10.51] -50.50; 48.50] -47.84; 159.64] -77.21; 57.29] -32.52; 79.12] -64.57; 63.97] -24.26; 88.26]	Weig 13.1 1.1 2.4 3.3 2.6 3.3 3.1
Heterogeneity: $l^2 = 83\%$ , $t^2 = 7$ Test for overall effect: $t_{16} = -0$ Author and Year Altieri et al., 2013 Álvarez-Blacco et al., 2011 Colombo et al., 2009 Cunha et al., 2019 Outler et al., 2019 Douglas et al., 2019 Lin et al., 2021	0.2904, $\chi^2_1$ .51 ( $\rho$ = 0.1 Total Me 100 253 22 325 8 347 39 196 87 284 30 245 80 303 40 355 165 165	PCOS an SI 0.0 87.0 0.5 108.1 5.5 108.1 0.5 137.1 0.6 113.1 0.0 173.0 0.173.0 2 178.1	<b>5</b> <b>) Tot</b> <b>1</b> <b>1</b> <b>1</b> <b>1</b> <b>1</b> <b>1</b> <b>1</b> <b>1</b> <b>1</b> <b>1</b>	al Mez 00 265 59 330 10 291 34 209 50 260 27 245 14 271 29 354 55 149	an SI .0 75.0 .0 128.0 .6 115. .5 153.0 .8 144. .9 132.3 .0 140.3 .2 207.4	0 0 1 6 1 3 3 4 1	Favors Control Favors	MD -12.00 -1.00 -5.5.90 [ -9.96 23.30 -0.30 32.00 3.00 18.40	<b>95%-CI</b> [-34.51; 10.51] [-50.50; 48.50] -47.84; 159.64] -77.21; 57.29] -32.52; 79.12] [-64.67; 63.97] -24.26; 88.26] -55.16; 61.16]	Weig 13.1 4.1 1.1 2.4 3.3 2.6 3.3 3.1 9.7
Heterogeneity: $l^2 = 83\%$ , $t^2 = 7$ Test for overall effect: $t_{16} = -0$ Author and Year Altieri et al., 2013 Aivarez-Blasco et al., 2011 Colombo et al., 2009 Cunha et al., 2019 Cutler et al., 2019 Dudgas et al., 2019 Lin et al., 2021 Lin et al., 2021 Metekoglu et al., 2020	0.2904, $\chi^2_1$ .51 ( $\rho$ = 0.1 Total Me 100 253 22 325 8 347 39 196 87 284 30 245 80 303 40 355 165 165	PCOS an SI 0 87.1 0 89.1 5 108.1 5 137.1 1 185.3 6 113.1 0 173.1 2 178.1 1 82.1 1 83.1 1 85.1 1 85.1	<b>5</b> <b>) Tot</b> <b>1</b> <b>1</b> <b>1</b> <b>1</b> <b>1</b> <b>1</b> <b>1</b> <b>1</b> <b>1</b> <b>1</b>	al Mea 00 265 59 330 10 291 34 209 50 260 27 245 14 271 29 354 35 149 16 232	an SI .0 75.0 .0 128.0 .6 115.1 .5 153.0 .8 144.1 .9 132.3 .0 140.3 .2 207.4 .7 84.1 .7 211.1	0 0 1 6 1 3 3 4 1 5	Favors Control Favors	MD -12.00 -100 55.90 -9.96 23.30 -0.30 32.00 3.00 18.40 46.01 [-	<b>95%-Cl</b> -34.51; 10.51] -50.50; 48.50] -47.84; 159.64] -77.21; 57.29] -32.52; 79.12] -64.57; 63.97] -24.26; 88.26] -155.16; 61.16] -10.26; 47.06] -10.26; 47.06]	Weig 13.1 4.1 2.4 3.3 3.3 3.1 9.7 0.4
Heterogeneity: $t^2 = 83\%$ , $t^2 = 7$ Test for overall effect: $t_{16} = -0$ Author and Year Altieri et al., 2013 Álvarez-Blasco et al., 2011 Colombo et al., 2009 Curlar et al., 2019 Curlar et al., 2019 Douglas et al., 2009 Lin et al., 2019 Lin et al., 2019 Lin et al., 2019 Melekoglu et al., 2010 Misir et al., 2016	0.2904, $\chi^2_1$ .51 ( $\rho$ = 0.1 <b>Total Me:</b> 100 255 22 325 8 347 39 195 87 284 30 245 80 303 40 357 65 166 12 276 409 277	PCOS an SI .0 87.1 .0 89.1 .5 108. .5 108. .5 108. .5 108. .1 185.3 .6 113.3 .0 173.1 .2 178.3 .1 82.1 .7 249.9 .0 119.1	<b>5</b> <b>7</b> <b>1</b> <b>1</b> <b>1</b> <b>1</b> <b>1</b> <b>1</b> <b>1</b> <b>1</b>	al Mea 00 265 59 330 10 291 34 209 50 260 27 245 14 271 29 354 35 149 16 232 57 265	an SI .0 75.0 .0 128.0 .6 115.1 .5 153.0 .8 144.1 .9 132.3 .0 140.3 .2 207.4 .7 84.1 .7 211.1	0 0 1 6 1 3 3 4 1 5 0	Favors Control Favors	MD -12.00 -100 -5.90 [ -9.96 23.30 -0.30 32.00 3.00 18.40 -0.30 3.00 -0.30	<b>95%-CI</b> [-34.51; 10.51] -50.50; 48.50] -47.84; 159.64] -77.21; 57.29] -64.57; 63.97] -24.26; 88.26] -55.16; 61.16] -10.26; 47.06] 129.32; 221.34] [-6.81; 16.81]	Weig 13.1 1.1 2.4 3.3 2.6 3.3 3.1 9.7 0.4 22.2
Heterogeneity: / <sup>2</sup> = 83%, r <sup>2</sup> = Test for overall effect: t <sub>16</sub> = -0 Author and Year Altieri et al., 2013 Álvarez-Blasco et al., 2011 Colombo et al., 2009 Cunha et al., 2019 Douglas et al., 2009 Lin et al., 2019 Lin et al., 2019 Lin et al., 2019 Lin et al., 2019 Meiekoglu et al., 2020 Misir et al., 2013 Mavaror-Latuente et al., 2022	0.2904, $\chi^2_1$ .51 ( $\rho$ = 0.1 <b>Total Me</b> 100 253 22 326 8 344 39 196 8 344 30 244 8 303 40 357 65 166 12 276 409 270 121 302	PCOS an SI 0. 87.1 0. 89.1 5. 108.1 5. 108.1 5. 137.1 0. 173.1 0. 173.1 0. 173.1 0. 115.1	<b>5</b> <b>7</b> <b>7</b> <b>7</b> <b>7</b> <b>7</b> <b>7</b> <b>7</b> <b>7</b>	al Mea 00 265 59 330 0 291 34 209 00 260 27 245 14 271 29 354 55 149 27 265 55 283	an SI .0 75.0 .0 128.0 .6 115. .5 153.0 .8 144. .9 132.3 .0 140.3 .2 207.4 .7 84. .7 211.5 .0 109.0 .4 124.0	0 0 1 6 1 3 3 4 1 5 0 6	Favors Control Favors	MD -12.00 -1.00 5.900 [ -9.96 23.30 -0.30 -0.30 32.00 3.00 18.40 -46.01 [- 5.00 18.60	<b>95%-CI</b> -34.51; 10.51] -50.50; 48.50] -47.84; 159.64] -32.52; 79.12] -64.57; 63.97] -24.26; 88.26] -55.16; 61.16; 129.32; 221.34] [ -6.81; 16.81] -9.77; 46.97]	Weig 13.1 4.1 1.1 2.4 3.3 3.1 9.7 0.4 22.2 9.8
Heterogeneity: $l^2 = 83\%$ , $t^2 = 7$ Test for overall effect: $t_{16} = -0$ Author and Year Altieri et al., 2013 Álvarez-Blasco et al., 2011 Colombo et al., 2009 Cunha et al., 2019 Cutler et al., 2019 Cutler et al., 2019 Lin et al., 2019 Lin et al., 2019 Lin et al., 2019 Meisre dal., 2010 Moran et al., 2013 Navarro-Lafuente et al., 2022 Wang et al. 2022	0.2904, $\chi^2_1$ .51 ( $\rho$ = 0.1 <b>Total Me:</b> 100 253 22 322 8 347 30 199 87 284 80 303 40 357 60 12 276 12 1302 202 493 202 493 203 493	PCOS an SI 0 87.1 0 89.1 5 108. 5 137.3 1 185.3 6 113.3 0 173.1 2 178.3 1 82.1 7 249.1 0 119.1 0 115.1 0 282.1	<b>5</b> <b>5</b> <b>7</b> <b>1</b> <b>1</b> <b>1</b> <b>1</b> <b>1</b> <b>1</b> <b>1</b> <b>1</b>	al Mez 200 265 200 265 200 260 200 291 24 209 20 260 27 245 149 20 354 20 354 20 354 20 354 20 354 21 20 25 283 25 283 25 413 25 283 25 413 25 283 25 283 25 283 25 283 25 283 26 285 28 285	an SI .0 75.0 .0 128.0 .6 115.1 .5 153.0 .8 144. .9 132.3 .0 140.3 .2 207.4 .7 84.1 .7 211.9 .0 109.0 .4 124.0 .9 210.0	0 0 0 1 6 1 3 3 4 1 5 0 6 6	Favors Control Favors	MD -12.00 -1.00 55.90 [ -9.96 23.30 -0.30 32.00 3.00 18.40 18.60 18.60 79.12	95%-Cl -34.51; 10.51] -50.50; 48.50] -47.84; 159.64] -77.21; 57.29] -32.52; 79.12] -64.57; 63.97] -55.16; 61.16] -10.26; 47.06] -10.26; 47.06] -10.26; 47.06] -10.26; 47.06] -10.26; 47.06] -10.27; 46.97] -9.77; 46.97]	Weig 13.1 4.1 1.1 2.4 3.3 3.3 3.3 3.3 3.3 9.7 0.4 22.2 9.8 4.8
Heterogeneity: / <sup>2</sup> = 83%, r <sup>2</sup> = Test for overall effect: t <sub>16</sub> = -0 Author and Year Altieri et al., 2013 Álvarez-Blasco et al., 2011 Colombo et al., 2009 Cunha et al., 2019 Douglas et al., 2009 Lin et al., 2019 Lin et al., 2019 Lin et al., 2019 Lin et al., 2019 Meiekoglu et al., 2020 Misir et al., 2013 Mavaror-Latuente et al., 2022	0.2904, $\chi^2_1$ .51 ( $\rho$ = 0.1 <b>Total Me:</b> 100 253 22 322 8 347 30 199 87 284 80 303 40 357 60 12 276 12 1302 202 493 202 493 203 493	PCOS an SI 0 87.1 0 89.1 5 108.1 5 108.1 1 185.1 6 113.1 0 173.1 1 185.1 6 113.1 0 173.1 1 185.1 2 178.1 1 19.1 0 178.1 0 119.1 0 128.2 0 119.1 0 128.2 1 0 128.1 1 19.1 19.1 19.1 19.1 19.1 19.1 19.1	<b>3</b> <b>5</b> <b>7</b> <b>1</b> <b>1</b> <b>1</b> <b>1</b> <b>1</b> <b>1</b> <b>1</b> <b>1</b>	al Mea 0 265 59 330 0 291 34 209 27 245 14 271 29 354 27 245 5 149 16 232 55 283 79 188	an SI .0 75.0 .0 128.0 .6 115. .5 153.0 .8 144. .9 132.3 .0 140.3 .2 207.4 .7 84. .7 211.5 .0 109.0 .4 124.0	0 0 1 1 6 1 3 3 4 1 5 0 6 6 2	Favors Control Favors	MD -12.00 -1.00 -5.90 [ -9.96 23.30 -0.30 32.00 38.40 18.40 18.60 18.60 18.60 18.60 18.60 11.01	<b>95%-CI</b> -34.51; 10.51] -50.50; 48.50] -47.84; 159.64] -32.52; 79.12] -64.57; 63.97] -24.26; 88.26] -55.16; 61.16; 129.32; 221.34] [ -6.81; 16.81] -9.77; 46.97]	Weig 13.1 4.1 1.1 2.4 3.3 3.1 9.1 0.4 22.1 9.5 8.0
Heterogeneity: $l^2 = 83\%$ , $t^2 = 7$ Test for overall effect: $t_{16} = -0$ Author and Year Altieri et al., 2013 Ålvarez-Blasco et al., 2011 Colombo et al., 2009 Cunha et al., 2019 Cutler et al., 2019 Cutler et al., 2019 Lin et al., 2021 Melekoglu et al., 2020 Misir et al., 2013 Moran et al., 2013 Navaro-Lafuente et al., 2022 Wingh et al., 2004	0.2904, $\chi^2_1$ .51 ( $p$ = 0.1 <b>Total Me</b> : 100 255 22 326 8 347 30 244 80 303 40 355 65 166 12 276 409 277 121 302 202 493 84 199	PCOS an SI 0 87.1 0 89.1 5 108.1 5 108.1 1 185.1 6 113.1 0 173.1 1 185.1 6 113.1 0 173.1 1 185.1 2 178.1 1 19.1 0 178.1 0 119.1 0 128.2 0 119.1 0 128.2 1 0 128.1 1 19.1 19.1 19.1 19.1 19.1 19.1 19.1	<b>3</b> <b>5</b> <b>7</b> <b>1</b> <b>1</b> <b>1</b> <b>1</b> <b>1</b> <b>1</b> <b>1</b> <b>1</b>	al Mez 00 265 59 330 00 291 50 260 59 330 00 291 59 354 42 291 50 260 57 265 57 265 57 265 57 265 57 263 57 265 57 283 25 413 91 188 91 186	an         SI           .0         75.0           .0         128.0           .6         115.1           .5         153.0           .8         144.1           .9         132.2           .0         140.3           .2         207.4           .7         841.2           .7         210.0           .4         124.0           .9         210.0           .8         89.2	0 0 1 1 6 1 3 3 4 1 5 0 6 6 2	Favors Control Favors	MD -12.00 -1.00 -5.90 [ -9.96 23.30 -0.30 32.00 38.40 18.40 18.60 18.60 18.60 18.60 18.60 11.01	95%-CI -34.51; 10.51] -50.50; 48.50] -47.84; 159.64] -77.21; 57.29] -64.57; 63.97] -24.26; 88.26] -55.16; 61.16] -10.26; 47.06] 129.32; 221.34] (-8.81; 16.81] (-9.77; 46.97] -24.92; 124.25] -21.91; 43.93]	Weig 13.1 1.1 2.4 3.3 3.1 9.7 4.8 8.0 12.0

### Figure 5. Continued

identified across studies reporting total carbohydrate and added sugar (all  $l^2 \le 89\%$ ; All  $P \le 0.001$ ).

Subgroup analyses based on age, BMI, PCOS criteria, dietary assessment tool, or country did not explain heterogeneity (Supplementary Table SIV). Further, the small number of studies (<2) in each subgroup did not allow subgroup analyses for added sugar intake. Sensitivity analyses of SMD results for total carbohydrates showed none of the individual studies influenced the overall effect size. We observed no publication bias for studies reporting carbohydrate intake and added sugar intakes (funnel plot, Supplementary Fig. S5A and B, respectively; All  $P \ge 0.14$ , Begg's tests; All  $P \ge 0.22$ , Egger's tests).

**Total fat, SFA, MUFA, PUFA and cholesterol.** Total fat consumption was similar between groups. (SMD: 0.11; 95% CI: -0.16 to 0.37; P = 0.43; Fig. 5A; N = 29) (Wright et al., 2004a; Douglas et al., 2006; Colombo et al., 2009; Álvarez-Blasco et al., 2011; Pourghassem Gargari et al., 2011; Altieri et al., 2013; Moran et al., 2013; Tsai et al., 2013; Pourghassem Gargari et al., 2015; Hart et al., 2016; Larsson et al., 2016; Misir et al., 2016; Shishehgar et al., 2016; Eslamian et al., 2017; Zaeemzadeh et al., 2018; Alipour et al., 2019; Barrea et al., 2019; Cunha et al., 2019; Cutler et al., 2019; Ganie et al., 2019; Lin et al., 2019; Shahdadian et al., 2019; Shishehgar et al., 2019; Melekoglu

et al., 2020; Liang et al., 2021; Lin et al., 2021; Noormohammadi et al., 2021; Soodi et al., 2021; Navarro-Lafuente et al., 2022; Wang et al., 2022). Likewise intakes of SFA (SMD: -0.06; 95% CI: -0.64 to 0.52; P = 0.83; Fig. 5B; N = 20) (Wright et al., 2004a; Douglas et al., 2006; Colombo et al., 2009; Álvarez-Blasco et al., 2011; Pourghassem Gargari et al., 2011; Altieri et al., 2013; Moran et al., 2013; Hart et al., 2016; Larsson et al., 2016; Misir et al., 2016; Shishehgar et al., 2016a; Eslamian et al., 2017; Zaeemzadeh et al., 2018; Barrea et al., 2019; Cunha et al., 2019; Lin et al., 2019; Melekoglu et al., 2020; Lin et al., 2021; Zirak Sharkesh et al., 2021; Navarro-Lafuente et al., 2022), MUFA (SMD: -0.06; 95% CI: -0.36; to 0.24; P=0.70; Fig. 5C; N = 17) (Wright et al., 2004a; Douglas et al., 2006; Colombo et al., 2009; Álvarez-Blasco et al., 2011; Pourghassem Gargari et al., 2011; Moran et al., 2013; Hart et al., 2016; Misir et al., 2016; Shishehgar et al., 2016a; Barrea et al., 2019; Lin et al., 2019; Melekoglu et al., 2020; Lin et al., 2021; Zirak Sharkesh et al., 2021; Navarro-Lafuente et al., 2022; Wang et al., 2022) and PUFA (SMD: -0.08; 95% CI: -0.40 to 0.25; P=0.62; Fig. 5D; N=17) (Wright et al., 2004a; Douglas et al., 2006; Colombo et al., 2009; Álvarez-Blasco et al., 2011; Pourghassem Gargari et al., 2011; Moran et al., 2013; Hart et al., 2016; Misir et al., 2016; Shishehgar et al., 2016a; Barrea et al., 2019;

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			P	cos		Co	ntrol				
Author and Year	1	Total	Mean	SD	Total	Mean	SD	Standardized Mean Difference	e SMD	95%-CI	Weigh
Alipour et al., 2019		45	65.5	17.5	45	63.2	16.7	±	0.14	[-0.28; 0.55]	3.3%
Altieri et al., 2013		100	81.1		100			200		[-0.15; 0.40]	3.5%
Alvarez-Blasco et al., 2011			106.0			102.0		<u>-</u>		[-0.36; 0.62]	3.1%
Barrea et al., 2019		112	87.0		112			1		[-0.41; 0.12]	3.6%
Colombo et al., 2009		8	96.7		10			T-	0.63		2.0%
Cunha et al., 2019		39	75.3		34			1		[-0.25; 0.67]	3.1%
Cutler et al., 2019		87	16.8		50	16.4		E		[-0.24; 0.45]	3.4%
Douglas et al., 2006		30	72.3		27	72.3				[-0.52; 0.52]	3.0%
Eslamian et al., 2017			111.6			110.0		2		[-0.07; 0.23]	3.7%
Ganie et al., 2019 (non-vegetarian sub-co	hort)	62	55.2		141			25		[-0.21; 0.38]	3.5%
Sanie et al., 2019 (vegetarian sub-cohort)		82	50.6		179			2	-0.09		3.6%
Hart et al., 2016		38	16.0		30			The second se		[-0.15; 0.81]	3.1%
arsson et al., 2016		51	18.0		29			<u>E</u>		[-0.23; 0.69]	3.2%
iang et al., 2020 (lean sub-cohort)		10	60.2		10						2.0%
		10	64.4		10					[-1.89; -0.02]	2.19
iang et al., 2021 (overweight sub-cohort)								T		[-1.49; 0.31]	
_in et al., 2019		80 40	86.0		44			王		[-0.32; 0.41]	3.4%
in et al., 2021			83.3		529			素		[-0.30; 0.34]	3.5%
Melekoglu et al., 2020		65	52.9		65			<b>T</b>		[-0.45; 0.24]	3.4%
Misir et al., 2016		12	81.7		16			1		[-0.21; 1.32]	2.4%
Moran et al., 2013		409	21.1	3.2	7057		3.3	2		[-0.04; 0.16]	3.8%
Navarro-Lafuente et al., 2022		121	91.8		155			1		[-0.24; 0.23]	3.6%
loormohammadi et al., 2021	2	303			588				0.73		3.7%
Pourghassem Gargari et al., 2011 and 201	5	30	49.9	2.4	30		5.9			[-4.67; -2.94]	2.2%
Shahdadian et al., 2019		225	89.4		345					[0.19; 0.53]	3.7%
Shishehgar et al., 2016		142	66.2		140			-		[-0.37; 0.10]	3.6%
Shishehgar et al., 2019		28	10.6		34			크		[-0.84; 0.17]	3.0%
Soodi et al., 2021		203	86.2		291			<u>9</u>		[-0.25; 0.11]	3.7%
Tsai et al., 2013		45	67.0		161					[-0.39; 0.27]	3.4%
Nang et al. 2022		202	51.2		325			- E		[-0.05; 0.30]	3.7%
Wright et al., 2004		84	65.7		79			_=		[-0.20; 0.41]	3.5%
Zaeemzadeh et al., 2018		151	71.8	25.2	31	88.5	30.9	*	-0.64	[-1.03; -0.24]	3.3%
Overall effect Heterogeneity: $l^2 = 85\%$ , $t^2 = 0.2639$ , $\chi^2_{30} =$ Test for overall effect: $l_{30} = -0.36$ ( $p = 0.72$	206.0	<b>3117</b> 6 (p <	0.001)		11198			-4 -2 0 2 4 Favors Control Favors PCOS		[-0.28; 0.20]	100.0%
uthor and Year	Total	Mea	PCOS n SD		al Mea	Contro an SD		Mean Difference	MD	95%-CI	Weigh
lipour et al., 2019	45	14.	1 4.7		5 13	.5 5.0	1	its.	0.54	-1.46; 2.54]	4.2%
lipour et al., 2019 Itieri et al., 2013	45	14.						12			4.2%
Ivarez-Blasco et al., 2011	22		0 11.0		59 22			20		-0.33; 2.53]	3.4%
	112	15.						3		-3.93; 5.93]	4.3%
arrea et al., 2019								11 I I I I I I I I I I I I I I I I I I		-2.82; -0.76]	
olombo et al., 2009	8	24.			0 24			-		-6.34; 6.54]	3.0%
unha et al., 2019	39	10.			4 11					[-4.36; 1.44]	4.0%
utler et al., 2019	87	19.			50 23					-6.53; -0.87]	4.0%
ouglas et al., 2006	30	14.			27 15					[-3.32; 2.32]	4.0%
slamian et al., 2017	281	12.								18.26; -16.74]	4.49
lart et al., 2016	38	15.			80 16					[-3.00; 2.00]	4.19
arsson et al., 2016	51	23.			9 22					[-2.49; 4.49]	3.89
iang et al., 2020 (lean sub-cohort)	10	9.			0 10					[-4.79; 1.31]	4.0%
iang et al., 2020 (overweight sub-cohort)	10	8.			0 12					-6.11; -0.17]	4.0%
in et al., 2019	80	24.			4 25					[-4.52; 2.52]	3.89
n et al., 2021	40	4.				.0 2.8				-0.85; 0.65]	4.49
Melekoglu et al., 2020	65	20.	7 7.7	6	5 25	8 9.7			-5.10 I	-8.11: -2.091	4.0%

# В

			14.1	4.1		13.5	5.0	200				
ltieri et al., 2013		100	19.3	5.0		18.2	5.3	10	1.10		; 2.53	
lvarez-Blasco et al., 2011		22	23.0			22.0	7.0	*	1.00		; 5.93	
arrea et al., 2019		112	15.4	3.7		17.2	4.2		-1.79			
Colombo et al., 2009		8	24.4	8.1		24.3	5.1	*	0.10		; 6.54	
Cunha et al., 2019		39	10.4	5.2		11.8	7.1	100 H	-1.46			
Cutler et al., 2019		87	19.6	5.9		23.3	9.2	-		[-6.53		
ouglas et al., 2006		30	14.9	3.3		15.4	6.8	- <del>-</del>		[-3.32		
slamian et al., 2017		281	12.0	5.3		29.5	4.9			[-18.26;		
lart et al., 2016		38	15.7	5.5		16.2	5.0		-0.50	[-3.00	; 2.00	
arsson et al., 2016		51	23.0	7.0		22.0	8.0		1.00		; 4.49	
iang et al., 2020 (lean sub-cohort		10	9.0	2.4		10.8	4.3		-1.74		; 1.31	
iang et al., 2020 (overweight sub-	-cohort)		8.9	1.8		12.1	4.4			[-6.11		
in et al., 2019		80	24.0	8.9		25.0	9.9		-1.00		; 2.52	
in et al., 2021		40	4.9	2.3	529	5.0	2.8	10	-0.10	[-0.85	; 0.65	4.49
felekoglu et al., 2020		65	20.7	7.7	65	25.8	9.7	100	-5.10	[-8.11	-2.09	4.0%
lisir et al., 2016		12	15.7	8.0	16	14.2	6.5	*	1.52	[-4.01	; 7.05	3.29
foran et al., 2013		409	20.2	8.4	7057	19.0	7.0	10	1.20		; 2.03	
lavarro-Lafuente et al., 2022		121	19.6	6.5	155	20.6	6.1	-	-1.00	[-2.50	; 0.50	4.39
loormohammadi et al., 2021		303	13.0	3.9	588	16.0	4.1	10	-3.00	[-3.55	-2.45	4.49
ourghassem Gargari et al., 2015		30	6.0	1.0	30	6.7	0.6		-0.70	[-1.12	-0.28	4.49
hishehgar et al., 2016		142	19.0	12.9	140	21.4	14.6	100	-2.35	[-5.57	; 0.87	3.99
hishehgar et al., 2019		28	25.6	12.7	34	26.5	17.5	-	-0.90	[-8.43	; 6.63	2.69
oodi et al., 2021		203	44.7	23.5	291	38.0	18.2	-	6.72	[ 2.87	10.57	3.79
Vang et al. 2022		202	7.7		325	8.9	4.2	10	-1.22	[ -1.89	-0.56	4.49
		202 151	7.7 33.4			8.9 76.3				[ -1.89 [ -57.97;		
Vang et al. 2022 Gaeemzadeh et al., 2018 Overall effect		151 2619	33.4	22.6 1	31 0293				-42.95		-27.94	1.2%
Vang et al. 2022 aeemzadeh et al., 2018 <b>Overall effect</b> leterogeneity: / <sup>2</sup> = 99%, τ <sup>2</sup> = 22.34	602, χ <sup>2</sup> <sub>25</sub>	151 <b>2619</b> = 1802.	33.4	22.6 1	31 0293				-42.95	[-57.97;	-27.94	1.2%
Vang et al. 2022 Gaeemzadeh et al., 2018 Overall effect	602, χ <sup>2</sup> <sub>25</sub> p = 0.13	151 <b>2619</b> = 1802.	33.4	22.6 1	31 0293			-40 -20 0 20 40	-42.95	[-57.97;	-27.94	1.2%
Vang et al. 2022 aeemzadeh et al., 2018 <b>Overall effect</b> leterogeneity: / <sup>2</sup> = 99%, τ <sup>2</sup> = 22.34	602, χ <sup>2</sup> <sub>25</sub> p = 0.13	151 <b>2619</b> = 1802.	33.4	22.6 1	31 0293				-42.95	[-57.97;	-27.94	1.2%
Vang et al. 2022 aeemzadeh et al., 2018 <b>Overall effect</b> leterogeneity: / <sup>2</sup> = 99%, τ <sup>2</sup> = 22.34	602, χ <sub>25</sub> p = 0.13	151 <b>2619</b> = 1802.	33.4	22.6 1	31 0293			-40 -20 0 20 40	-42.95	[-57.97;	-27.94	1.2%
Vang et al. 2022 aeemzadeh et al., 2018 <b>Overall effect</b> leterogeneity: / <sup>2</sup> = 99%, τ <sup>2</sup> = 22.34	602, χ <sup>2</sup> <sub>25</sub> p = 0.13	151 <b>2619</b> = 1802.	33.4	22.6 1	31 0293			-40 -20 0 20 40	-42.95	[-57.97;	-27.94	1.29
Vang et al. 2022 aeemzadeh et al., 2018 <b>Overall effect</b> leterogeneity: / <sup>2</sup> = 99%, τ <sup>2</sup> = 22.34	602, χ <sub>25</sub> p = 0.13	151 2619 = 1802.	33.4 87 (p •	22.6 1	31 0293	76.3 4	41.4	-40 -20 0 20 40	-42.95	[-57.97;	-27.94	1.29
Vang et al. 2022 aeemzadeh et al., 2018 <b>Overall effect</b> leterogeneity: / <sup>2</sup> = 99%, τ <sup>2</sup> = 22.34	p = 0.13	151 2619 = 1802. ))	33.4 87 (p -	22.6 1 < 0.001	31 0293	ntrol	41.4	-40 -20 0 20 40	-42.95	6 [-57.97; 2 [-4.43	-27.94	1.29
Vang et al. 2022 acemzadeh et al., 2018 <b>Overall effect</b> leterogeneity: $l^2 = 99\%$ , $\tau^2 = 22.34$ est for overall effect: $t_{25} = -1.58$ ( $t_{25}$	p = 0.13	151 2619 = 1802. ))	33.4 87 (p -	22.6 1 < 0.001	31 0293 Co Mean	ntrol SD	41.4	-40 -20 0 20 40 Favors Control Favors PCOS	-42.95 -1.92 MD	; [-57.97; ; [-4.43 9;	-27.94 ; 0.59] 5%-CI	1.29 100.09 Weigh
Vang et al. 2022 (acemzadeh et al., 2018) <b>Overall effect</b> leterogeneity: $l^2 = 99\%$ , $\tau^2 = 22.34$ est for overall effect: $t_{26} = -1.58$ ( $t_{26}$ <b>Author and Year</b> Álvarez-Blasco et al., 2011	p = 0.13 Total 22	151 2619 = 1802. ) P Mean 0.5	33.4 87 (p - COS SD 3.8	22.6 1 0.001) <b>Total</b> 59	31 0293 Co Mean 1.2	ntrol SD 3.1	41.4	-40 -20 0 20 40 Favors Control Favors PCOS	-42.95 -1.92 MD -0.71	9: [-2.48;	-27.94 ; 0.59] 5%-CI 1.06]	1.29 100.09 Weigh 15.49
Vang et al. 2022 laeemzadeh et al., 2018 <b>Dverall effect</b> leterogeneity: $l^2 = 99\%, \tau^2 = 22.34$ est for overall effect: $t_{25} = -1.58$ ( $t$ <b>Author and Year</b> Álvarez-Blasco et al., 2011 Colombo et al., 2009	p = 0.13 Total 22 8	151 2619 = 1802. ) P Mean 0.5 1.6	33.4 87 (p • COS SD 3.8 1.6	22.6 1 0.001; <b>Total</b> 59 10	31 0293 Co Mean 1.2 9.5	ntrol SD 3.1 15.7	41.4	-40 -20 0 20 40 Favors Control Favors PCOS	-42.95 -1.92 MD -0.71 -7.90	9: [-2.48; [-17.69;	-27.94 ; 0.59] 5%-CI 1.06] 1.89]	1.29 100.09 Weigh 15.49 0.59
Vang et al. 2022 acemzadeh et al., 2018 <b>bverall effect</b> letterogeneity: $l^2 = 99\%$ , $t^2 = 22.34$ est for overall effect: $t_{25} = -1.58$ ( $t_{25}$ <b>Author and Year</b> <b>Author and Year</b> Alvarez—Blasco et al., 2011 Colombo et al., 2009 Lin et al., 2019	<b>Total</b> 22 8 80	151 2619 = 1802. ) P Mean 0.5 1.6 9.0	33.4 87 (p • • COS SD 3.8 1.6 15.6	22.6 1 0.001) <b>Total</b> 59 10 44	31 0293 Co Mean 1.2 9.5 9.0	ntrol SD 3.1 15.7 13.2	41.4	-40 -20 0 20 40 Favors Control Favors PCOS	-42.95 -1.92 MD -0.71 -7.90 0.00	9: [-2.48; [-17.69; [-5.18;	-27.94 ; 0.59 5%-CI 1.06] 1.89] 5.18]	1.29 100.09 Weigh 15.49 0.59 1.89
Vang et al. 2022 acemzadeh et al., 2018 <b>Overall effect</b> letterogeneity: $l^2 = 99\%$ , $\tau^2 = 22.34$ est for overall effect: $t_{25} = -1.58$ ( $t_{25}$ <b>Author and Year</b> <b>Author and Year</b> Álvarez–Blasco et al., 2011 Colombo et al., 2009 Lin et al., 2013	<b>Total</b> 22 8 80 409	151 2619 = 1802. )) P Mean 0.5 1.6 9.0 8.4	33.4 87 (p - 87 (p - 3.8 1.6 15.6 13.4	22.6 1 0.001) Total 59 10 44 7057	31 0293 Co Mean 1.2 9.5 9.0 9.3	ntrol SD 3.1 15.7 13.2 13.4		-40 -20 0 20 40 Favors Control Favors PCOS	-42.95 -1.92 MD -0.71 -7.90 0.00 -0.90	9 [-2.48; [-17.69 [-5.18; [-2.24;	-27.94 ; 0.59] 5%-CI 1.06] 1.89] 5.18] 0.44]	1.29 100.09 Weigh 15.49 0.59 1.89 27.09
Vang et al. 2022 acemzadeh et al., 2018 <b>bverall effect</b> letterogeneity: $l^2 = 99\%$ , $t^2 = 22.34$ est for overall effect: $t_{25} = -1.58$ ( $t_{25}$ <b>Author and Year</b> <b>Author and Year</b> Alvarez—Blasco et al., 2011 Colombo et al., 2009 Lin et al., 2019	<b>Total</b> 22 8 80 409	151 2619 = 1802. ) P Mean 0.5 1.6 9.0 8.4	33.4 87 (p • • COS SD 3.8 1.6 15.6	22.6 1 0.001) <b>Total</b> 59 10 44	31 0293 Co Mean 1.2 9.5 9.0 9.3	ntrol SD 3.1 15.7 13.2		-40 -20 0 20 40 Favors Control Favors PCOS	-42.95 -1.92 MD -0.71 -7.90 0.00 -0.90	9: [-2.48; [-17.69; [-5.18;	-27.94 ; 0.59] 5%-CI 1.06] 1.89] 5.18] 0.44]	1.2 100.0 Weigh 15.4 0.5 1.8 27.0
Vang et al. 2022 acemzadeh et al., 2018 <b>Overall effect</b> letterogeneity: $l^2 = 99\%$ , $\tau^2 = 22.34$ est for overall effect: $t_{25} = -1.58$ ( $t_{25}$ <b>Author and Year</b> <b>Author and Year</b> Álvarez–Blasco et al., 2011 Colombo et al., 2009 Lin et al., 2013	<b>Total</b> 22 8 80 409	151 2619 = 1802. ) P Mean 0.5 1.6 9.0 8.4	33.4 87 (p - 87 (p - 3.8 1.6 15.6 13.4	22.6 1 0.001) Total 59 10 44 7057	31 0293 Co Mean 1.2 9.5 9.0 9.3	ntrol SD 3.1 15.7 13.2 13.4		-40 -20 0 20 40 Favors Control Favors PCOS	-42.95 -1.92 MD -0.71 -7.90 0.00 -0.90 -1.00	9 [-2.48; [-17.69 [-5.18; [-2.24;	-27.94 ; 0.59 ; 0.59 5%-CI 1.06] 1.89] 5.18] 0.44] -0.07]	1.29 100.09 Weigh 15.49 0.59 1.89 27.09 55.39
Vang et al. 2022 acemzadeh et al., 2018 <b>Everall effect</b> leterogeneity: $l^2 = 99\%, \tau^2 = 22.34$ est for overall effect: $t_{25} = -1.58$ ( $t_{25}$ <b>Author and Year</b> <b>Author and Year</b> Álvarez–Blasco et al., 2011 Colombo et al., 2009 Lin et al., 2019 Moran et al., 2013 Navarro–Lafuente et al., 2022 <b>Overall effect</b>	<b>Total</b> 22 8 80 409 121 <b>640</b>	151 <b>2619</b> = 1802. ) <b>P</b> Mean 0.5 1.6 9.0 8.4 2.8	33.4 87 (p + SD 3.8 1.6 15.6 13.4 3.7	22.6 <b>Total</b> 59 10 44 7057 <b>7325</b>	31 0293 Co Mean 1.2 9.5 9.0 9.3	ntrol SD 3.1 15.7 13.2 13.4		-40 -20 0 20 40 Favors Control Favors PCOS	-42.95 -1.92 MD -0.71 -7.90 0.00 -0.90 -1.00	9 [-2.48; [-17.69; [-2.24; [-2.24; [-1.93;	-27.94 ; 0.59 ; 0.59 5%-CI 1.06] 1.89] 5.18] 0.44] -0.07]	1.29 100.09 Weigh 15.49 0.59 1.89 27.09 55.39
Vang et al. 2022 acemzadeh et al., 2018 <b>bverall effect</b> letterogeneity: <i>I</i> <sup>2</sup> = 99%, τ <sup>2</sup> = 22.34 est for overall effect: <i>t</i> <sub>25</sub> = -1.58 ( <i>t</i> <b>Author and Year</b> <b>Author and Year</b> <b>Author and Year</b> <b>Aivarez-Biasco</b> et al., 2011 Colombo et al., 2009 Lin et al., 2019 Moran et al., 2013 Navarro-Lafuente et al., 2022 <b>Overall effect</b> Heterogeneity: <i>I</i> <sup>2</sup> = 0%, τ <sup>2</sup> < 0.	<b>Total</b> 22 8 80 409 121 <b>640</b> .0001, ;	151 2619 = 1802. ) P Mean 0.5 1.6 9.0 8.4 2.8 $\chi^2_4 = 2.1$	33.4 87 (p + SD 3.8 1.6 15.6 13.4 3.7	22.6 <b>Total</b> 59 10 44 7057 <b>7325</b>	31 0293 Co Mean 1.2 9.5 9.0 9.3	ntrol SD 3.1 15.7 13.2 13.4		-40 -20 0 20 40 Favors Control Favors PCOS	-42.95 -1.92 MD -0.71 -7.90 0.00 -0.90 -1.00	9 [-2.48; [-17.69; [-2.24; [-2.24; [-1.93;	-27.94 ; 0.59 ; 0.59 5%-CI 1.06] 1.89] 5.18] 0.44] -0.07]	1.29 100.09 Weigh 15.49 0.59 1.89 27.09 55.39
Vang et al. 2022 acemzadeh et al., 2018 <b>Everall effect</b> leterogeneity: $l^2 = 99\%, \tau^2 = 22.34$ est for overall effect: $t_{25} = -1.58$ ( $t_{25}$ <b>Author and Year</b> <b>Author and Year</b> Álvarez–Blasco et al., 2011 Colombo et al., 2009 Lin et al., 2019 Moran et al., 2013 Navarro–Lafuente et al., 2022 <b>Overall effect</b>	<b>Total</b> 22 8 80 409 121 <b>640</b> .0001, ;	151 2619 = 1802. ) P Mean 0.5 1.6 9.0 8.4 2.8 $\chi^2_4 = 2.1$	33.4 87 (p + SD 3.8 1.6 15.6 13.4 3.7	22.6 <b>Total</b> 59 10 44 7057 <b>7325</b>	31 0293 Co Mean 1.2 9.5 9.0 9.3	ntrol SD 3.1 15.7 13.2 13.4		-40 -20 0 20 40 Favors Control Favors PCOS	-42.95 -1.92 MD -0.71 -7.90 0.00 -0.90 -1.00	9 [-2.48; [-17.69; [-2.24; [-2.24; [-1.93;	-27.94 ; 0.59 ; 0.59 5%-CI 1.06] 1.89] 5.18] 0.44] -0.07]	1.29 100.09 Weigh 15.49 0.59 1.89 27.09 55.39

**Figure 6.** Forest plots for protein, fiber and alcohol intake in women with and without PCOS. (A) protein intake; (B) fiber intake expressed as g/day; and (C) alcohol intake expressed as g/day. MD, mean difference; SMD, standardized mean difference.

Lin et al., 2019; Melekoglu et al., 2020; Lin et al., 2021; Zirak Sharkesh et al., 2021; Navarro-Lafuente et al., 2022; Wang et al., 2022) were comparable. In contrast, women with PCOS had higher cholesterol intakes (MD: 12.78, 95% Cl: 1.48 to 24.08 mg/day; P = 0.03; Fig. 5E; N = 15) (Wright et al., 2004a; Douglas et al., 2006; Colombo et al., 2009; Álvarez-Blasco et al., 2011; Altieri et al., 2006; Colombo et al., 2013; Misir et al., 2016; Cunha et al., 2019; Cutler et al., 2019; Lin et al., 2019; Melekoglu et al., 2020; Lin et al., 2021; Zirak Sharkesh et al., 2021; Navarro-Lafuente et al., 2022; Wang et al., 2022). We observed high heterogeneity across all studies that reported data on fat intake (all  $l^2 \ge 83\%$ ; All P < 0.001), but studies on cholesterol intake were homogeneous ( $l^2 = 18\%$ ; P = 0.24).

For more clarity, we reported effect estimates for relevant fat intake outcomes separately in g/day and %energy intake/day. Total fat intake in g/day (MD: 3.12, 95% CI: -2.40 to 8.63; P = 0.26; Supplementary Fig. S6A; N = 24) (Wright et al., 2004a; Douglas et al., 2006; Colombo et al., 2009; Álvarez-Blasco et al., 2011; Pourghassem Gargari et al., 2011; Altieri et al., 2013; Tsai et al., 2013; Pourghassem Gargari et al., 2015; Misir et al., 2016; Shishehgar et al., 2016b; Eslamian et al., 2017; Zaeemzadeh et al., 2018; Alipour et al., 2019; Barrea et al., 2019; Cunha et al., 2019; Ganie et al., 2019; Lin et al., 2019; Shahdadian et al., 2019; Melekoglu et al., 2020; Liang et al., 2021; Lin et al., 2021; Noormohammadi et al., 2021; Soodi et al., 2021; Navarro-Lafuente et al., 2022; Wang et al., 2022) and in %energy intake/day (MD: 1.02, 95% CI: -1.70 to 3.74; P=0.36 Supplementary Fig. S6B; N = 5) (Moran et al., 2013; Hart et al., 2016; Larsson et al., 2016; Cutler et al., 2019; Shishehgar et al., 2019) were comparable between groups.

With respect to SFA, intakes in g/day (MD: 0.11, 95% CI: -2.15 to 2.38 P = 0.92; Supplementary Fig. S6C; N = 16) (Wright et al., 2004a; Douglas et al., 2006; Colombo et al., 2009; Álvarez-Blasco et al., 2011; Pourghassem Gargari et al., 2011; Altieri et al., 2013; Misir et al., 2016; Shishehgar et al., 2016a; Eslamian et al., 2017; Zaeemzadeh et al., 2018; Barrea et al., 2019; Cunha et al., 2019; Lin et al., 2019, 2021; Zirak Sharkesh et al., 2021; Navarro-Lafuente et al., 2022) and in %energy intake/day (MD: 0.92, 95% CI: -1.18 to 3.01; P = 0.26; Supplementary Fig. S6D; N = 4) (Moran et al., 2013; Hart et al., 2016; Larsson et al., 2016; Melekoglu et al., 2020) were similar.

Regarding unsaturated fats, intakes in g/day (MUFA: MD: -0.31, 95% CI: -2.27 to 1.65; P=0.74; Supplementary Fig. S6E, PUFA: MD: -0.33, 95% CI: -1.41 to 0.76; P=0.53; Supplementary Fig. S6F, N = 14) (Wright et al., 2004a; Douglas et al., 2006; Colombo et al., 2009; Álvarez-Blasco et al., 2011; Pourghassem Gargari et al., 2011; Misir et al., 2016; Shishehgar et al., 2016a; Barrea et al., 2019; Lin et al., 2019, 2021; Zirak Sharkesh et al., 2021; Navarro-Lafuente et al., 2022; Wang et al., 2022) and in %energy intake/day (MUFA: MD: 0.69, 95% CI: -1.46 to 2.85; P=0.30; Supplementary Fig. S6G; N=3; PUFA: MD: 0.30, 95% CI: -1.03 to 1.64; P=0.43; Supplementary Fig. S6H, N=3) (Moran et al., 2013; Hart et al., 2016; Melekoglu et al., 2020) were comparable.

Subgroup analyses did not explain heterogeneity for SFA, MUFA, and cholesterol. Conversely, PCOS groups demonstrated higher total fat versus Controls in the subgroup of studies that used food records for dietary assessment (SMD: 0.20; 95% Cl: 0.02 to 0.38; P = 0.03). We observed higher intakes of PUFA in PCOS versus Controls in the

subgroup of studies that used AEPCOS criteria (SMD: 0.17; 95% CI: 0.13 to 0.19; P < 0.01; Supplementary Table SIV). None of the effect estimates for fat intake were sensitive to individual studies except one study: omitting the study by Zirak Sharkesh *et al.*, (2021) resulted in the loss of differences for overall effect estimate in cholesterol intake; however, the direction of effect estimate was retained (MD: 12.10; 95% CI: -0.59 to 24.79; P = 0.06). We observed no evidence of publication bias (funnel plots, Supplementary Fig. S7A–E; All  $P \le 0.16$ , Begg's tests; All  $P \ge 0.22$ , Egger's tests).

**Total protein, fiber and alcohol.** Groups had similar total protein intakes (Wright et al., 2004a; Douglas et al., 2006; Colombo et al., 2009; Álvarez-Blasco et al., 2011; Pourghassem Gargari et al., 2011; Altieri et al., 2013; Moran et al., 2013; Tsai et al., 2013; Pourghassem Gargari et al., 2015; Hart et al., 2016; Larsson et al., 2016; Misir et al., 2016; Shishehgar et al., 2016a; Eslamian et al., 2017; Zaeemzadeh et al., 2018; Alipour et al., 2019; Barrea et al., 2019; Cunha et al., 2019; Ganie et al., 2019; Lin et al., 2019; Shahdadian et al., 2019; Shishehgar et al., 2019; Moormohammadi et al., 2021; Soodi et al., 2021; Navarro-Lafuente et al., 2022; Wang et al., 2022) (SMD: -0.04; 95% CI: -0.28 to 0.20; P = 0.72; Fig. 6A; N = 29).

Protein intakes were similar in studies that reported in g/day (MD: -0.26, 95% CI: -3.31 to 2.79; P = 0.86; Supplementary Fig. S8A; N = 24) (Wright et al., 2004a; Douglas et al., 2006; Colombo et al., 2009; Álvarez-Blasco et al., 2011; Pourghassem Gargari et al., 2011; Altieri et al., 2013; Tsai et al., 2013; Pourghassem Gargari et al., 2015; Misir et al., 2016; Shishehgar et al., 2016a; Eslamian et al., 2017; Zaeemzadeh et al., 2018; Alipour et al., 2019; Barrea et al., 2019; Cunha et al., 2019; Ganie et al., 2019; Lin et al., 2019; Shahdadian et al., 2019; Melekoglu et al., 2020; Liang et al., 2021; Lin et al., 2021; Noormohammadi et al., 2022; or in %energy intake/day (MD: 0.18, 95% CI: -0.26 to 0.62; P = 0.81; N = 5; Supplementary Fig. S8B) (Moran et al., 2013; Hart et al., 2016; Larsson et al., 2016; Cutler et al., 2019; Shishehgar et al., 2019).

Similarly, pooling analyses showed that PCOS and Control groups had similar intakes of fiber (MD: -1.92, 95% CI: -4.43 to 0.59 g/day; P = 0.13; Fig. 6B; N = 24) (Douglas et al., 2006; Colombo et al., 2009; Álvarez-Blasco et al., 2011; Altieri et al., 2013; Moran et al., 2013; Pourghassem Gargari et al., 2015; Hart et al., 2016; Larsson et al., 2016; Misir et al., 2016; Shishehgar et al., 2016a; Eslamian et al., 2017; Zaeemzadeh et al., 2018; Alipour et al., 2019; Barrea et al., 2019; Cutler et al., 2019; Lin et al., 2019; Shishehgar et al., 2019; Melekoglu et al., 2020; Liang et al., 2021; Noormohammadi et al., 2021; Soodi et al., 2021; Navarro-Lafuente et al., 2022; Wang et al., 2022). In contrast, women with PCOS had lower alcohol intakes (MD: -0.95, 95% CI: -1.67 to -0.22 g/day; P = 0.02; Fig. 6C; N = 5) (Colombo et al., 2009; Álvarez-Blasco et al., 2011; Moran et al., 2013; Lin et al., 2019; Navarro-Lafuente et al., 2022). Studies reporting protein and fiber intakes were heterogeneous (all  $l^2 \le 85\%$ ; P < 0.001), unlike those reporting alcohol intakes ( $l^2 = 0\%$ ; P = 0.71).

Subgroup analyses based on age, BMI, PCOS criteria, dietary assessment tool or country did not explain heterogeneity except lower

# А

			PCOS			Control	
Author and Year	Total	Mean	SD	Total	Mean	SD	Mean Difference
Cutler et al., 2019	87	256.5	148.9	50	283.5	112.4	*
Lin et al., 2019	80	275.0	102.4	44	303.0	123.8	
Lin et al., 2021	40	565.9	1248.4	529	470.7	1922.4	
Melekoglu et al., 2020	65	291.3	103.4	65	341.9	109.9	墨
Moran et al., 2013	409	253.0	107.0	7057	242.0	87.0	
Pourghassem Gargari et al., 2011	30	119.2	63.5	30	165.4	128.6	
Szczuko et al., 2021	40	221.2	65.4	15	262.8	78.6	
Wang et al. 2022	202	214.8	90.7	325	251.0	110.8	10
Zirak Sharkesh et al. 2021	203	573.8	176.3	291	551.8	154.6	
Overall effect	1156			8406			•
Heterogeneity: $l^2 = 79\%$ , $\tau^2 = 584$	.8722,	$r_{o}^{2} = 37.$	51 (p < 0	0.001)			
Test for overall effect: $t_8 = -2.19$ (							-400 -200 0 200 4
							Favors Control Favors PC

ifference	MD	95%-C	Weight
	-27.00	[-71.15; 17.15]	10.2%
per la construcción de la constr	-28.00	[-70.91; 14.91]	10.4%
	- 95.20	[-324.93; 515.33]	0.2%
	-50.60	[-87.28; -13.92]	11.9%
	11.00	[ 0.43; 21.57	18.1%
	-46.23	[-97.55; 5.09]	8.8%
	-41.60	[-86.22; 3.02]	10.1%
•	-36.25	[-53.61; -18.89]	16.8%
	21.99		
	-20.80	[-42.65; 1.05]	100.0%
0 200 40	0		

Favors Control Favors PCOS

# В

D		PCOS		Co	ntrol				
Author and Year	Total	Mean SD	Total	Mean	SD	Mean Difference	MD	95%-CI	Weight
Álvarez-Blasco et al., 2011	22	6.2 3.4	59	6.1	2.9	-	0.10	[-1.50; 1.70]	10.4%
Cutler et al., 2019	87	2.5 2.5	50	2.2	1.7		0.32	[-0.39; 1.03]	11.3%
Lerchbaum et al., 2021	180	0.8 1.1	150	1.2	0.9	<b>1</b>	-0.48	[-0.70; -0.25]	11.5%
Liang et al., 2020 (lean sub-cohort)	10	0.8 0.6	10	7.7	10.4		-6.97	[-13.44; -0.50]	4.1%
Liang et al., 2020 (overweight sub-cohort)	10	1.1 0.9	10	4.4	5.2	- <u></u>	-3.36	[-6.65; -0.07]	7.9%
Lin et al., 2019	80	6.0 4.5	44	6.0	5.0		0.00	[-1.76; 1.76]	10.2%
Lin et al., 2021	40	7.0 4.1	529	7.4	6.7		-0.40	[-1.79; 0.99]	10.6%
Pourghassem Gargari et al., 2011	30	2.3 2.5	30	9.4	1.4		-7.19	[-8.20; -6.18]	11.0%
Wang et al. 2022	202	0.9 0.6	325	0.9	0.2		0.00	[-0.09; 0.09]	11.5%
Zirak Sharkesh et al. 2021	203	1.6 1.2	291	2.0	1.3	<b>P</b>	-0.37	[-0.59; -0.15]	11.5%
Overall effect	864		1498				-1.44	[-3.38; 0.50]	100.0%
Heterogeneity: $l^2 = 96\%$ , $\tau^2 = 6.1249$ , $\chi_9^2 =$	217.24	(p < 0.001	)						
Test for overall effect: $t_9 = -1.68 (p = 0.13)$						-10 -5 0 5 10			
						Favors Control Favors PCOS			

# С

		PC	OS		Co	ntrol				
Author and Year	Total	Mean	SD	Total	Mean	SD	Mean Difference	MD	95%-CI	Weight
Cutler et al., 2019	87	11.6	4.2	50	13.3	2.7		-1.70	[-2.85; -0.55]	16.2%
Lin et al., 2019	80	17.0	6.7	44	16.0	6.6		1.00	[-1.44; 3.44]	8.8%
Lin et al., 2021	40	20.0	9.2	529	21.7	33.0		-1.70	[-5.70; 2.30]	4.4%
Melekoglu et al., 2020	65	10.6	3.3	65	12.2	4.0	<u> </u>	-1.60	[-2.86; -0.34]	15.5%
Moran et al., 2013	409	12.3	5.4	7057	11.6	4.5		0.70	[0.17; 1.23]	20.1%
Wang et al. 2022	202	14.1	5.8	325	14.8	5.2		-0.62	[-1.60; 0.36]	17.5%
Zirak Sharkesh et al. 2021	203	17.9	5.7	291	17.1	5.2		0.87	[-0.11; 1.85]	17.4%
Overall effect	1086			8361				-0.33	[-1.45; 0.80]	100.0%
Heterogeneity: $I^2 = 78\%$ , $\tau^2$	<sup>2</sup> = 1.08	$337, \chi_6^2 =$	= 26.	90 (p •	< 0.001)	0				
Test for overall effect: $t_6 = -$	-0.71 (	0 = 0.50	)				-4 -2 0 2 4			
							Favors Control Favors PCOS			

# D

			PCOS		C	ontrol				
Author and Year	Total	Mean	SD	Total	Mean	SD	Mean Difference	MD	95%-CI	Weight
Álvarez-Blasco et al., 2011	22	1090.0	503.0	59	1135.0	497.0		-45.00	[-290.48; 200.48]	2.9%
Cutler et al., 2019	87	606.5	281.2	50	637.5	245.9		-31.00	[-121.19; 59.19]	13.2%
Liang et al., 2020 (lean sub-cohort)	10	375.7	287.9	10	524.2	172.2		-148.50	[-356.42; 59.42]	3.8%
Liang et al., 2020 (overweight sub-cohort)	10	481.6	268.2	10	598.5	262.0		-116.90	[-349.31; 115.51]	3.2%
Lin et al., 2019	80	1117.0	531.9	44	1187.0	726.2		-70.00	[-314.18; 174.18]	2.9%
Lin et al., 2021	40	1098.3	520.0	529	1084.4	673.1		13.90	[-157.15; 184.95]	5.4%
Melekoglu et al., 2020	65	527.0	226.8	65	515.5	161.1		11.50	[-56.13; 79.13]	17.6%
Moran et al., 2013	409	874.0	324.0	7057	848.0	288.0	1	26.00	[ -6.11; 58.11]	26.2%
Pourghassem Gargari et al., 2011	30	539.2	185.3	30	712.9	327.9	<u> </u>	-173.64	[-308.42; -38.86]	7.8%
Soodi et al., 2021	203	953.2	402.5	291	993.9	375.5		-40.67	[-110.86; 29.52]	17.0%
<b>Overall effect</b> Heterogeneity: $l^2 = 36\%$ , $\tau^2 = 1622.3937$ , $\tau^2$	<b>956</b> $r^2 = 14$		1 12)	8145				-27.71	[-73.47; 18.05]	100.0%
Test for overall effect: $t_9 = -1.37$ ( $p = 0.20$ )		. 10 (p = 0					-300 -100 0 100 200 300 Favors Control Favors PCOS			

**Figure 7.** Forest plots for micronutrient intake in women with and without PCOS. (A) Folic acid intake expressed as  $\mu g/day$ ; (B) vitamin D intake expressed as  $\mu g/day$ ; (C) iron intake expressed as mg/day; (D) calcium intake expressed as mg/day; (E) magnesium intake expressed as mg/day; (F) zinc intake expressed as mg/day; and (G) sodium intake expressed as mg/day. MD, mean difference.

# Ε

L										
Author and Year	Total	Mean	PCOS	Total N	Con	trol SD	Mean Difference	MD	95%-CI	Waight
Author and fear	Total	wean	50		lean	50	Mean Difference	IVID	95%-CI	weight
Álvarez-Blasco et al., 2011		372.0			61.0 10				[-57.64; 79.64]	4.3%
Cutler et al., 2019		236.7			92.7 15		- <u></u>		[-106.14; -5.86]	6.4%
Douglas et al., 2006		235.9	35.4		56.1 10				[-60.92; 20.52]	7.8%
Liang et al., 2020 (lean sub-cohort)		215.3	41.5		02.6 10				[-154.71; -19.89]	4.4%
Liang et al., 2020 (overweight sub-coho		231.2	51.9		01.5 10				[-143.62; 3.02]	3.9%
Lin et al., 2019		357.0	38.5	44 3		6.7	- <u>i</u>	-18.00	[-39.43; 3.43]	11.5%
Lin et al., 2021		332.2			24.2 14		agest and a		[-45.88; 61.88]	5.9%
Melekoglu et al., 2020 Moran et al., 2013	409	248.2	97.9	7057 2	04.1 12	35.0	and a second sec	-55.90	[-95.00; -16.80] [ 3.73; 24.27]	8.1% 13.4%
Pourghassem Gargari et al., 2011		120.8	35.5			57.8			[-73.72; -18.96]	10.4%
Wang et al. 2022		205.6			17.8 7			-12.22	[-26.42; 1.99]	12.9%
Zirak Sharkesh et al. 2021		400.3			96.5 12			3.80	[-20.10; 27.70]	
							IT			
Overall effect	1188		1	3497		_	<b></b>	-21.46	[-41.01; -1.91]	100.0%
Heterogeneity: $I^2 = 76\%$ , $\tau^2 = 534.7955$	$\chi^2_{11} = 45$	.04 (p <	0.001)					7		
Test for overall effect: $t_{11} = -2.42$ ( $p = 0$	.03)						100 -50 0 50 100			
						Fav	ors Control Favors PCO	S		
F										
1		PCOS	5	Co	ontrol					
Author and Year 7	Total Me	an SD	) Total	Mean	SD	N	lean Difference	MD	95%-CI Wei	ight
							in 1			
Cutler et al., 2019		7.8 3.6		8.7						.9%
Lin et al., 2019		3.0 6.7		13.0						.6%
Lin et al., 2021		4.5 6.3			11.7					.3%
Melekoglu et al., 2020		7.4 2.5		7.7						.7%
Moran et al., 2013		1.2 4.6		10.7					· · · · · · · · · · · · · · · · · · ·	.3%
Pourghassem Gargari et al., 2011		5.4 1.7		7.1		_				.7%
Shahrokhi et al., 2020		7.5 0.7		10.6						.4%
Wang et al. 2022		8.6 3.8		8.3			1 <del>    </del>			.0%
Zaeemzadeh et al., 2018		0.0 4.1		14.2						.2%
Zirak Sharkesh et al. 2021	203 1	1.5 3.8	3 291	11.7	3.7		-	-0.18 [-	0.85; 0.49] 11	.0%
Querrell affect	1007		0540					4 00 5	0 40. 0 001 400	00/
Overall effect Heterogeneity: $l^2 = 96\%$ , $\tau^2 = 2.1272$	1327	0 04 /-	8512					-1.08 [-	2.19; 0.03] 100	.0%
Heterogeneity: $I^2 = 96\%$ , $\tau^2 = 2.127$ .	$2, \chi_{g} = 20$	9.61 (p	< 0.00	)						
Test for overall effect: $t_9 = -2.21$ (p =	= 0.05)					-4	-2 0 2 4			
						Favors	Control Favors PCOS			
C										
G						<b>•</b> • • •				
Author and Year	Tata	l Mear	PCOS	5 ) Total	Mean	Control SD	Mean Difference		MD 9	5%-Cl Weigh
Author and fear	Tota	i wear	1 31	o iotai	wear	50	wean Difference		WD 9	5%-Ci weigh
Álvarez-Blasco et al., 2011	22	2 4699.0	0 2105.0	59	4919.0	2075.0	m	-22	0.00 [-1246.67; 8	306.67] 4.19
Cutler et al., 2019	87	7 2188.0	0 1005.	1 50	2182.1	874.3			5.90 [-315.56; 3	327.36] 12.49
Douglas et al., 2006			3 1183.4			677.1			9.00 [-115.52; 8	
Liang et al., 2020 (lean sub-cohort)			6 1546.			1311.7			2.53 [-1244.15; 12	
Liang et al., 2020 (overweight sub-coh			4 1478.4			1812.1	*		8.11 [-1657.62; 12	
Lin et al., 2019			0 1780.0			1980.0			0.00 [-803.15; 6	
Lin et al., 2021 Moran et al., 2013		2296.0	6 1561.3 0 923.0		2233.0	1530.7 819.0	100		3.80 [-387.32; 6 3.00 [-28.47; 1	
Pourghassem Gargari et al., 2011			2 378.		1727.1				6.93 [-619.80;	
Shishehgar et al., 2016			7 1394.4			1063.4			4.10 [ 594.92; 11	
Zirak Sharkesh et al. 2021			5 1985.			2035.7	+		4.76 [ -84.87; 6	
Overall effect	1063	3		8247			<b>∲</b>	14	8.39 [ -92.70; 3	89.48] 100.0%
Heterogeneity: $l^2 = 73\%$ , $\tau^2 = 97023.31$	135, $\chi_{10}^{2} =$	37.29 (p	< 0.001	)						
Test for overall effect: $t_{10} = 1.37 (p = 0.1)$	20)					-	1500 -500 0 500 10 Favors Control Favors P			
							Favors Control Favors P	005		

### Figure 7. Continued

intakes of fiber in women with PCOS in studies conducted in China (MD: -1.33; 95% Cl: -2.58 to -0.09g/day; P=0.04) and lower alcohol intakes in the older ( $\geq$ 30 years) subgroup (MD: -0.97; 95% Cl: -1.56 to -0.37g/day; P=0.03) (Supplementary Table SIV). No study influenced the overall effect size for protein and fiber intakes, revealed by sensitivity analyses. However, removing studies by (Álvarez-Blasco et al., 2011) (MD: -0.99; 95% Cl: -2.01 to 0.03g/day; P=0.05), (Moran et al., 2013) (MD: -0.96; 95% Cl: -2.08 to 0.15g/day; P=0.07), and (Navarro-Lafuente et al., 2022) (MD: -0.88; 95% Cl: -2.30 to 0.54g/day; P=0.14) resulted in the loss of differences in alcohol intakes. We observed no evidence of publication bias for protein, fiber, and alcohol (funnel plots, Supplementary Fig. S9A–C; All  $P \ge 0.06$ , Begg's tests; All  $P \ge 0.11$ , Egger's tests).

Folic acid, vitamin D, iron, calcium, magnesium, zinc sodium. Figure 7A–G demonstrates pooling data for select *a priori* micronutrient intake. Women with PCOS had comparable intakes of folic acid (MD: -20.80, 95% Cl: -42.65 to  $-1.05 \,\mu$ g/day; P=0.06; Fig. 7A; N=9) (Pourghassem Gargari et al., 2011; Moran et al., 2013; Cutler et al., 2019; Lin et al., 2019; Melekoglu et al., 2020; Lin et al., 2021;

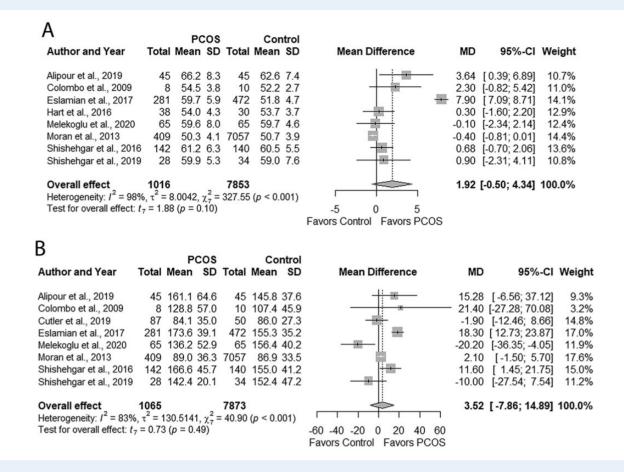


Figure 8. Forest plots for status of dietary glycemic indices in women with and without PCOS. (A) glycemic index and (B) glycemic load. MD, mean difference.

Szczuko et al., 2021; Zirak Sharkesh et al., 2021; Wang et al., 2022); vitamin D (MD: -1.44, 95% CI: -3.38 to  $0.50 \,\mu$ g/day; Fig. 7B; N = 9) (Álvarez-Blasco et al., 2011; Pourghassem Gargari et al., 2011; Cutler et al., 2019; Lin et al., 2019; Lerchbaum et al., 2021; Liang et al., 2021; Lin et al., 2021; Zirak Sharkesh et al., 2021; Wang et al., 2022); iron (MD: -0.33, 95% CI: -1.45 to 0.80 mg/day; Fig. 7C; N = 7) (Moran et al., 2013; Cutler et al., 2019; Lin et al., 2019; Melekoglu et al., 2020; Lin et al., 2021; Zirak Sharkesh et al., 2021; Wang et al., 2022); calcium (MD: -27.71, 95% CI: -73.47 to 18.05 mg/day; Fig. 7D; N = 8) (Alvarez-Blasco et al., 2011; Pourghassem Gargari et al., 2011; Moran et al., 2013; Cutler et al., 2019; Lin et al., 2019; Melekoglu et al., 2020; Lin et al., 2021; Soodi et al., 2021); zinc (MD: -1.08, 95% CI: -2.19 to 0.03 mg/day; P = 0.05; Fig. 7F; N = 10) (Pourghassem Gargari et al., 2011; Moran et al., 2013; Zaimzadeh et al., 2018; Cutler et al., 2019; Lin et al., 2019; Melekoglu et al., 2020; Shahrokhi and Naeini, 2020; Lin et al., 2021; Zirak Sharkesh et al., 2021; Wang et al., 2022); or sodium (MD: 148.39, 95% CI: -92.70 to 389.48 mg/day; Fig. 7G; N = 10 (Douglas et al., 2006; Álvarez-Blasco et al., 2011; Pourghassem Gargari et al., 2011; Moran et al., 2013; Shishehgar et al., 2016a; Cutler et al., 2019; Lin et al., 2019; Liang et al., 2021; Lin et al., 2021; Zirak Sharkesh et al., 2021) intakes versus Controls (All  $P \ge 0.05$ ). In contrast, women with PCOS exhibited lower magnesium intakes (MD: -21.46, 95% CI: -41.03 to -1.91 mg/day; P=0.03

Fig. 7E; N = 11) (Douglas et al., 2006; Álvarez-Blasco et al., 2011; Pourghassem Gargari et al., 2011; Moran et al., 2013; Cutler et al., 2019; Lin et al., 2019; Melekoglu et al., 2020; Liang et al., 2021; Lin et al., 2021; Zirak Sharkesh et al., 2021; Wang et al., 2022). There was high heterogeneity across all studies that reported micronutrient intake data (all  $l^2 \ge 76\%$ ; All P < 0.001) except homogenous studies reporting calcium intake ( $l^2 = 36\%$ ; P = 0.12).

Subgroup analyses based on age, PCOS criteria, dietary assessment tool, or country did not explain heterogeneity for all micronutrients, except folic acid and magnesium. Lower intakes of folic acid (MD, -41.77; 95% CI: -65.33 to -18.21 µg/day; P=0.01) and magnesium (MD: -38.98; 95% CI: -69.44 to -8.53 mg/day; P=0.03) were evident in PCOS versus Control subgroups who were younger (<30 years) and that used Rotterdam criteria. Also, lower intakes of magnesium were evident in the subgroups who were leaner (BMI < 30 kg/m<sup>2</sup>; MD: -33.33; 95% CI: -63.45 to -3.21 mg/day; P = 0.03) and where dietary intakes were assessed using the 24-hour recalls (MD: -54.44; 95% CI: -78.71 to -30.17 mg/day; P < 0.01). Regarding other dietary assessment tools, PCOS groups showed lower intakes of vitamin D (MD: -0.36; 95% CI: -0.59 to  $-0.12 \,\mu g/day$ ; P = 0.02) and higher intakes of iron (MD: 0.75; 95% CI: 0.49 to 1.01 mg/day; P < 0.01) where FFQ was used (Supplementary Table SIV). None of the individual studies influenced the overall effect size for vitamin D,

iron, calcium and sodium, evidenced by sensitivity analyses. In contrast, removing the study by (Moran et al., 2013) (MD: -27.63; 95% CI: -49.58 to -5.67 mg/day; P = 0.02) led to significant differences between groups for folic acid. This observation was similar to removing certain individual studies for zinc intake: ((Moran et al., 2013) (MD: -1.29, 95% CI: -2.46 to -0.11 mg/day; P = 0.04) and (Wang et al., 2022) (MD: -1.26, 95% CI: -2.46 to -0.06 mg/day; P = 0.04). Conversely, removing studies by Cutler et al. (2019) (MD: -18.94, 95% CI: 39.27 to 1.40 mg/day; P=0.06), Liang et al. (2021) (lean subcohort, MD: -17.97, 95% CI: 36.43 to 0.50 mg/day; P=0.06) (overweight sub-cohort, MD: -19.32, 95% CI: 39.21 to 0.57 mg/day; P=0.06), Melekoglu et al. (2020) (MD: -17.94, 95% CI: 38.01 to 2.13 mg/day; P=0.06) and Pourghassem Gargari et al. (2011) (MD: -18.28, 95% CI: 39.15 to 2.60 mg/day; P = 0.06) from magnesium pooled analyses resulted in the loss of differences between groups, albeit the direction of effect estimates remained unchanged. We observed no evidence of publication bias for folic acid, vitamin D, iron and sodium (funnel plot, Supplementary Fig. S10A, B, C and F; all  $P \ge 0.45$ , Begg's tests; All  $P \ge 0.15$ , Egger's tests). However, publication bias was detected for calcium and magnesium (funnel plot, Supplementary Fig. 10D and E, respectively) evidenced by the Egger's test results (All  $P \le 0.02$ ) unlike the Begg's tests results (all P > 0.53).

**GI and GL.** Groups had comparable GI (MD: 1.92, 95% CI: -0.50 to 4.34; P = 0.10; Fig. 8A; N = 8) (Colombo et al., 2009; Moran et al., 2013; Hart et al., 2016; Shishehgar et al., 2016a; Eslamian et al., 2017; Alipour et al., 2019; Shishehgar et al., 2019; Melekoglu et al., 2020) and GL (MD: 3.52, 95% CI: -7.68 to 14.89; P = 0.49; Fig. 8B; N = 8) (Colombo et al., 2009; Moran et al., 2013; Shishehgar et al., 2016a; Eslamian et al., 2017; Alipour et al., 2019; Cutler et al., 2016a; Eslamian et al., 2017; Alipour et al., 2019; Cutler et al., 2019; Shishehgar et al., 2019; Melekoglu et al., 2020). Studies were moderately heterogeneous ( $l^2 = 41\%$ ; P = 0.12).

Subgroup analyses based on age, PCOS criteria, or dietary assessment tools did not explain heterogeneity (Supplementary Table SIV). Subgroup analyses could not be undertaken for BMI or country due to an insufficient number (<2) of studies. None of the individual studies influenced the overall effect sizes, evidenced by sensitivity analyses. We observed no evidence of publication bias (funnel plots, Supplementary Fig. SI I A and B; all  $P \ge 0.32$ , Begg's test; All  $P \ge 0.60$ , Egger's test).

# Discussion

### Main findings

To our knowledge, this is the first systematic review and meta-analysis to synthesize evidence on lifestyle behaviors in women with PCOS. The most significant results of this comprehensive review of 54 observational studies involving 39471 reproductive-aged women are that women with PCOS exhibit an overall adverse lifestyle behavior, specifically poorer dietary intakes (lower diet quality, higher cholesterol, lower magnesium, tendency for lower zinc), and lower total PA compared to those without PCOS, despite lower alcohol intakes. We also observed worse or similar consumption of core food groups (grains, fruits, vegetables, proteins, seeds, nuts, dairy) for women with PCOS. On subgroup analyses, higher energy and lower fiber intakes were evident in the PCOS groups from select countries, including Iran and China, respectively. Also, lower folic acid intakes were noted in

younger (<30 years) PCOS populations. Higher total fat and PUFA intakes were evident in PCOS when food records were used, whereas lower vitamin D and higher iron intakes were observed in PCOS when FFQs were used. Women with PCOS had higher PUFA when PCOS was diagnosed by the AEPCOS criteria. Collectively, these data support the likelihood of poorer lifestyle behaviors in women with PCOS.

### **Comparison with other studies**

Our observations add a novel dimension to current evidence and align with previous systematic reviews and meta-analyses on the relation between poor diet and/or lack of exercise and higher prevalence of chronic disorders with pathophysiologic underpinning similar to PCOS, including gestational diabetes (Mijatovic-Vukas et al., 2018), type 2 diabetes (Lee et al., 2017; Schwingshackl et al., 2018), obesity (de Menezes et al., 2019; Syngelaki et al., 2019) or longitudinal weight gain (Tobias et al., 2015), infertility (Best et al., 2017) and cardiovascular disease (Liyanage et al., 2016). While meta-analyses on micronutrient status in PCOS are sparse, our observations of higher cholesterol and lower magnesium intakes corroborate systematic reviews and metaanalyses reporting higher serum total cholesterol (Wekker et al., 2020) and lower serum magnesium (Babapour et al., 2021) concentrations in PCOS versus Controls. Hypercholesterolemia has been implicated in the development of cardiometabolic and reproductive disruptions, including type 2 diabetes and hyperandrogenemia in PCOS, as elaborated in previous reviews (Wild et al., 2010; Wekker et al., 2020). Magnesium insufficiency has been implicated in the development of IR, or impaired glucose tolerance in PCOS (Babapour et al., 2021), consistent with emerging hypotheses that altered trace mineral status may play a role in PCOS pathogenesis. Furthermore, our observation of lower dietary vitamin D and higher iron intakes in reproductive-aged women with PCOS was consistent with the results of systematic reviews and meta-analyses reporting lower serum vitamin D (Bacopoulou et al., 2017) and higher ferritin (a cellular biomarker of iron storage) (Yin et al., 2020) concentrations in this clinical population. Vitamin D deficiency has been associated with impaired glycemic, hormonal, ovulatory, oxidative and inflammatory status beyond known risks for bone metabolism in PCOS (Nandi et al., 2016; Di Bari et al., 2021; Zhao et al., 2021). Similarly, elevated iron levels in PCOS may be associated with metabolic complications, including IR and adiposity (Yin et al., 2020), albeit data are sparse, warranting further research.

Our results also corroborate those of previous systematic reviews and meta-analyses of no relations between select macronutrients (e.g. total carbohydrate (Liu et al., 2021), total protein (Alhazmi et al., 2012), total fat (Zhu et al., 2019), SFA (de Souza et al., 2015; Zhu et al., 2019), MUFA (Zhu et al., 2019), micronutrients (folic acid (Heinz et al., 2009), calcium (Chung et al., 2016), sodium (Milajerdi et al., 2019), GI (Mulholland et al., 2009; Nagle et al., 2013) or GL (Mulholland et al., 2009; Turati et al., 2019) and chronic disease risk in non-PCOS populations, albeit, contradictory findings exist (Pittas et al., 2007; Meng et al., 2017; Wang et al., 2017; Khan et al., 2018; Zhao et al., 2020)). Small sample sizes and numbers of eligible studies to evaluate some outcome measures (e.g. four studies for added sugar) likely lowered our statistical power to observe real differences. Therefore, further research is needed to confirm our observations for select dietary components.

### **Explanation of results**

The mechanisms through which dietary and PA behaviors may be suboptimal in PCOS are unknown, yet complex interactions between genetic predisposition and environmental determinants may be at play. We and others have attributed an overall lower diet quality, poorer consumption of certain food groups and/or nutrients to physiological (e.g. appetite regulation factors) or psychosocial factors (Robinson et al., 1992; Moran et al., 2004; Barry et al., 2011). Accordingly, we hypothesize that suboptimal lifestyle habits of women with PCOS may be, in part, attributed to disrupting factors including: circadian rhythm (Moore et al., 2021); appetite regulation (Romualdi et al., 2018); energy expenditure (Franks et al., 1996); gut microbiota (Guo et al., 2021); psychosomatic factors (e.g. depression, anxiety) (Barber et al., 2019); lack of education about healthy lifestyle (Steegers-Theunissen et al., 2020); and/or lack of appropriate healthcare access tailored to the needs of this clinical cohort, especially for long-term monitoring (Kazemi et al., 2019c). Presently, the relative contributions of these individual factors to PCOS lifestyle behaviors are unknown, pointing to a persistent research gap.

### Strengths and limitations

Strengths of our study include a comprehensive search strategy, extensive outcome measures to assess dietary and PA status, inclusion of a considerable pool of studies and application of conservative statistical methods to interpret findings. We observed no evidence of substantial publication bias in the evaluated outcomes, as evidenced by the Begg's and Egger's test results, except for calcium and magnesium intakes. Nevertheless, less symmetric funnel plots observed in select nutrients (e.g. energy, fiber, vitamin D) likely point to publication bias or a difference between studies of higher and lower precision (e.g. small study effects) (Sterne et al., 2011). Our observations had limitations inherent to the sample sizes of some eligible studies and small numbers of studies included for select outcomes. Therefore, our results may be interpreted with caution. The lack of a universal definition for diet quality, dietary and PA assessment tools, and PCOS criteria are also limitations corroborated in previous systematic reviews and meta-analyses of this type (Harrison et al., 2011; Lim et al., 2019; Jalili et al., 2020; Kazemi et al., 2020a,d, 2021a). Failure to account for variations in age, adiposity, lifestyle, socioeconomic status, race and ethnicity, acculturation status, the proportion of PCOS phenotypes across studies, the use of retrospective data or data primarily collected from clinical settings may have contributed to heterogeneity in our observations (Moran et al., 2015; Kazemi et al., 2021a,b,c). Most studies were conducted in medical centers (41/45; 91%) and consisted of cohorts presenting with overweight/obesity (31/43; 73%), indicating our results may be skewed toward severe clinical phenotypes (Ezeh et al., 2013; Kazemi et al., 2019d). Our study was limited by disordered eating or the inability to account for eating disorders that could influence evaluated outcomes. We and others have shown a higher prevalence of disordered eating or eating disorders, including binge-eating disorder, in PCOS (Naessén et al., 2019; Pirotta et al., 2019; Tay et al., 2019b), attributed to obesity, warranting surveillance and management by dietitians and allied health providers (Pirotta et al., 2019). Furthermore, we sumption or supplemen

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were unable to compare dietary inositol consumption or supplementation between women with and without PCOS owing to lack of data. However, we recognize a proposed role for inositol (a natural sugaralcohol) in the management of cardiometabolic and reproductive deregulation in PCOS (Artini *et al.*, 2018; Facchinetti *et al.*, 2020) and note that our findings of poorer intakes of core food groups that contain inositol, including beans, whole grains, nuts, and seeds may signal lower inositol intakes in PCOS.

We observed instability in the significance of the pooled effect estimates with the removal of single studies during sensitivity analyses resulting in the loss of significance for PA, alcohol and magnesium and gaining significance for energy (higher), folic acid and zinc (lower), supporting the need for more research to confirm our observations. Overall, our subgroup results based on established confounders (e.g. age, BMI, dietary and PA assessment tool, PCOS diagnostic criteria, country) did not reveal the sources of heterogeneity across all measures. This was unsurprising given that all women included in the present work were relatively homogenous being of reproductive age (21.0-48.2 years), mostly defined using the Rotterdam criteria (29/45. 64%) and presented with overweight/obesity in medical centers (41/ 45; 91%), making it challenging to conduct more discrete subgroup analyses (e.g. self-reported definition of PCOS). Nonetheless, our observations of less favorable dietary intakes in certain subgroups, including lower magnesium and folic acid intakes in younger women or higher total fat and PUFA intakes captured by food record tools, may have implications for the dietary management of young at-risk women during their early reproductive stages or highlight the utility of a food record to more accurately capture differences in dietary behaviors (Thompson et al., 2015). Additionally, providing macronutrient intakes using %energy versus g/day may have better reflected individual intakes and subsequently capture any real difference between groups based on acceptable macronutrient distribution range. Furthermore, we considered performing additional subgroup analyses to account for race, ethnicity, previous knowledge of having PCOS, basal metabolic rate, tobacco use, medication use (metformin, hormonal contraceptives), supplements, inflammatory status, reproductive hormones or type of biochemical assays used to measure total testosterone, including liquid chromatography-mass spectrometry. Unfortunately, we lacked sufficient or no data for these analyses, limiting our abilities to understand where real differences lie, which is not uncommon in studies of this type (Gasevic et al., 2015; Kakoly et al., 2018; Rich et al., 2018; Babapour et al., 2021; Hadi et al., 2021). Particularly, reverse causation (improved lifestyle behaviors following PCOS diagnosis) is a significant confounder that has been poorly addressed in PCOS lifestyle research owing to the lack of longitudinal data.

### **Recommendations for further research**

Several questions remain unanswered about which, why, and how dietary and PA behaviors differ in women with PCOS versus Controls, as current evidence on any underlying mechanisms of these differences is sparse and contradictory, making any robust conclusions impossible. Clarifying the role of factors that contribute to adverse lifestyle behaviors in women with PCOS, including abnormalities in appetite regulation or energy expenditure, body composition, genetic and sociodemographic status, with reliable and reproducible tools is needed for both short- and long-term success of lifestyle intervention in this high-risk population (Hoeger et al., 2004; Marsh et al., 2010; Ladson et al., 2011a; Kazemi et al., 2019a,c; 2021c). Filling these knowledge gaps across various reproductive life stages, including puberty, pregnancy, and menopause, and the phenotypic spectrum of PCOS (Rotterdam ESHRE/ASRM-sponsored PCOS Consensus Workshop Group 2004) with variable degrees of metabolic disturbances and body composition alterations is a priority. Namely, elucidating any suboptimal lifestyle behaviors of pregnant women with PCOS is of particular clinical relevance (Chiu et al., 2018) yet remains poorly studied. Also, research should delineate whether correction of the suboptimal dietary patterns and nutrient intakes and sedentary behaviors identified herein would lead to clinically meaningful improvements in patient-pressing complications, including obesity, infertility and type 2 diabetes in PCOS. These clarifications are critical to elucidate the effectiveness of personalized lifestyle management strategies to improve the cardiometabolic, reproductive, and psychological health of this high-risk cohort. This is in keeping with the emerging era of precision lifestyle medicine in investigating innovative management strategies in other chronic diseases, including type 2 diabetes or cardiovascular disease.

### Implications for clinical practice

Our observations have implications for allied healthcare providers (dietitians and exercise physiologists) and physicians to prioritize the identification of suboptimal dietary and PA behaviors in women with PCOS and to guide evidence-based lifestyle management for this prevalent and at-risk population. These findings highlight the importance of early lifestyle intervention at the time of PCOS diagnosis to address modifiable extrinsic factors that can prevent or minimize longitudinal weight gain and associated health complications (Awoke et al., 2021). Provider recommendations should target meeting daily energy intake requirements and adequate consumption of select nutrients (magnesium, vitamin D) and core foods (whole grains, seafood, fish, plant proteins [pulses], nuts, seeds, low-fat dairy) to achieve and maintain optimal health, healthy body weight and prevent long-term weight gain. These recommendations are prudent as we and others have shown PCOS cohorts exhibit poor adherence to energy-restricted diets (Hoeger et al., 2004; Ladson et al., 2011b; Lin et al., 2014; Turner-McGrievy et al., 2014), a propensity for obesity (Kazemi et al., 2018b; Awoke et al., 2021), perception of an inevitability for weight gain (Lin and Lujan, 2014; Lin et al., 2017; Kazemi et al., 2019c), and higher longitudinal weight gain (Teede et al., 2013; Kazemi et al., 2018a; Awoke et al., 2021). Providers may also benefit from improving their ability to work with women with PCOS to improve the lifestyle behaviors of this clinical cohort (Lin et al., 2017; Kazemi et al., 2019c, 2021c).

# Conclusion

Collective evidence supports that women with PCOS have lower overall diet quality, poorer dietary intakes (higher cholesterol, lower magnesium and zinc) and lower total PA compared to those without PCOS. Given the observational nature of included studies, we cannot infer causality. Heterogeneity among studies reinforces the need for research to delineate any relative contributions of other factors (genetic, metabolic, sociodemographic) to the observed differences in the era of precision lifestyle medicine. Our findings highlight that providing education on lifestyle modification is crucial for women with PCOS to improve their short- and long-term reproductive, metabolic, and psychological health.

## Supplementary data

Supplementary data are available at Human Reproduction Update online.

# Data availability

The data underlying this article will be shared on reasonable request to the corresponding authors.

# **Authors' roles**

The authors' contributions were the following: M.K. conceived the topic. M.K. designed the review question, and study with contributions from M.E.L. and L.J.M. M.K. and L.J.M. designed the search strategy and performed searches on the bibliographic databases. C.W., J.D.X., I.Y.K., I.M., I.B.X., K.G. and M.K. screened and reviewed the literature. C.T.T., J.A.G., I.B.X., K.G., J.M., C.W., J.D.X., J.Y.K. and M.K. completed data extraction. C.W., J.D.X., J.Y.K., J.M., I.B.X., K.G. and M.K. conducted the quality assessments of study methods. M.K. resolved all disagreements related to the literature review, data extraction, quality assessment and critical review of outcome data with contributions from M.E.L. and L.J.M. M.K. performed the statistical analyses with contributions from S.A.P. M.K. interpreted the results and wrote the manuscript. All authors reviewed and approved the final version of the manuscript and contributed to the scientific review of study results. The authors are responsible for the study design and conception, data collection and analysis, decision to publish and manuscript preparation. M.K. and M.E.L. supervised the study and had primary responsibility for the final content.

# Funding

Funds from the Division of Nutritional Sciences at Cornell University and the National Institutes of Health (Grant No. R01-HD0937848) were used to support the authors (M.E.L. and M.K.) throughout the study period and manuscript preparation. L.J.M. was funded by a National Heart Foundation Future Leader Fellowship. J.A.G. was funded by a National Health and Medical Research Council Ideas Grant (GNT 2000905). M.K. was also funded by the Canadian Institutes of Health Research Postdoctoral Fellowship (No. 459075). The funders had no role in the study design, collection, analyses, interpretation of data, writing of the manuscript or decision to publish.

# **Conflict of interest**

The authors confirm no conflict of interest would confound the proposed study or cause any adverse effect upon past or subsequent interaction with any study participants.

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