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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

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TABLE OF CONTENTS

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% CIs. The risk of bias was assessed by the Newcastle-Ottawa scale (NOS).

OUTCOMES: Fifty-four studies (N = 39 471 participants; [n = 8736 PCOS; 30 735 Controls]) were eligible (96%; [52/54] NOS scores ≥ 7). Women with PCOS had higher cholesterol (MD: 12.78, 95% CI: 1.48 to 24.08 mg/day; $P = 0.03$; $I^2 = 19\%$), lower magnesium (MD: −21.46, 95% CI: −41.03 to −1.91 mg/day; $P = 0.03$; $I^2 = 76\%$), and a tendency for lower zinc (MD: −1.08, 95% CI: −2.19 to 0.03 mg/day; $P = 0.05$; $I^2 = 96\%$) intake, despite lower alcohol consumption (MD: −0.95, 95% CI: −1.67 to −0.22 g/day; $P = 0.02$; $I^2 = 0\%$) versus Controls. Also, women with PCOS had lower total PA (standardized mean difference: −0.38, 95% CI: −0.72 to −0.03; $P = 0.03$; $I^2 = 98\%$). Conversely, energy, macronutrients (carbohydrate, fat, protein, fiber), micronutrients (folic acid, iron, calcium, sodium), glycemic index and glycemic load were similar (all: $P \geq 0.06$). 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 meta-analysis 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 I^2 statistics were reported. The I^2 value describing the percentage variation between studies was calculated as $100\% \times ([Q - df])/Q$, Q being the χ^2 value, and df corresponding to degrees of freedom. Low, moderate and high heterogeneity were defined using the I^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 ≥30 years); BMI (<30 or ≥30 kg/m^2); 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 devised surveys); and groups' country of origin (categorical) on study outcomes where sufficient data were available (≥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 16 370 records (Fig. 1), of which 54 studies (61 publications) were deemed eligible and included. The selected studies comprised a total of 39 471 participants and 116 experimental arms (n = 8736 PCOS [59 arms]; 30 735 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; Panjeshahin *et al.*, 2020; Shahrokhi and Naeini, 2020; Badri-Fariman *et al.*, 2021; Noormohammadi *et al.*, 2021; Soodi *et al.*, 2021; Zirak Sharkesh *et al.*, 2021), Italy (Orio *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; Navarro-Lafuente *et al.*, 2022), Australia (Thomson *et al.*, 2009; Moran *et al.*,

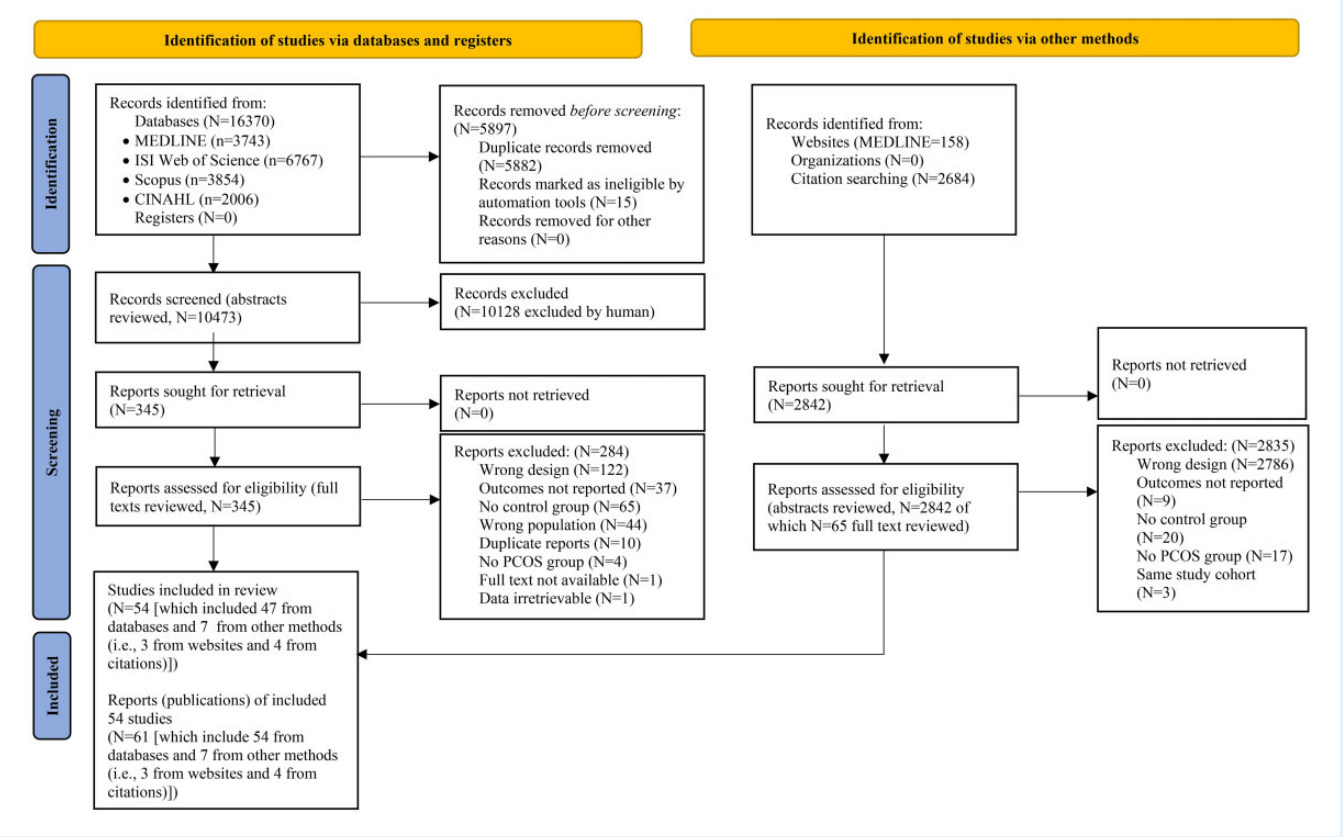

Figure 1. PRISMA 2020 flow diagram for new systematic reviews, including searches of databases, registers and other sources.

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^2, 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

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

Author, yrs. (reference), and country	Participants' characteristics (n, mean age [yrs.], mean BMI [kg/m^2]), PCOS definition, and racial/ethnic composition	Study design, setting, data analysis	Dietary/PA assessment tool	Reported outcomes of interest
Alipour *et al.*, 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	• **Design:** Case-control • **Setting:** AMC • **Analysis:** Prospective	3-d 24-h food recall (2 weekdays and 1 weekend d) **PA:** NR	**Diet:** • ***a priori* diet outcomes:** ↔E, ↑total CHO, ↑starch, ↔total F, ↔total P, ↔total fiber, ↑GI, ↔GL, ↔total sugar • **Other diet outcomes:** NR **PA:** • ***a priori* PA outcomes:** NR • **Other PA outcomes:** NR
Altieri *et al.*, 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	**Diet:** • ***a priori* diet outcomes:** ↔E, ↔total CHO (%E and g), mono- and ↔oligosaccharides (total and oligo-rich 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 • **Other diet outcomes:** ↔glycosylated end products, ↑starchy sweet with high GI, ↔starchy food (LGI or HGI), ↔ice cream, ↔chocolate, ↔egg, ↑oil, ↓cooking fat, ↔wine, ↔beer **PA:** • ***a priori* PA outcomes:** NR • **Other PA outcomes:** NR
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	**Design:** Case-control **Setting:** AMC **Analysis:** Prospective	**Diet:** Semi-quantitative FFQ (modified, Harvard Service FFQ for Spanish population) **PA:** Self-reported questionnaire (unspecified)	**Diet:** • ***a priori* diet outcomes:** ↔E, ↔total CHO (%E and g), ↔total F (%E and g), ↔Chol, ↔SFA (%E and g), ↔MUFA (%E and g), ↔PUFA (%E and g), ↔trans F, ↔total P (%E and g), ↔total fiber, ↔alcohol, ↔Vit D, ↔Ca, ↔Mg, ↔Na • **Other diet outcomes:** ↔n-3, ↔n-6, ↔Vit C, ↔Vit E, ↔K, ↔caffeine

Continued

Table I Continued

Author, yrs. (reference), and country	Participants' characteristics (n, mean age [yrs.], mean BMI [kg/m^2]), PCOS definition, and racial/ethnic composition	Study design, setting, data analysis	Dietary/PA assessment tool	Reported outcomes of interest
				PA: • ***a priori* PA outcomes:** ↔PA (%women exercising regularly in hrs/wk) • **Other PA outcomes:** NR
Badri-Fariman *et al.*, 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 questionnaire (Momenan *et al.*, 2012) (validated, consisted of 9 different MET levels and 3 categories of PA)	**Diet:** • ***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 • **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-tocopherol, ↓Ph, ↓Cu, ↓K, ↔Se, ↔Mn, ↔Chromium, ↓egg, ↑caffeine, ↑ tea and coffee, ↑SSB, ↔French fries and potato chips, ↔pickles and salinity, ↔liquid oils, ↓tomato, ↑salt, ↑sugar, ↑sweets, ↑desserts, ↑industrial juice and soft drinks ↓docosahexaenoic acid, ↔linoleic acid, ↔linolenic acid, ↔eicosapentaenoic acid, ↑oleic acid, ↓beta-cryptoxanthin, ↓beta-carotene, ↓alpha-carotene, ↓lutein, ↓lycopene, ↓glucose, ↓galactose, ↓fructose, ↓lactose, ↔maltose, ↑sucrose, ↑fluorine **PA:** • ***a priori* PA outcomes:** ↓PA (MET-hr, ↑PCOS risk), ↓MET-hr (rest or sleep) • **Other PA outcomes:** NR

Continued

Table I Continued

Author, yrs. (reference), and country	Participants' characteristics (n, mean age [yrs.], mean BMI [kg/m^2]), PCOS definition, and racial/ethnic composition	Study design, setting, data analysis	Dietary/PA assessment tool	Reported outcomes of interest
Banting *et al.*, 2014, 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	**Design:** Cross-sectional **Setting:** Community setting **Analysis:** 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:** ↓PA (%women physically active)
Barrea *et al.*, 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 assessment and 7-d food diary for dietary intake assessment (nutritionist-administered by face-to-face interviews) **PA:** Questionnaire (self-reported habitual aerobic exercise engagement for a minimum of 30 min/d [yes/no]) (Barrea *et al.*, 2018)	**Diet:** • ***a priori* 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 fiber, ↔total fruit ≥3 servings/wk, ↔total vegetable ≥2 servings/wk, ↔%red or processed meat < 1 serving/d, ↔%poultry more than red meat, ↓%fish/seafood ≥3 servings/wk, ↓%legume ≥3 servings/wk, ↓%tree nuts ≥3/wk, total sugar, ↔%carbonated < 1/d SSB • **Other diet outcomes:** ↑%Low adherence to MED, ↔%average adherence to MED, ↔%high adherence MED, ↓%use of extra virgin oil as main culinary lipid, ↔%extra virgin oil > 4 tablespoons, ↔%butter, cream, margarine < 1 serving/d, ↔wine glasses ≥ 7/wk, ↔%commercial sweets and confectionary ≤2/wk, ↔%use of sofrito sauce ≥2 servings/wk, ↓unsaturated fat **PA:** • ***a priori* PA outcomes:** NR • **Other PA outcomes:** ↔%Moderate PA, ↔%sedentary behaviors

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Author, yrs. (reference), and country	Participants' characteristics (n, mean age [yrs.], mean BMI [kg/m^2]), PCOS definition, and racial/ethnic composition	Study design, setting, data analysis	Dietary/PA assessment tool	Reported outcomes of interest
Colombo *et al.*, 2009, Italy	**Total**, 18 (PCOS, 8; functional hypothalamic amenorrhea, 10) **PCOS**, age: 21.0, BMI: 24.3 **Control**, age: 26.0, BMI: 19.9 **PCOS definition**, AEPCOS **Ethnic composition**, White	**Design:** Cross-sectional **Setting:** AMC **Analysis:** Prospective	**Diet:** 188-item FFQ (European Prospective Investigation into Cancer and Nutrition) (Pisani *et al.*, 1997; Pala *et al.*, 2003) and 7-d food diary **PA:** NR	**Diet:** • ***a priori* 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), ↔Chol (mg and mg/FFMkg/d), ↑SFA (g and g/FFMkg/d), ↔MUFA (g and g/FFMkg/d), ↔PUFA (g and g/FFMkg/d), ↔total P (g and %E and g/FFMkg/d), ↔total fiber, ↔alcohol, ↔GI, ↔GL • **Other diet outcomes:** ↑Animal fat, ↔soluble sugar, ↔water **PA:** • ***a priori* PA outcomes:** NR • **Other PA outcomes:** NR
Copp *et al.*, 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	**Design:** Cross-sectional analysis of ALSWH Study **Setting:** AMC **Analysis:** Prospective	**Diet:** Survey to assess vegetable intake (non-validated, self-reported) **PA:** Active Australia Survey (Brown *et al.*, 2008; Fjeldsoe *et al.*, 2013) (self-reported)	**Diet:** • ***a priori* diet outcomes:** ↔Total vegetable • **Other diet outcomes:** NR **PA:** • ***a priori* PA outcomes:** ↔Total PA time or intensity • **Other PA outcomes:** NR
Cunha *et al.*, 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	**Diet:** 7-d food report **PA:** NR	**Diet:** • ***a priori* 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) • **Other diet outcomes:** NR **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^2]), PCOS definition, and racial/ethnic composition	Study design, setting, data analysis	Dietary/PA assessment tool	Reported outcomes of interest
Cutillas-Tolín *et al.*, 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	**Diet:** • ***a priori* diet outcomes:** ↑AHEI-2010 associated with ↓risk of PCOS HA-OA but ↔Other dietary patterns (AHEI, rMED, aMED, DASH) and PCOS risk • **Other diet outcomes:** NR **PA:** • ***a priori* PA outcomes:** NR • **Other PA outcomes:** NR
Cutler *et al.*, 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	**Design:** Cross-sectional **Setting:** AMC **Analysis:** Prospective	**Diet:** 3-d food record (2 weekdays, 1 weekend d) **PA:** 3-d PA record (2 weekdays, 1 weekend d) and pedometer (SM-2000 Step Pedometer by Heart Rate Monitors USA) for steps/d	**Diet:** • ***a priori* diet outcomes:** ↔E, ↔total CHO, ↔total F, ↔Chol, ↔total P, ↓total fiber, ↔Vit B9, ↔Vit D, ↓Fe (only crude but ↔in adjusted models for E), ↔Ca, ↓Mg, ↔Zn, ↔Na, ↔GL • **Other diet outcomes:** ↓Vit A, ↔Vit C **PA:** • ***a priori* PA outcomes:** NR • **Other PA outcomes:** ↔steps
Dantas *et al.*, 2015, Brazil	**Total**, 27 (PCOS, 15; Control, 12) **PCOS**, age: 24.8, BMI: 32.8 **Control**, age: 29.6, BMI: 30.3 **PCOS definition**, Rotterdam	**Design:** NR **Setting:** AMC **Analysis:** Prospective	**Diet:** NR **PA:** Accelerometer (Actigraph accelerometer) with Freedson activity cut	**Diet:** • ***a priori* diet outcomes:** NR • **Other diet outcomes:** NR

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Author, yrs. (reference), and country	Participants' characteristics (n, mean age [yrs.], mean BMI [kg/m^2]), 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 intensity (Freedson *et al.*, 1998)	**PA:** • ***a priori* PA outcomes:** NR • **Other PA outcomes:** ↔Total steps/d
Douglas *et al.*, 2006, USA	**Total**, 57 (PCOS, 30; Control, 27) **PCOS**, age: 28.9, BMI 29.7 **Control**, age: 28.9, BMI 29.1 **PCOS definition**, NIH **Ethnic composition**, PCOS (83.3% White, 13.3% Black, 3.3% Other); Controls (85.1% White, 11.1% Black, 3.7% Other)	**Design:** Baseline analysis of a cohort study **Setting:** AMC **Analysis:** Prospective	**Diet:** 4-d food record (2 weekdays and 2 weekends, self-reported) and multiple-choice food questionnaire (unspecified, self-reported) **PA:** NR	**Diet:** • ***a priori* diet outcomes:** ↔E, ↔total CHO, ↔total F, ↔Chol, ↔SFA, ↔MUFA, ↔PUFA, ↔trans F, ↔total P, ↔total fiber, ↔soluble fiber, ↔Mg, ↔Na • **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) **PA:** • ***a priori* PA outcomes:** NR • **Other PA outcomes:** ↔Exercise sessions (frequency)
Eslamian *et al.*, 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 (outpatient clinics) **Analysis:** Prospective	**Diet:** Semi-quantitative FFQ for Iranians (usual dietary intake in past 1 yr., validated) **PA:** Questionnaire (unspecified and validated, consisted of 9 different MET categories ranging from sleep to high intensity)	**Diet:** • ***a priori* diet outcomes:** ↑E, ↑total CHO, ↔starch, ↑total F, ↑SFA, ↔total P, ↓total fiber, ↑GI, ↑GL, grains (↓whole, ↑refined), ↔total sugar • **Other diet outcomes:** NR **PA:** • ***a priori* PA outcomes**: ↓Total PA time • **Other PA outcomes**: NR
Ganie *et al.*, 2019, India	**Total**, 464 (PCOS, 144 [82 vegetarian 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	**Design:** Case-control **Setting:** Medical lefts **Analysis:** Prospective	**Diet:** FFQ and 72-hr dietary recalls (dietitians administered) **PA:** NR	**Diet:** • ***a priori* diet outcomes:** ↑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* non-vegetarian control), ↔total P (g and % of E in

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Author, yrs. (reference), and country	Participants' characteristics (n, mean age [yrs.], mean BMI [kg/m^2]), PCOS definition, and racial/ethnic composition	Study design, setting, data analysis	Dietary/PA assessment tool	Reported outcomes of interest
				both vegetarian PCOS *versus* vegetarian control and non-vegetarian PCOS *versus* non-vegetarian control) • **Other diet outcomes:** NR **PA:** • ***a priori* PA outcomes:** NR • **Other PA outcomes:** NR
Hart *et al.*, 2016, UK	**Total**, 68 (PCOS, 38; Control, 30) **PCOS**, age: 30.8, BMI: 24.5 **Control**, age: 29.3, BMI: 23.5 **PCOS definition**, Clinically established diagnosis (unspecified) **Ethnic composition**, 94% White, 3% Black, 3% Asian	**Design:** NR **Setting:** AMC **Analysis:** Retrospective	**Diet:** 7-d food diary **PA:** 7-d activity diary and pedometer (YamaxDigi-walker SW-200, worn for 7 consecutive d)	**Diet:** • ***a priori* diet outcomes:** ↔E, ↓total CHO, ↑total F, ↑SFA, ↑MUFA, ↔PUFA, ↔total P, ↔total fiber, ↔GI, ↔GL, ↓total sugar • **Other diet outcomes:** NR **PA:** • ***a priori* PA outcomes:** ↔Total PA time or intensity • **Other PA outcomes:** ↔Steps/d
Hosseini *et al.*, 2017, Iran	**Total**, 297 (PCOS, 99; Control, 198) **PCOS**, age: 29.0, BMI: 26.6 **Control**, age: 29.5, BMI: 26.0 **PCOS definition**, AEPCOS **Ethnic composition**, Iranian	**Design:** Case-control **Setting:** AMC **Analysis:** Prospective	**Diet :** 168-item FFQ (assessed food intake over the past yr. daily, weekly, or monthly, interview-administered and validated) (Mirmiran *et al.*, 2010) **PA:** IPAQ (Craig *et al.*, 2003)	**Diet:** • ***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 **PA:** • ***a priori* PA outcomes:** ↔Total PA time or intensity • **Other PA outcomes:** NR
		Design: Case-cohort		**Diet:**

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Author, yrs. (reference), and country	Participants' characteristics (n, mean age [yrs.], mean BMI [kg/m^2]), PCOS definition, and racial/ethnic composition	Study design, setting, data analysis	Dietary/PA assessment tool	Reported outcomes of interest
Huijgen *et al.*, 2015, Netherlands	**Total**, 1017 (PCOS, 218 [HA = 112, non-HA = 106]; Control, 799) **PCOS**, age: 28.5, BMI: 25.6 **Control**, age: 33.1, BMI: 24.5 **PCOS definition**, Rotterdam **Ethnic composition**, 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	• ***a priori* diet outcomes:** ↑Diet inadequacy by Preconception Dietary Risk score (all PCOS cohorts *versus* control) • **Other diet outcomes:** ↔%inadequate whole wheat intake (**<**4–5 slices of bread/d, HA phenotype *versus* non-HA phenotype *versus* control); ↔%inadequate intake of MUFA or PUFA (unspecified threshold, HA phenotype *versus* non-HA phenotype *versus* control); ↔%inadequate vegetable intake (**<**200 g/d, HA phenotype *versus* non-HA phenotype *versus* control); ↑%inadequate meat intake (**<**3–4 servings/wk, HA phenotype *versus* control); ↑%inadequate fish intake (**<**2 servings/wk, HA phenotype *versus* control)**PA:** • ***a priori* PA outcomes:** NR • **Other PA outcomes:** ↔%Women not participating in PA
Jurewicz *et al.*, 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	**Diet:** Questionnaire (unspecified; self-reported) **PA:** NR	**Diet:** • ***a priori* diet outcomes:** ↔Alcohol • **Other diet outcomes:** NR**PA:** • ***a priori* PA outcomes:** NR • **Other PA outcomes:** NR
Kazemi Jaliseh *et al.*, 2017, Iran	**Total**, 1702 (PCOS, 178; Control, 1524)	**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^2]), 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	**PA:** Lipid Research Clinic questionnaire (Ainsworth *et al.*, 1993) (self-reported, validated)	• **Other diet outcomes:** NR **PA:** • ***a priori* PA outcomes:** ↔Exercise level (light, moderate, strenuous) • **Other PA outcomes:** NR
Khademi *et al.*, 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	**Design:** Case-control **Setting:** Female employees of a company who had regular physical activities **Analysis:** Prospective	**Diet:** NR **PA:** Questionnaire (unspecified; interviewer-administered)	**Diet:** • ***a priori* diet outcomes:** NR • **Other diet outcomes:** NR **PA:** • ***a priori* PA outcomes:** ↔total PA time, ↔regular walking • **Other PA outcomes:** NR
Larsson *et al.*, 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	**Design:** Case-control **Setting:** AMC **Analysis:** Prospective	**Diet:** FFQ (verified in Swedish adolescents, interview-administered by dietitians) (Sjöberg *et al.*, 2003) **PA:** NR	**Diet:** • ***a priori* diet outcomes:** ↔E, ↑total CHO (g and AMDR), ↔total F (g and AMDR), ↔SFA, ↔total P, ↔total fiber, ↓alcohol, ↔whole grains • **Other diet outcomes:** ↔Total EAT Score, ↔unsaturated fatty acids**PA:** • ***a priori* PA outcomes:** NR • **Other PA outcomes:** NR
Lerchbaum *et al.*, 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	**Design**: Baseline characteristic (cross-sectional analysis) of a randomized clinical trial **Setting**: AMC **Analysis**: Retrospective	**Diet**: Questionnaire (unspecified) **PA**: NR	Diet: • **a priori diet outcomes:** ↔Vit D • **Other diet outcomes:** NR**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^2]), PCOS definition, and racial/ethnic composition	Study design, setting, data analysis	Dietary/PA assessment tool	Reported outcomes of interest
Liang *et al.*, 2021, China	**Total**, 40 (PCOS, 20 [lean 10, overweight 10]; Control, 20 [lean 10, overweight 10]) **PCOS**, lean age: 24.13, BMI: 20.46; overweight age: 28.94, BMI: 27.34 **Control**, lean age: 25.08, BMI: 20.43; overweight age: 30.12, BMI: 26.05 **PCOS definition**, Rotterdam **Ethnic composition**, Han from Southern China	**Design:** Case-control **Setting:** AMC **Analysis:** Prospective	**Diet:** Questionnaire (3-d 24-hr recalls; interviewer-administered) **PA:** NR	**Diet:** • ***a priori* diet outcomes:** ↔E, ↔total CHO, ↔total F, ↓total P, ↓total fiber, ↓Vit D, ↔Ca, ↔Mg, ↔Na • **Other diet outcomes:** ↔Vit A, ↓Vit E, ↔iodine, ↔Se, ↔Cu, ↔Mn, ↔K **PA:** • ***a priori* 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 accelerometer) and self-reported PA (Women's Health Initiative Physical Activity Questionnaire) (Meyer *et al.*, 2009)	**Diet:** • ***a priori* 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 • **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 acids**PA:** • ***a priori* PA outcomes:** Total PA time and intensity • **Other PA outcomes:** NR
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	**Diet:** CARDIA diet history questionnaire (past 28 d; interviewer-administered) **PA:** CARDIA PA questionnaire (past yr.; self-reported)	**Diet:** • ***a priori* 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

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Author, yrs. (reference), and country	Participants' characteristics (n, mean age [yrs.], mean BMI [kg/m^2]), PCOS definition, and racial/ethnic composition	Study design, setting, data analysis	Dietary/PA assessment tool	Reported outcomes of interest
				• **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 acids**PA:** • ***a priori* PA outcomes:** ↔Total PA time, duration, intensity), exercise level (↔moderate, ↔vigorous) • **Other PA outcomes:** NR
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 *et al.*, 2005) (validated) **PA:** Structured questionnaire (unspecified, interview-administered)	**Diet:** • ***a priori* diet outcomes:** ↔E, ↔alcohol, ↔total n-3 PUFA, ↓long chain n-3 PUFA • **Other diet outcomes:** ↔fish oil supplements, ↔alpha-linoleic acid, ↓eicosapentaenoic acid, ↓docosapentaenoic acid, ↓docosahexaenoic acid **PA:** • ***a priori* PA outcomes:** ↓Total PA time (frequency/wk) • **Other PA outcomes:** NR
Melekoglu *et al.*, 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	**Diet:** 3-d integrated food and PA record **PA:** 3-d integrated food and PA record	**Diet:** • ***a priori* diet outcomes:** ↓E, ↔total F (g and AMDR), ↑SFA, ↔MUFA, ↔PUFA, ↔trans F, ↔total P, ↓total fiber, soluble fiber, insoluble fiber, ↓Vit B9, ↓Fe, ↔Ca, ↓Mg, ↔Zn, ↔Na, ↔GI, ↓GL, vegetable (↔total or starchy vegetable), ↔total dairy • **Other diet outcomes:** ↑Vit B12

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Author, yrs. (reference), and country	Participants' characteristics (n, mean age [yrs.], mean BMI [kg/m^2]), PCOS definition, and racial/ethnic composition	Study design, setting, data analysis	Dietary/PA assessment tool	Reported outcomes of interest
				PA: • ***a priori* PA outcomes:** ↑PA (calculated as TEE/BMR (Third report of the national cholesterol education program (NCEP) expert panel on detection, evaluation, and treatment of high blood cholesterol in adults, 2002) • **Other PA outcomes:** NR
Misir *et al.*, 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	**Design:** Cross-sectional **Setting:** AMC **Analysis:** Prospective	**Diet:** 24-hr diet recall **PA:** Survey on the Basic data, Dietary Habits, and PA (unspecified)	**Diet:** • ***a priori* diet outcomes**: ↔Total CHO (g and AMDR), ↔total F (g and AMDR), ↔SFA, ↔MUFA, ↔PUFA, ↑total P, ↑animal P, ↔total fiber • **Other diet outcomes:** ↑Vit B12, ↔linoleic acid**PA:** • ***a priori* PA outcomes:** NR • **Other PA outcomes:** ↔Work index, ↔sport index, ↓free time index
Moran *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 dietary pattern and PA data age: 33.5, BMI: 29.3 for all Other dietary factors **Control,** age: 33.7, BMI: 25.4 for dietary pattern and PA data age: 33.7, BMI: 25.6 for all Other dietary 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)	**Diet:** • ***a priori* diet outcomes:** ↑diet quality by Dietary Guideline Index, dietary glycemic indices, MED pattern (crude or adjusted), ↑meat/fish/poultry and take-away dietary pattern (crude only), and ↔unhealthy non-core foods (crude or adjusted; all patterns indicate associations with PCOS status), ↑E, ↔total CHO (g and AMDR), ↔mono- and oligosaccharides, ↔Other polysaccharides, ↔total F (g and AMDR), ↔Chol, ↓SFA, ↔MUFA, ↔PUFA, ↑total fiber, ↔alcohol, ↑Vit B9, ↑Fe, ↑Ca, ↑Mg, ↑Zn, ↑Na, ↓GI, ↑GL • **Other diet outcomes**: ↔Vit A, ↔Vit B1, ↔Vit B2, ↑Vit B3, ↑Vit B12, ↔Vit C, ↑Vit E**PA:** • ***a priori* PA outcomes**: ↔Total PA time, ↑sedentary/sitting time • **Other PA outcomes:** NR
Navarro-Lafuente *et al.*, 2022, Spain	**Total**, 276 (PCOS, 121; Control, 155)	**Design:** Case-control **Setting:** AMC	**Diet:** 101-food item semi-	**Diet:**

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Author, yrs. (reference), and country	Participants' characteristics (n, mean age [yrs.], mean BMI [kg/m^2]), 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) **PA:** IPAQ-SF (Baecke *et al.*, 1982)	• ***a priori diet outcomes:*** ↔E, ↔total CHO, ↔total F , ↑Chol, ↔SFA, ↔MUFA, ↔PUFA, ↔trans F, ↔total P, ↔total fiber, ↓alcohol • **Other diet outcomes:** ↔Omega 3, ↔omega 6, ↔omega 6/omega 3, ↔α carotene, ↔β carotene, ↔lycopene, ↔cryptoxanthin, ↔lutein, ↔zeaxanthin, ↓caffeine **PA:** • **a priori PA outcomes:** ↔PA (hr/wk of moderate-vigorous exercise) • **Other PA outcomes:** NR
Neubronner *et al.*, 2021, Singapore	**Total**, 389 (PCOS, 134 [Normal BMI, 61; High BMI, 73]; Control, 255 [Normal BMI, 152; High BMI, 103]) **PCOS**, age: 29.84, BMI: 25.14 **Control**, age: 32.24, BMI: 23.08 **PCOS definition**, Rotterdam **Ethnic composition**, Chinese 71%, Malay 9%, Indian 8%, Other 12%	**Design:** Cross-sectional **Setting:** AMC **Analysis:** Prospective	**Diet:** NR **PA:** NR	**Diet:** • ***a priori* diet outcomes:** NR • **Other diet outcomes:** ↔Alcohol intake (%PCOS [obese, lean] *versus* control [obese, lean]), ↔coffee intake (%PCOS [obese, lean] *versus* control [obese, lean]) **PA:** • ***a priori* PA outcomes:** ↔Total PA time, ↔regular walking • **Other PA outcomes:** NR
Noormohammadi *et al.*, 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	**Diet:** 168-item FFQ (semi-quantitative, validated) **PA:** PA scale (self-reported, validated in Danish men and women)	**Diet:** • ***a priori* 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 • **Other diet outcomes:** ↓Ratio of MUFA to trans F **PA:** • ***a priori* PA outcomes:** Total PA (time or intensity) • **Other PA outcomes:** NR
Orio *et al.*, 2006, Italy	**Total**, 90 (PCOS, 45; Control, 45) **PCOS**, age: 21.3, BMI: 29.4	**Design:** Case-control **Setting:** AMC	**Diet:** NR	**Diet:** • ***a priori* diet outcomes:** NR

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Table I Continued

Author, yrs. (reference), and country	Participants' characteristics (n, mean age [yrs.], mean BMI [kg/m^2]), 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	**PA:** Cardiopulmonary test on bicycle ergometer	• **Other diet outcomes:** NR **PA:** • ***a priori* PA outcomes:** NR • **Other PA outcomes:** ↔PA score
Panjeshahin *et al.*, 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	**Diet:** 178-item FFQ (semi-quantitative, modified version of a 168-item FFQ used in the Tehran Lipid and Glucose Study) **PA:** IPAQ-SH (Baecke *et al.*, 1982)	**Diet:** • ***a priori* diet outcomes:** ↑High GI-high fat dietary pattern and ↑PCOS risk, ↑anti-inflammatory dietary patterns and ↓PCOS risk, ↔low GI-low fat dietary pattern and PCOS risk], ↑E • **Other diet outcomes:PA:** • ***a priori* PA outcomes:** ↓Total PA time and intensity • **Other diet outcomes:** NR
Pokorska-Niewiada *et al.*, 2021, Poland	**Total**, 63 (PCOS, 47 [with insulin resistance, 28; without insulin resistance, 19]; Control, 16) **PCOS**, age: 28.3, BMI: 29.95 **Control**, age: 29.0, BMI: 23.3 **PCOS definition**, Rotterdam **Ethnic composition**, NR	**Design:** Cross-sectional **Setting:** AMC **Analysis:** Prospective	**Diet:** 4-d dietary food record (2 weekdays and 2 weekend d) **PA:** NR	**Diet:** • ***a priori* diet outcomes:** ↔Fe, ↔Mg, ↔Zn, ↔Na (for matched groups based on insulin resistance) • **Other diet outcomes:** ↔K, ↔Cu (for matched groups based on insulin resistance) **PA:** • ***a priori* PA outcomes:** NR • **Other PA outcomes:** NR
Pourghassem Gargari *et al.*, 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	**Diet:** 24-hr recall (2 workday and 1 weekend) and FFQ (weekly, monthly, and usual dietary intake in past 1 yr.; unspecified)	**Diet:** • ***a priori* diet outcomes:** ↓E, ↓total CHO (g and %E), ↓total F (g, but ↔%E), ↓SFA, ↔MUFA, ↔PUFA, ↓total P (g, but ↔%E), ↔total fiber, ↔Vit B9, ↓Vit D,

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Table I Continued

Author, yrs. (reference), and country	Participants' characteristics (n, mean age [yrs.], mean BMI [kg/m^2]), PCOS definition, and racial/ethnic composition	Study design, setting, data analysis	Dietary/PA assessment tool	Reported outcomes of interest
			PA: NR	↓Ca, ↓Mg, ↔Zn, ↔Na, ↓total fruit, ↓nuts, ↓total dairy and milk, ↔total sugar • **Other diet outcomes:** ↔Chromium, ↔Vit B12, ↑onion **PA:** • ***a priori* PA outcomes:** NR • **Other PA outcomes:** NR
Rajaeieh *et al.*, 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	**Diet:** 168-item FFQ (developed for the Tehran Lipid and Glucose Study) **PA:** IPAQ-SH (Baecke *et al.*, 1982)	**Diet:** • ***a priori* diet outcomes:** ↔E, dairy (↔total and low fat and whole fat milk, ↔total, high-fat, and low-fat yogurt, ↔cheese) • **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 cream**PA:** • ***a priori* PA outcomes:** ↔PA (% of women with mild, moderate, or vigorous PA) • **Other PA outcomes:** NR
Sedighi *et al.*, 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	**Diet:** 28-item researcher-devised questionnaire (0–112 points wherein higher scores showed adherence to a more appropriate diet) **PA:** IPAQ	**Diet:** • ***a priori* diet outcomes:** ↓Diet score • **Other diet outcomes:** NR**PA:** • ***a priori* PA outcomes:** ↓Total PA time and intensity • **Other PA outcomes:** NR
Shahdadian *et al.*, 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	**Diet:** 168-item semi-quantitative FFQ (verified in Iranian population) (Asghari *et al.*, 2012) **PA:** IPAQ-SH (Baecke *et al.*, 1982)	**Diet:** • ***a priori* diet outcomes:** ↔E, ↑total CHO, ↑total F, ↑total P • **Other diet outcomes:** NR**PA:**

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Table I Continued

Author, yrs. (reference), and country	Participants' characteristics (n, mean age [yrs.], mean BMI [kg/m^2]), PCOS definition, and racial/ethnic composition	Study design, setting, data analysis	Dietary/PA assessment tool	Reported outcomes of interest
				• ***a priori* PA outcomes:** ↓Total PA time and intensity • **Other PA outcomes:** NR
Shahrokhi and Naeini, 2020, 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	**Diet:** 168-item FFQ (self-reported in the past yr., on daily, weekly, or monthly basis) **PA:** IPAQ-SH (Baecke *et al.*, 1982)	**Diet:** • ***a priori* diet outcomes:** ↑E, ↓Zn • **Other diet outcomes:** ↓Vit C, ↓Vit E, ↓Se, ↓beta-carotene**PA:** • ***a priori* PA outcomes:** ↔Total PA time or intensity • **Other PA outcomes:** NR
Shishehgar *et al.*, 2016a, Iran	**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	**Design:** Case-control **Setting:** AMC **Analysis:** Prospective	**Diet:** 147-item FFQ (interview-administered; participants reported the frequency of consumption of each food item per d, wk or mo during previous yr.) **PA:** IPAQ	**Diet:** • ***a priori* diet outcomes:** ↔E, ↔total CHO (g and %E), ↔total F (g and %E), ↔SFA (g and % E), ↔MUFA (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 • **Other diet outcomes:** ↑Egg, ↔oil, ↔fast foods, ↑high GI foods, ↔medium and low GI foods **PA:** • ***a priori* PA outcomes:** ↔ Total PA time, ↔exercise level (mild, moderate, vigorous), ↑sitting time • **Other PA outcomes:** NR
Shishehgar *et al.*, 2019, Iran	**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 characteristic (cross-sectional analysis) of an interventional study **Setting:** AMC **Analysis:** Prospective	**Diet:** 3-d dietary food record (2 working days and 1 weekend d) **PA:** IPAQ-SH (Baecke *et al.*, 1982)	**Diet:** • ***a priori* diet outcomes:** ↔E, ↔total CHO (g and %E), ↔total F (%E), ↔total P (%E), ↔total fiber, ↔GI, ↔GL • **Other diet outcomes:** NR**PA:**

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Table I Continued

Author, yrs. (reference), and country	Participants' characteristics (n, mean age [yrs.], mean BMI [kg/m^2]), PCOS definition, and racial/ethnic composition	Study design, setting, data analysis	Dietary/PA assessment tool	Reported outcomes of interest
				• ***a priori* PA outcomes:** ↔ Total PA time or intensity • **Other PA outcomes:** NR
Soodi *et al.*, 2021 and Zirak Sharkesh *et al.*, 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 (validated for Iranian population) (Esfahani *et al.*, 2010) (self-reported) **PA:** IPAQ-SH (Baecke *et al.*, 1982)	**Diet:** • ***a priori* diet outcomes:** ↓Total DDS, ↔grain DDS, ↔fruit DDS, ↓vegetables DDS, ↓meat/meat product DDS, ↔dairy DDS, ↔E, ↑total CHO, ↔total F, ↑Chol, ↔SFA, ↓MUFA, ↔PUFA, ↔total P, ↓total fiber, ↔Vit B9, ↓Vit D, ↔Fe, ↔Ca, ↔Mg, ↔Zn, ↔Na, grains (↔whole, ↑refined), ↔fruit, ↔vegetables, ↔protein food (↔red and processed meat, plant protein [(↔legume]), ↓dairy • **Other diet outcomes:** ↔ Vit B6, ↓Vit B12, ↔Vit C, ↔Vit E, ↔K, ↔ Ph ↔ chromium , ↔caffeine, ↔fructose, ↔glucose, ↓omega 3**PA:** • ***a priori* PA outcomes:** ↓Total PA time (MET min/d) • **Other PA outcomes:** NR
Szczuko *et al.*, 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	**Diet:** 3-d food diary (interview-administered) **PA:** NR	**Diet:** • ***a priori* diet outcomes:** ↓Vit B9 • **Other diet outcomes:** ↔Vit B1, ↔Vit B2, ↔Vit B3, ↔Vit B6, ↔Vit B12, ↓Vit C**PA:** • ***a priori* PA outcomes**: NR • **Other PA outcomes:** NR
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)	**Diet:** • ***a priori* diet outcomes:** NR • **Other diet outcomes:** ↔Alcohol drinking pattern (% of non-, low-risk, and high-risk drinkers)**PA:** • ***a priori* PA outcomes:** ↓Total PA time, ↑sedentary/sitting time • **Other PA outcomes:** ↓%Meeting Australian PA guidelines for weight maintenance (≥500 MET.min/wk), ↓%meeting Australian PA guidelines for weight

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Table I Continued

Author, yrs. (reference), and country	Participants' characteristics (n, mean age [yrs.], mean BMI [kg/m^2]), PCOS definition, and racial/ethnic composition	Study design, setting, data analysis	Dietary/PA assessment tool	Reported outcomes of interest
				loss (≥833 MET.min/wk), ↔high (≥8 hrs/d) sitting time
Thara and Divakar, 2017, India	**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	**Diet:** 24-hr recall (structured questionnaire, unspecified) **PA:** NR	**Diet:** • ***a priori* diet outcomes:** ↑E, ↑total CHO, ↑total F, ↑total P, ↓Fe, ↔Ca • **Other diet outcomes:** ↓Vit A **PA:** • ***a priori* PA outcomes:** NR • **Other PA outcomes:** NR
Thomson *et al.*, 2009, Australia	**Total**, 26 (PCOS, 10; Control, 16) **PCOS**, age: 33.6, BMI: 34.1 **Control**, age: 36.8, BMI: 35.5 **PCOS definition**, Rotterdam **Ethnic composition**, NR	**Design:** Cross-sectional **Setting:** AMC **Analysis:** Prospective	**Diet:** NR **PA:** IPAQ-SF (Baecke *et al.*, 1982) and questionnaires (Philippaerts *et al.*, 1999) (self-reported and recorded PA over the previous yr., unspecified).	**Diet:** • ***a priori* diet outcomes:** NR • **Other diet outcomes:** NR**PA:** • ***a priori* PA outcomes:** ↔Exercise level (light, moderate, strenuous), ↔leisure activity level (walking, gardening) • **Other PA outcomes:** ↔Total PA score, ↔time to exhaustion (exercise tolerance)
Tsai *et al.*, 2013, Taiwan, ROC	**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	**Diet:** 3-d dietary record (2 weekdays and 1 weekend d; records were verified by a registered dietitian via telephone interview) **PA:** NR	**Diet:** • ***a priori* diet outcomes:** ↓E, ↓total CHO, ↔F (g, but ↑%E), ↔total P • **Other diet outcomes:** NR**PA:** • ***a priori* PA outcomes:** NR • **Other PA outcomes:** NR
Wang *et al.*, 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	**Diet:** • ***a priori* diet outcomes:** ↔E, ↔alcohol, ↓total vegetable, ↔total fruit • **Other diet outcomes:** ↔Sugary drink, ↔savory snack ↔sweet snack **PA:**

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Table I Continued

Author, yrs. (reference), and country	Participants' characteristics (n, mean age [yrs.], mean BMI [kg/m^2]), PCOS definition, and racial/ethnic composition	Study design, setting, data analysis	Dietary/PA assessment tool	Reported outcomes of interest
			International®, Bunschoten, The Netherlands)	• ***a priori* PA outcomes:** ↔Total PA (moderate to vigorous min/wk) • **Other PA outcomes:** ↔Leisure time (moderate to vigorous min/wk), ↔commuting time (moderate to vigorous min/wk), ↔steps
Wang *et al.*, 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	**Diet**: Chinese Health and Nutrition Survey questionnaire (Zhang *et al.*, 2014) **PA**: NR	**Diet:** • ***a priori* 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 • **Other diet outcomes**: ↓Vit A, ↓Vit C, ↔Vit E, ↔Vit B1, ↔Vit B2, ↔Vit B3, ↔Vit B6, ↑Vit B12, ↓beta-carotene, ↔daidzein, ↑Se**PA:** • ***a priori* PA outcomes**: ↔Total PA time (MET-hrs/wk) • **Other PA outcomes**: NR
Wright *et al.*, 2004a, USA	**Total**, 163 (PCOS, 84; Control, 79) **PCOS**, age: 46.7, BMI: 32.1 **Control**, age: 48.2, BMI: 29.0 **PCOS definition**, NIH (OA+[HA/↑LH/FSH]) **Ethnic composition**, PCOS [83% White, 17% Non-White]; Controls [90% White, 10% Non-White]	**Design:** Case-control **Setting:** AMC **Analysis:** Prospective	**Diet:** 109-item diet history questionnaire (Block *et al.*, 1986) (self-administered and modified version of the Block questionnaire **PA:** Paffenbarger PA Questionnaire (self-reported or interviewer-administered)	**Diet:** • ***a priori* diet outcomes:** ↔E, ↔total CHO, ↔total F, ↔Chol, ↔MUFA, ↔PUFA, ↔total P • **Other diet outcomes:** NR**PA:** • ***a priori* PA outcomes:** Exercise level (↔light, ↔moderate, ↔strenuous), ↔sedentary/sitting time • **Other PA outcomes:** ↔Sleeping/reclining time
Zaeemzadeh *et al.*, 2018, Iran	**Total**, 182 (PCOS, 151 [subgroups: frank: 41; non-PCO, 37; ovulatory: 33; mild: 40]; Control, 31) **PCOS,** 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	**Diet:** 168-food item FFQ (validated for Iranian population) **PA:** NR	**Diet:** • ***a priori* diet outcomes:** ↑E (all PCOS subgroups *versus* control), ↑total CHO (all PCOS subgroup *versus* control), ↓total F (all PCOS subgroup *versus* control), ↔SFA (all PCOS subgroup *versus* control), ↓MUFA (only frank PCOS subgroup *versus* control), ↓PUFA (only frank PCOS subgroup *versus* control), ↑trans F only frank PCOS subgroup *versus* control), ↔total P

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Table I Continued

Author, yrs. (reference), and country	Participants' characteristics (n, mean age [yrs.], mean BMI [kg/m^2]), 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			(all PCOS subgroup *versus* control), ↓total fiber (all PCOS subgroup *versus* control), ↔soluble fiber, ↓Zn (all PCOS subgroup *versus* control) • **Other diet outcomes:** ↔Chromium (only non-PCO PCOS subgroup *versus* control), ↓Se (all PCOS subgroup *versus* control), ↔carotenoid (all PCOS subgroup *versus* control) **PA:** • ***a priori* PA outcomes:** ↔Total PA time • **Other PA outcomes:** NR
Zhang *et al.*, 2020, China	**Total**, 2496 (PCOS, 2217 [subgroups: 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	**Design:** Baseline analysis of a cohort study **Setting:** AMC **Analysis:** Prospective	**Diet:** Questionnaire (interviewer-administered; unspecified) **PA:** Questionnaire (interviewer-administered; unspecified)	**Diet:** • ***a priori* diet outcomes:** ↔%Alcohol consumption (either PCOS with or without OA subgroup *versus* control) • **Other diet outcomes:** ↑%Tea drinking, 1 cup/d for at least 6 mo (either PCOS with or without OA subgroup *versus* control)**PA:** • ***a priori* PA outcomes:** ↔Total PA time in either PCOS with or without OA subgroup *versus* control • **Other PA outcomes:** NR

↑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, gram; GI, glycemic index; GL, glycemic load; h, hour; HA, hyperandrogenic; HA-OA, hyperandrogenism + oligo/amenorrhea; HEI, Healthy Eating Index; HGI, high glycemic index; hr(s), hour(s); IPAQ, International Physical Activity Questionnaire; IPAQ-SH, International Physical Activity Questionnaire Short Form; K, potassium; kcal, kilocalories; LGI, low glycemic index; LH/FSH, LH/FSH ratio.↔Denote comparable evaluated outcome measures between PCOS and control groups; MED, Mediterranean Diet; MET, metabolic equivalent of task; Mg, magnesium; min, minute; Mn, manganese; mo, month; MUFA, monounsaturated fatty acids; NIH, National Institutes of Health; NR, not-reported; P, protein; PA, physical activity; Ph, phosphorus; PREDIMED, Prevención con Dieta Mediterránea; PUFA, polyunsaturated fatty acids; rMED, relative Mediterranean Dietary Score; Se, selenium; SFA, saturated fatty acids; SSB, sugar-sweetened beverages; TEE, total energy expenditure; Vit, 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 self-reported 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 ≥ 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., 2014; Moran et al., 2015; 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.

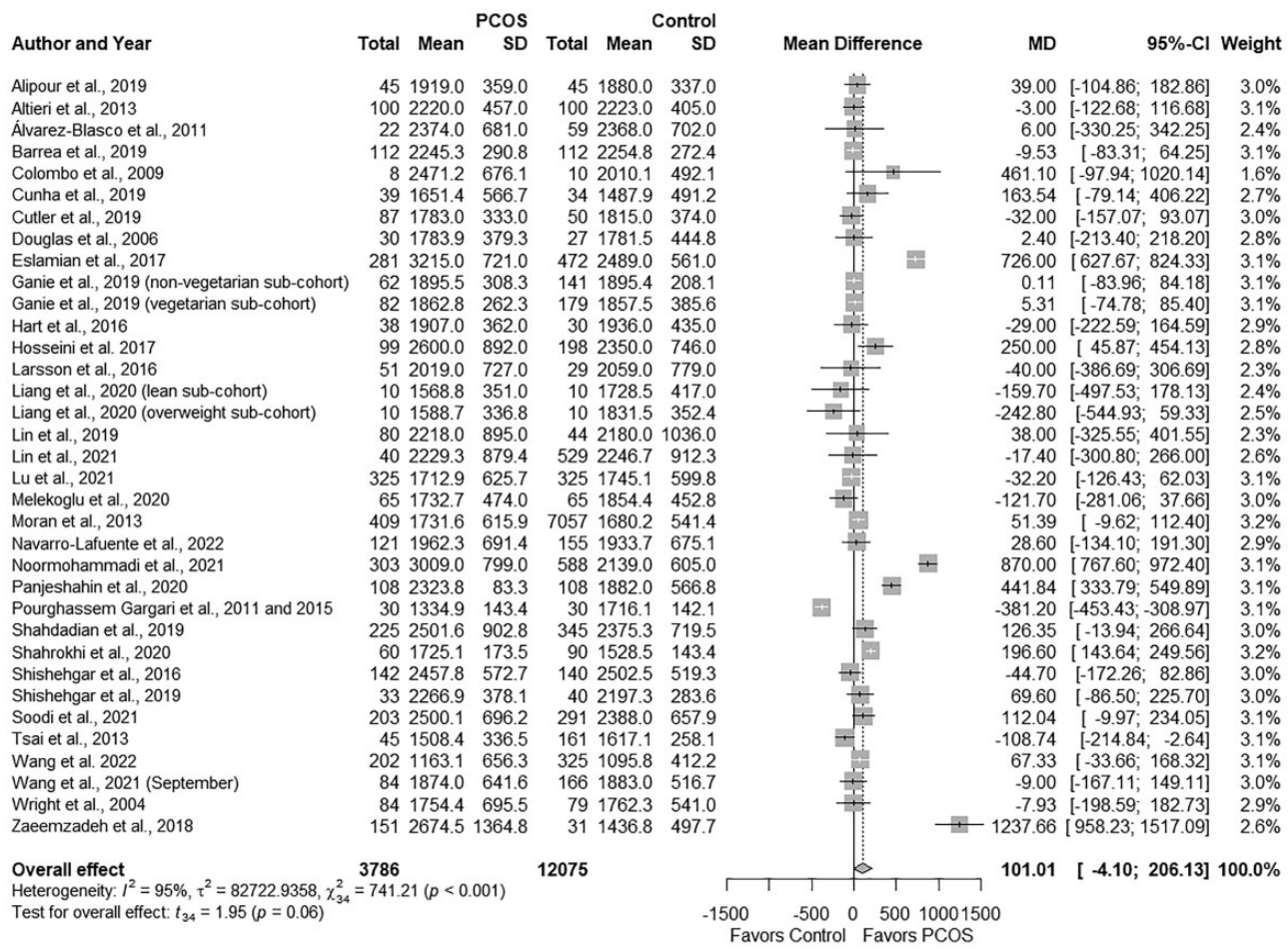


Figure 2. Forest plot for energy intake in women with and without PCOS with effect estimate expressed as kcal/day. MD, mean difference.

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

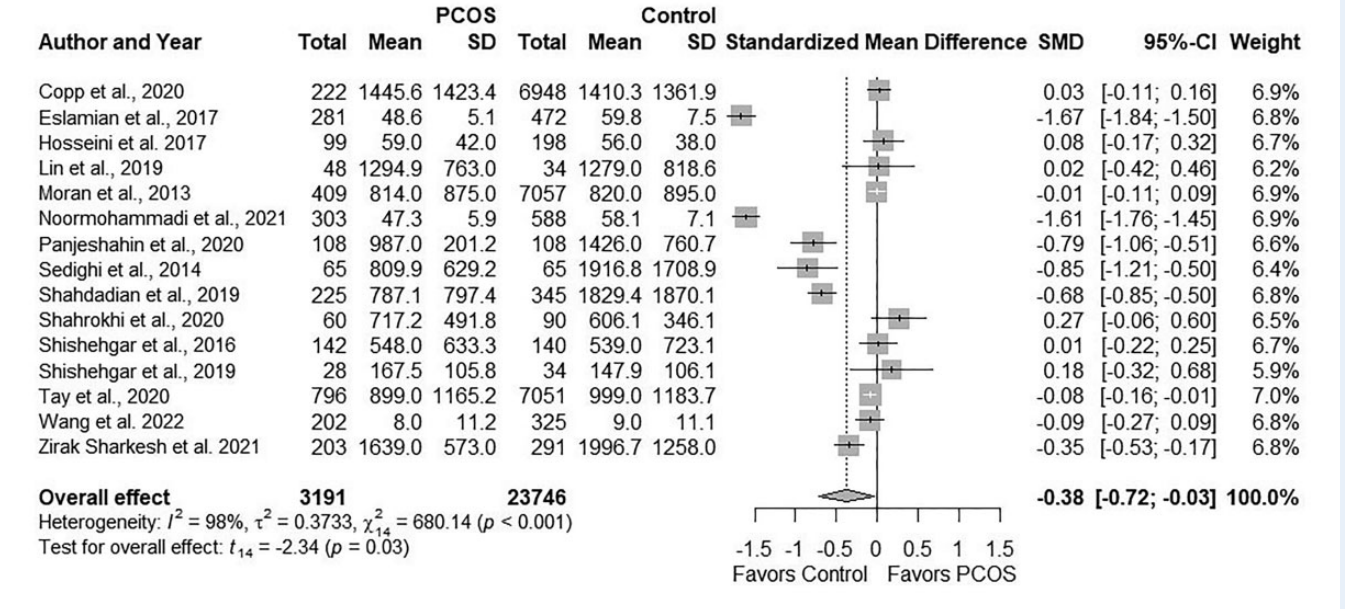


Figure 3. Forest plot for total physical activity level in women with and without PCOS. SMD, standardized mean difference.

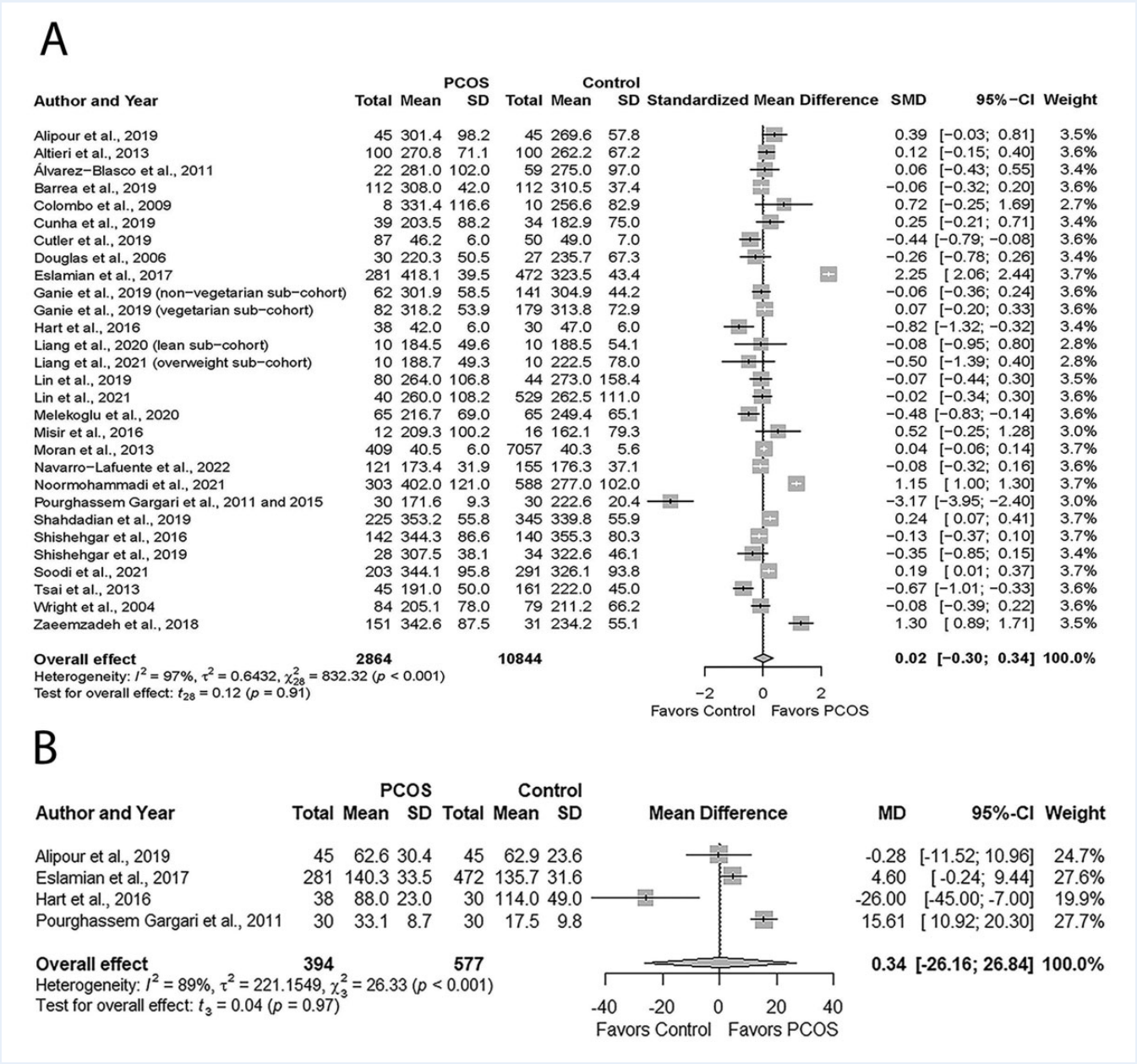


Figure 4. Forest plots for carbohydrate intake in women with and without PCOS. (**A**) Total carbohydrate intake. (**B**) Added sugar expressed in g/day. MD, mean difference; SMD, standardized mean difference.

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., 2019; Shahdadian et al., 2019; Shishehgar et al., 2019; Melekoglu 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 $P \leq 0.04$), 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).

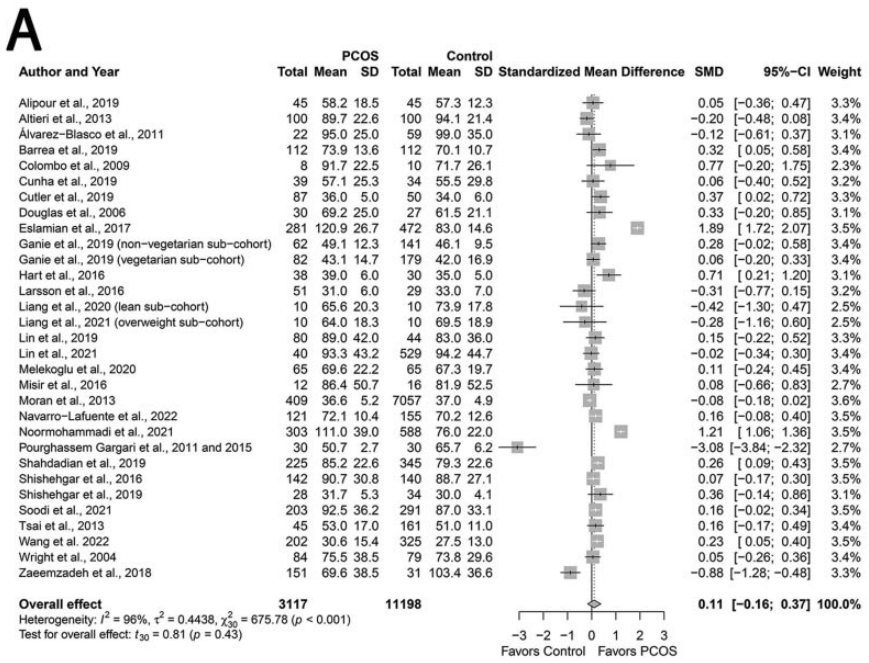
Total PA. Pooling data for total PA across 15 studies (Moran et al., 2013; Sedighi et al., 2014; Shishehgar et al., 2016b; Eslamian et al., 2017; Hosseini et al., 2017; Lin et al., 2019; Shahdadian et al., 2019; Shishehgar et al., 2019; Copp et al., 2020; Panjeshahin et al., 2020; Shahrokhi and Naeini, 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 $I^2 = 98\%$; $P \leq 0.001$). 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% CI: −16.69 to 4.92; $P = 0.18$; Supplementary Fig. S2A), and the remaining 11 (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% CI: −524.90 to 41.10; $P = 0.09$; Supplementary Fig. S2B).

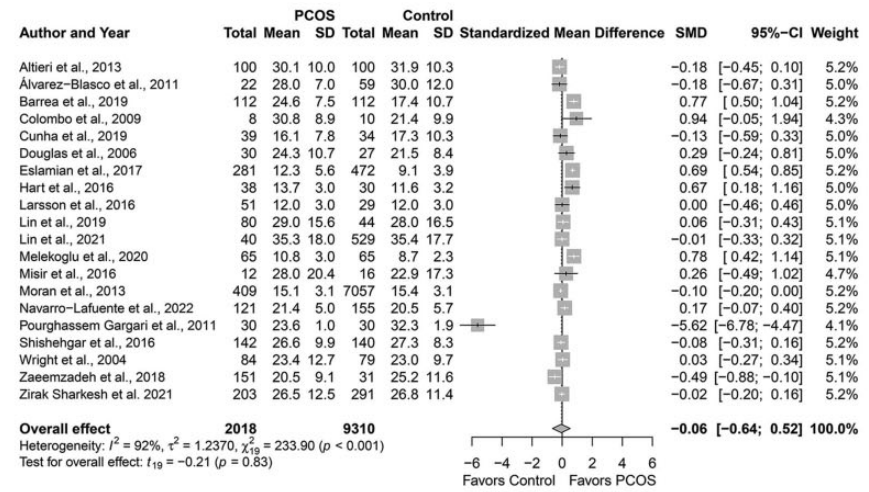
Subgroup analyses based on age, BMI, or country did not explain heterogeneity; however, PA was lower in PCOS versus Controls in analyses of subgroups that used Rotterdam criteria (SMD: −0.79; 95% CI: −1.45 to −0.14; $P = 0.03$; Supplementary Table SIV). Sensitivity analyses for SMD findings on PA showed 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: −0.34; 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% CI: −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., 2011; Altieri 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., 2016b; 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).

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% CI: −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





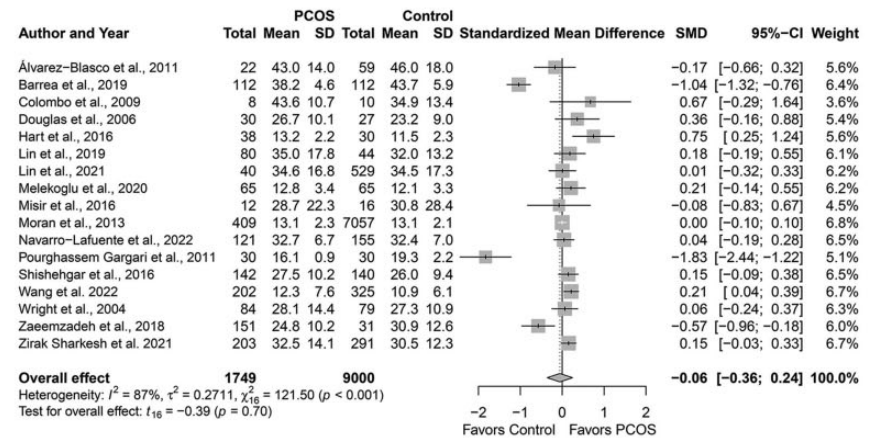


Figure 5. Forest plots for fat intake in women with and without PCOS. (**A**, total fat; **B**, saturated fatty acids [SFA]; **C**, monounsaturated fatty acids [MUFA]; **D**, polyunsaturated fatty acids [PUFA]; **E**, cholesterol) with cholesterol expressed as mg/day. SMD, standard mean difference.

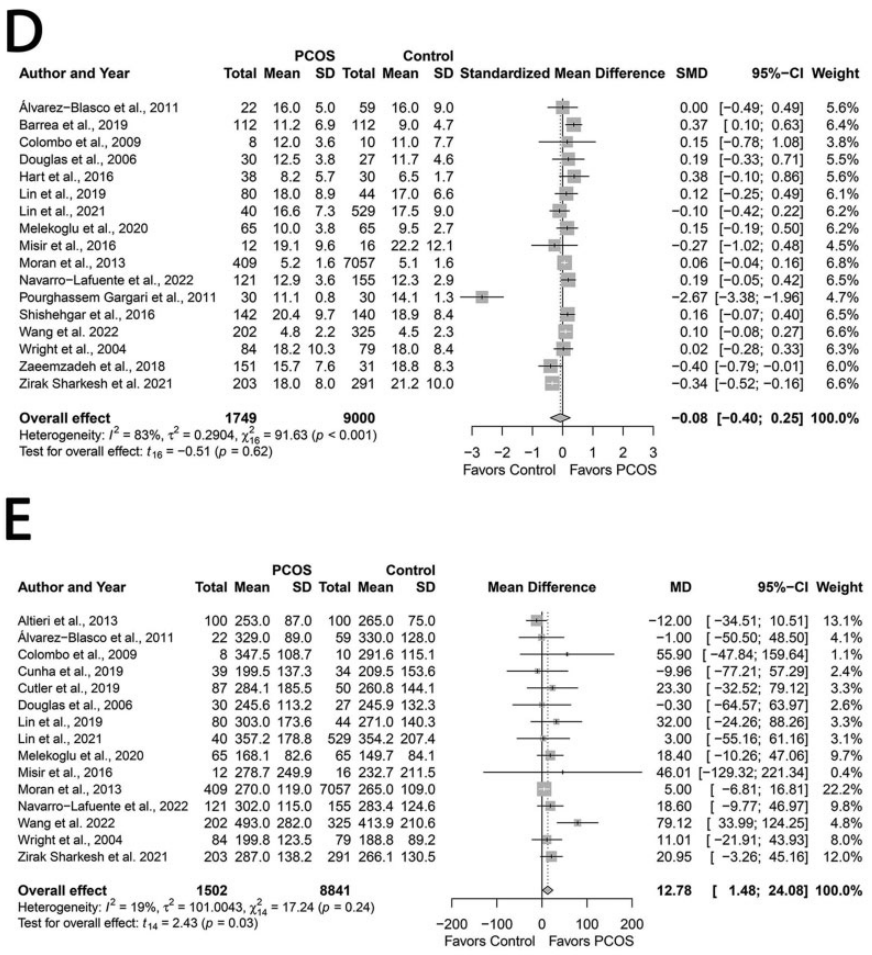


Figure 5. Continued

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

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 $P \geq 0.14$, Begg's tests; All $P \geq 0.22$, 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.*, 2016b; 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;

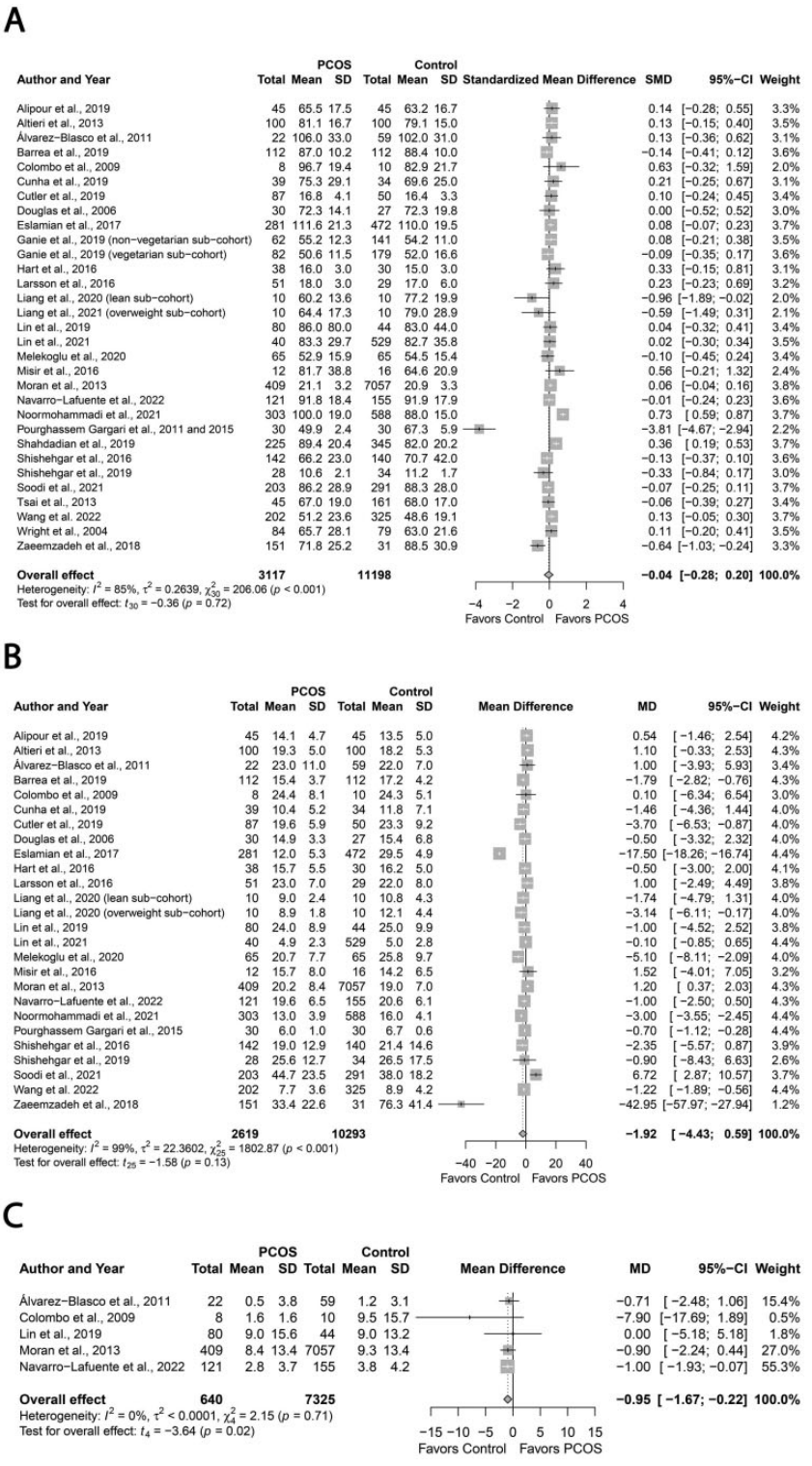


Figure 6. Forest plots for protein, fiber and alcohol intake in women with and without PCOS. (**A**) protein intake; (**B**) fiber intake expressed as g/day; and (**C**) alcohol intake expressed as g/day. MD, mean difference; SMD, standardized mean difference.

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% CI: 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., 2013; Moran 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 $I^2 \geq 83\%$; All $P < 0.001$), but studies on cholesterol intake were homogeneous ($I^2 = 18\%$; $P = 0.24$).

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% CI: 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 $P \leq 0.16$, Begg's tests; All $P \geq 0.22$, 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; 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) (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., 2021; Soodi et al., 2021; Navarro-Lafuente et al., 2022; Wang 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 $I^2 \leq 85\%$; $P < 0.001$), unlike those reporting alcohol intakes ($I^2 = 0\%$; $P = 0.71$).

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

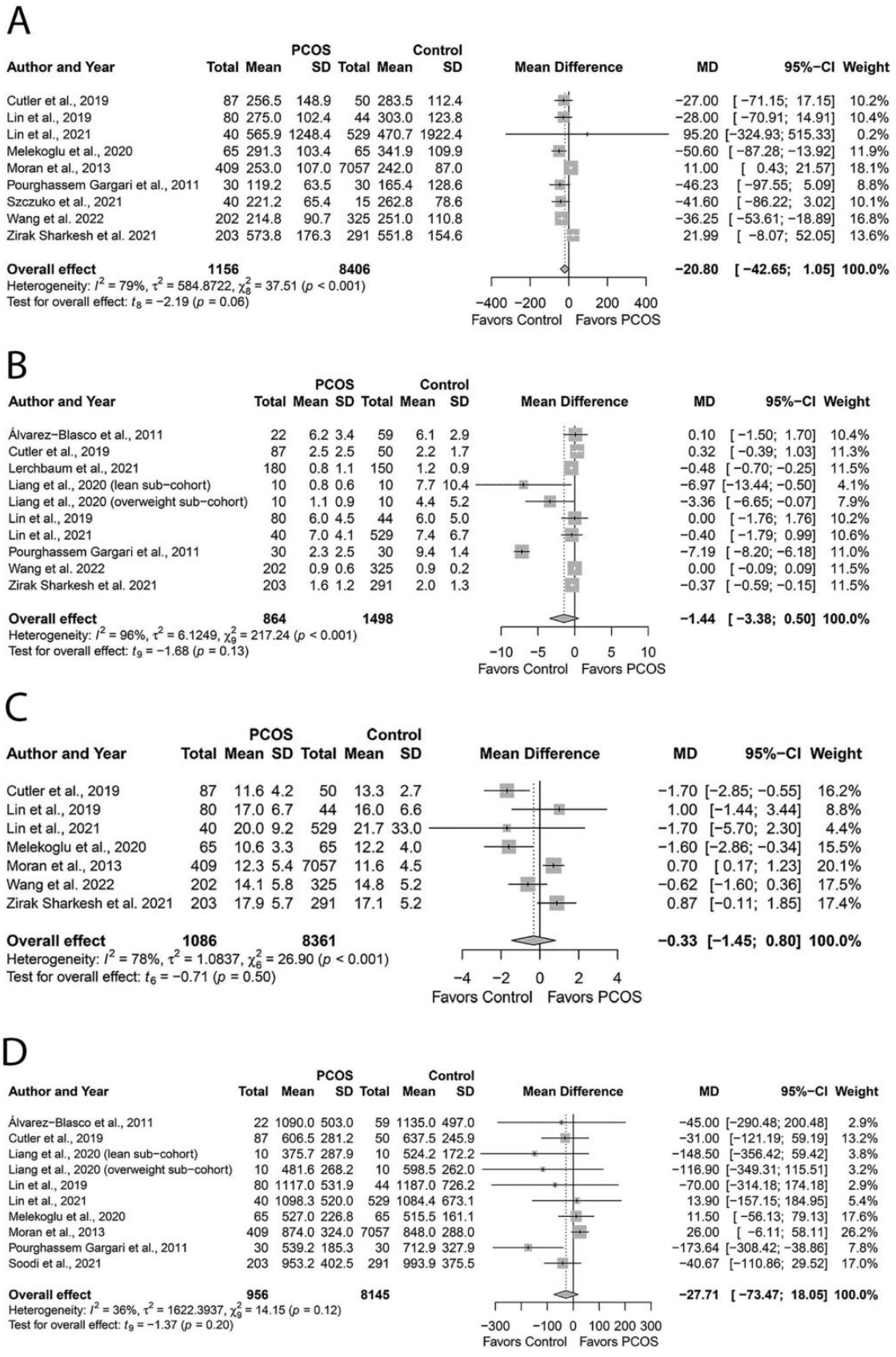


Figure 7. Forest plots for micronutrient intake in women with and without PCOS. (**A**) Folic acid intake expressed as μg/day; (**B**) vitamin D intake expressed as μg/day; (**C**) iron intake expressed as mg/day; (**D**) calcium intake expressed as mg/day; (**E**) magnesium intake expressed as mg/day;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

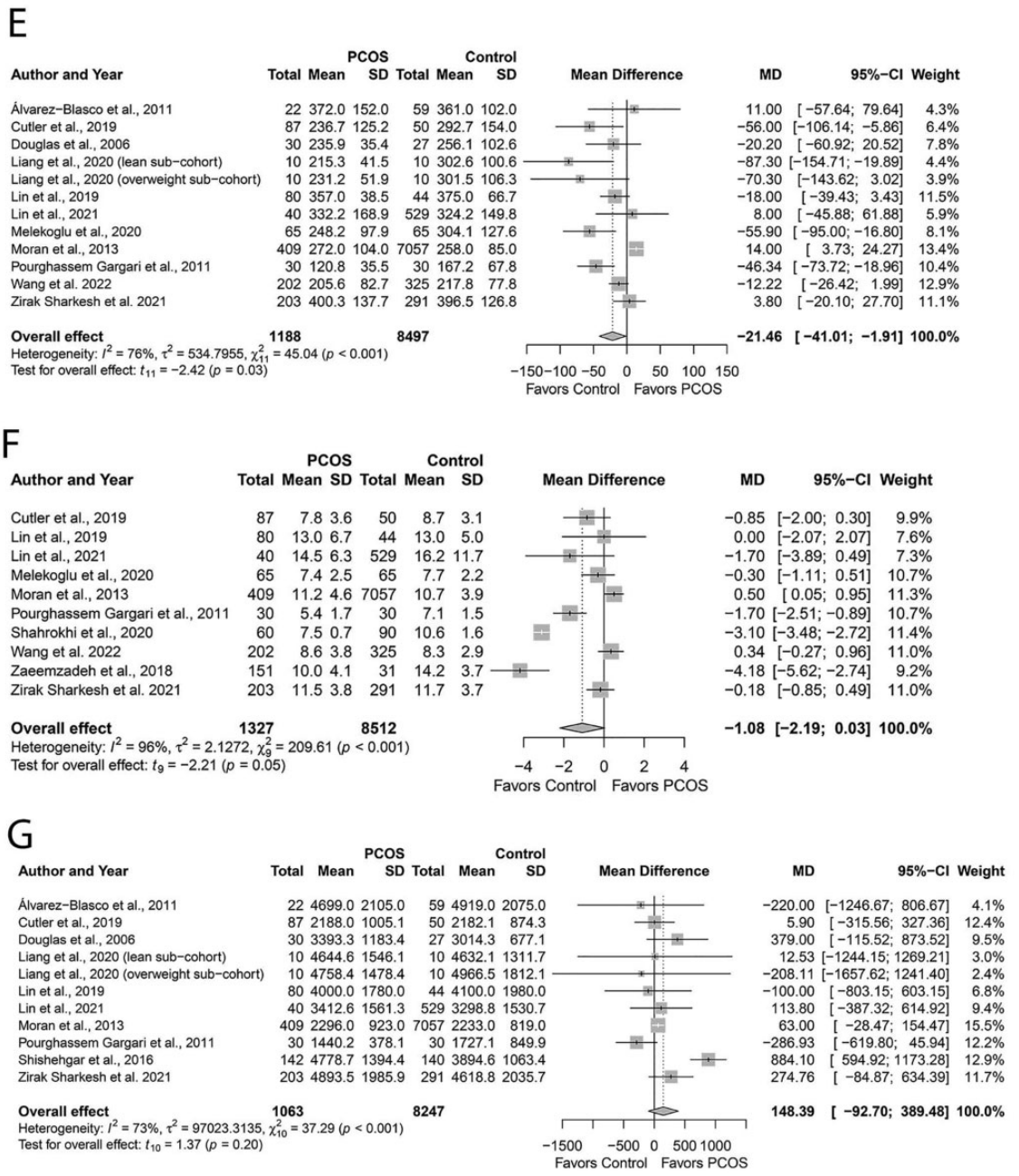


Figure 7. Continued

intakes of fiber in women with PCOS in studies conducted in China (MD: −1.33; 95% CI: −2.58 to −0.09 g/day; $P = 0.04$) and lower alcohol intakes in the older (≥30 years) subgroup (MD: −0.97; 95% CI: −1.56 to −0.37 g/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% CI: −2.01 to 0.03 g/day; $P = 0.05$), (Moran *et al.*, 2013) (MD: −0.96; 95% CI: −2.08 to 0.15 g/day; $P = 0.07$), and (Navarro-Lafuente *et al.*, 2022) (MD: −0.88; 95% CI: −2.30 to 0.54 g/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 $P \geq 0.06$, Begg's tests; All $P \geq 0.11$, 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% CI: −42.65 to −1.05 µg/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;

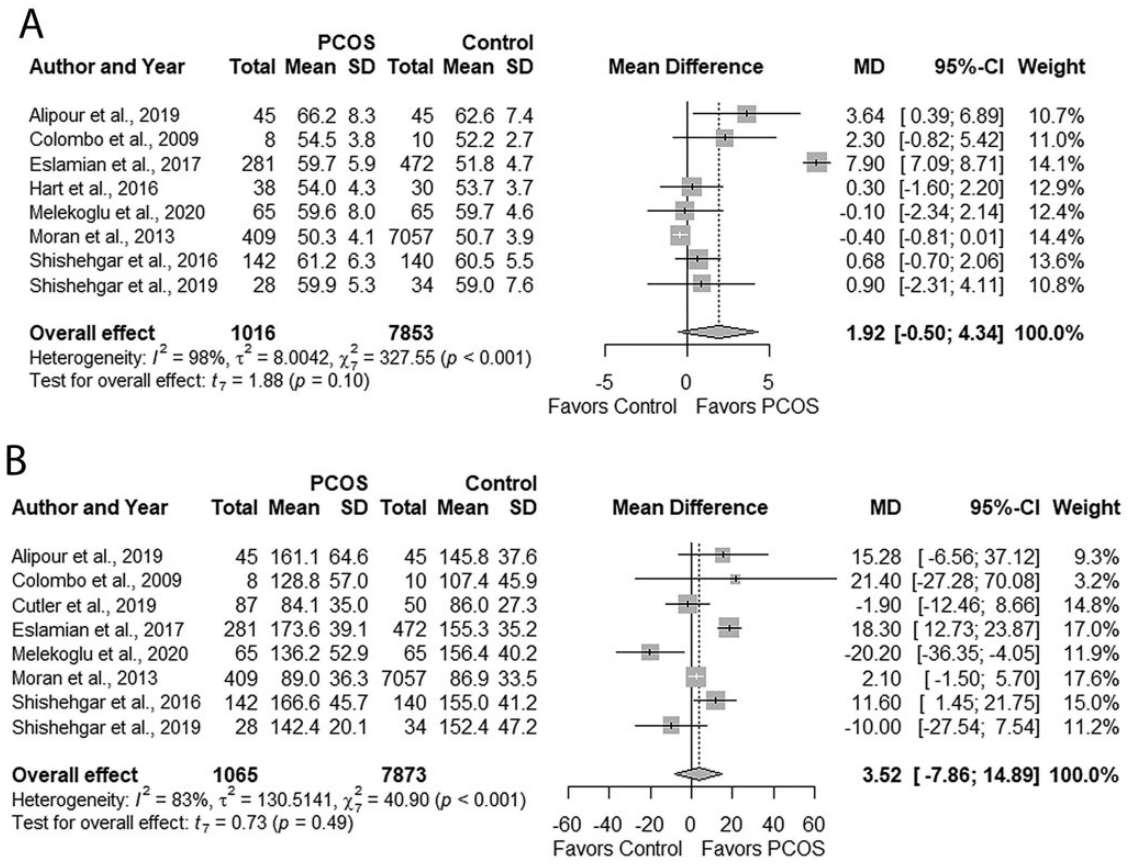


Figure 8. Forest plots for status of dietary glycemic indices in women with and without PCOS. (**A**) glycemic index and (**B**) glycemic load. MD, mean difference.

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 µg/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) (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; 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 $P \geq 0.05$). 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 $I^2 \geq 76\%$; All $P < 0.001$) except homogenous studies reporting calcium intake ($I^2 = 36\%$; 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^2; 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 µg/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 sub-cohort, 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 $P \geq 0.45$, Begg's tests; All $P \geq 0.15$, 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 $P \leq 0.02$) unlike the Begg's tests results (all $P \geq 0.53$).

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., 2019; Shishehgar et al., 2019; Melekoglu et al., 2020). Studies were moderately heterogeneous ($I^2 = 41\%$; $P = 0.12$).

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. S11A and B; all $P \geq 0.32$, Begg's test; All $P \geq 0.60$, 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 39 471 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 meta-analyses 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 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 sugar-alcohol) 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., J.Y.K., J.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.

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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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