

Systematic Review

Postmenopausal Health Outcomes in Women With Polycystic Ovary Syndrome: A Systematic Review and Meta-Analysis

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Abstract

Backgrounds: Polycystic ovary syndrome (PCOS) is one of the most common reproductive and endocrine disorders and may impact a woman's health throughout her lifespan. Research has historically focused on adolescents and women of reproductive age. However, the health status of postmenopausal women with PCOS remains insufficiently characterized. **Methods:** We conducted this systematic review and meta-analysis to identify the association between the health status of postmenopausal women and PCOS. Embase, the Cochrane Library, Web of Science, and PubMed were searched through July 4, 2025. Study quality was assessed using the Newcastle-Ottawa Scale (NOS). Weighted mean differences (WMDs) and corresponding 95% confidence intervals (CIs) were calculated for relevant health parameters comparing postmenopausal women with PCOS and healthy controls. Pooled odds ratios (ORs) were used to evaluate the association between PCOS and specific health outcomes. Heterogeneity was evaluated using the Q-test and I^2 statistic. A random-effects model meta-analysis was applied when $I^2 > 50\%$, which indicated significant heterogeneity; otherwise, a fixed-effects model was applied. **Results:** Our study included 20 studies with 1042 postmenopausal women with PCOS and 5289 controls. Compared to the controls, postmenopausal women with PCOS exhibited significantly higher body mass index (BMI) (WMD = 2.75; 95% CI: 0.98 to 4.51; $I^2 = 96\%$, $p = 0.002$), waist circumference (WMD = 7.85; 95% CI: 4.12 to 11.57; $I^2 = 73\%$, $p < 0.001$), waist/hip ratio (WMD = 0.04; 95% CI: 0.01 to 0.06; $I^2 = 92\%$, $p = 0.008$), systolic blood pressure (WMD = 3.30; 95% CI: 1.27 to 5.32; $I^2 = 27\%$, $p = 0.001$), blood glucose (WMD = 7.44; 95% CI: 2.59 to 12.30; $I^2 = 75\%$, $p = 0.003$), insulin levels (WMD = 4.27; 95% CI: 3.17 to 5.37; $I^2 = 0\%$, $p < 0.001$), insulin resistance (WMD = 0.89; 95% CI: 0.15 to 1.63; $I^2 = 91\%$, $p = 0.020$), low-density lipoprotein (WMD = 5.14; 95% CI: 0.74 to 9.53; $I^2 = 49\%$, $p = 0.020$), triglycerides (WMD = 0.25; 95% CI: 0.11 to 0.40; $I^2 = 55\%$, $p < 0.001$), total testosterone (WMD = 0.40; 95% CI: 0.25 to 0.55; $I^2 = 81\%$, $p < 0.001$), and free androgen index (WMD = 1.86; 95% CI: 1.37 to 2.36; $I^2 = 83\%$, $p < 0.001$). In addition, postmenopausal women with PCOS were at increased risk of developing type 2 diabetes (OR = 3.08; 95% CI: 1.51 to 6.27; $I^2 = 78\%$, $p = 0.002$), hypertension (OR = 1.72; 95% CI: 1.04 to 2.85; $I^2 = 69\%$, $p = 0.040$), and metabolic syndrome (OR = 3.93; 95% CI: 1.17 to 13.22; $I^2 = 67\%$, $p = 0.030$). **Conclusions:** This study demonstrates that women with PCOS may have a higher risk of developing type 2 diabetes, hypertension, and metabolic syndrome after menopause. These findings highlight the need for early intervention and targeted health management strategies in postmenopausal women with PCOS. **Registration:** The study has been registered on <https://www.crd.york.ac.uk/PROSPERO/> (registration number: CRD42022370637; registration link: <https://www.crd.york.ac.uk/PROSPERO/view/CRD42022370637>).

Keywords: metabolic syndrome; postmenopause; polycystic ovary syndrome; type 2 diabetes; body mass index

1. Introduction

Menopause is the permanent cessation of menstruation resulting from the depletion of ovarian follicles and the consequent decline in estrogen and progesterone production [1,2]. As a non-pathologic physiologic process, menopause marks the end of a woman's reproductive years and transition into the later stages of life for most women [3]; therefore, the health status of menopausal women is always a public concern from either a medical or social perspective. Postmenopausal women suffer more from chronic diseases, such as hypertension, diabetes, and cardiovascular

diseases. Studies have reported the health determinants in postmenopausal women, including family history, mental factors, smoking, higher fasting glucose and triglycerides, obesity, and poor lifestyle [4,5]. Although early intervention can improve postmenopausal women's quality of life, the association between the history of polycystic ovary syndrome (PCOS) and health status at postmenopausal age is unclear.

PCOS is the most common endocrine and metabolic disorder among women at reproductive age [6]. Besides polycystic ovary morphology, PCOS is associated with



ovulatory dysfunction, infertility, and excess androgen [7], and affects patients throughout their lives. However, most studies have focused on PCOS during adolescence or child-bearing age. Few studies have focused on postmenopausal women with PCOS.

The purpose of this systematic review and meta-analysis was to investigate the health status of postmenopausal women with PCOS.

2. Methods

The protocol of this study was developed in accordance with Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA), and was registered on PROSPERO (CRD42022370637). The checklist of PRISMA Statement is shown in **Supplementary Table 1**.

2.1 Search Strategy

PubMed, Cochrane Library, Web of Science, and Embase databases were used to search the relevant studies published through July 4, 2025. Based on the medical subject headings combining with keywords, we used the following terms as searching strategy: menopause[Tiab] OR postmenopause[Tiab] OR post menopause[Tiab] OR menopausal woman[Tiab] OR postmenopausal woman[Tiab] OR postmenopausal woman[Tiab] OR menopausal women[Tiab] OR postmenopausal women[Tiab] OR postmenopausal women[Tiab], polycystic ovary syndrome[Tiab] OR polycystic ovarian syndrome[Tiab] OR polycystic ovary syndrome 1[Tiab] OR sclerocystic ovarian degeneration[Tiab] OR sclerocystic ovary syndrome[Tiab] OR sclerocystic ovar*[Tiab] OR stein leventhal syndrome[Tiab]. We also searched the references cited in the included studies.

2.2 Eligibility Criteria

Twenty studies were selected based on specific inclusion criteria: case-control/cohort-based comparisons of postmenopausal or perimenopausal women with a history of PCOS, focusing on metabolic/cardiovascular (hypertension, diabetes, metabolic syndrome), comparing somatic parameters and utilizing odds ratios (ORs) to evaluate correlations. Exclusion criteria included: (1) studies not reporting sufficient data for effect size extraction; (2) non-human research; and (3) letters, duplicates, reviews, or case report studies. The studies with the largest sample size and the longest follow-up were included in case of overlapping data [8]. The quality evaluation details of eligible studies are shown in **Supplementary Tables 2,3**.

2.3 Data Extraction

Two authors independently completed the data extraction process based on the eligibility and exclusion criteria. Data were collected as follows: (1) first author; (2) publication year; (3) country; (4) type of study; (5) sample size;

(6) number of postmenopausal women with PCOS history; and (7) age of participants.

When studies reported data in different units, standardized conversions were applied to ensure consistency across studies. Specifically, blood glucose was converted from mmol/L to mg/dL ($\times 18.018$), low-density lipoprotein (LDL) from mmol/L to mg/dL ($\times 38.669$), triglycerides from mg/dL to mmol/L ($\times 0.0113$), total testosterone from ng/mL to nmol/L ($\times 3.4671$) or from ng/dL to nmol/L ($\times 0.0347$), and waist circumference from inches to cm ($\times 2.54$). Insulin levels in mU/L were considered equivalent to $\mu\text{U/mL}$. Regarding LDL cholesterol, the measurement method (direct enzymatic assay or Friedewald calculation) was recorded when reported in the original studies. Detailed information on the extraction and conversion of units for each outcome from each included study is provided in **Supplementary Table 4**. Fasting status was reported in most studies; exceptions are noted in the table.

For dichotomous outcomes (diabetes, hypertension, cardiovascular diseases, and metabolic syndrome), the numbers of events and total participants in the postmenopausal PCOS group and the control group were extracted from each study. Pooled odds ratios (ORs) were calculated using the Inverse Variance (IV) method under a random-effects or fixed-effects model depending on heterogeneity. All extracted data were crude event counts; therefore, the pooled ORs represent unadjusted (crude) estimates. When original studies reported both crude and adjusted ORs, we consistently extracted the crude event data to ensure uniformity across all included studies. A supplementary table (**Supplementary Table 5**) documents the outcome definitions, and where available, the covariates that were adjusted for in the original studies (although these adjusted estimates were not used in the present meta-analysis).

2.4 Quality Assessment

Newcastle-Ottawa Scale (NOS) was used to appraise the qualities of the included studies [9]. Three domains were included in the NOS tool: (1) selection of representative cases and controls; (2) comparability of baseline characteristics; and (3) outcome assessment (cohort studies) or exposure assessment (case-control studies). The scores range from 0 to 9 stars. The quality of each study was categorized as low quality (zero to three stars), medium quality (four to six stars), and high quality (seven to nine stars) according to total stars [10].

2.5 Statistical Analyses

The meta-analysis was performed using Review Manager Software (RevMan 5.4.1; Cochrane Collaboration, Oxford, UK). Postmenopausal women with PCOS and controls were compared for weighted mean differences (WMDs) and their 95% confidence intervals (CIs) [11,12]. Pooled ORs were calculated to evaluate the association be-

tween PCOS and health status. The heterogeneity between the included studies was evaluated using the Q-test and the I^2 statistic. A random effect model meta-analysis was conducted if $I^2 > 50\%$, which indicated significant heterogeneity; otherwise, a fixed-effect model was applied [13]. Publication bias was assessed using funnel plots. $p < 0.05$ was considered statistically significant.

2.6 Certainty of Evidence Assessment

The certainty of evidence for each outcome was assessed using the Grading of Recommendations Assessment, Development and Evaluation (GRADE) framework. The assessment considered five domains: risk of bias (based on NOS scores), inconsistency (based on I^2 statistic), indirectness (based on population, intervention, comparator, and outcome alignment), imprecision (based on sample size and width of confidence intervals), and publication bias (based on funnel plot symmetry and Egger's/Begg's tests). The overall certainty was classified as high ($\oplus\oplus\oplus\oplus$), moderate ($\oplus\oplus\oplus\circ$), low ($\oplus\oplus\circ\circ$), or very low ($\oplus\circ\circ\circ$). Details of the GRADE assessment are provided in **Supplementary Table 6**.

3. Results

3.1 Search Results

As shown in Fig. 1, our literature search initially identified 4152 articles, of which 1481 were from Embase, 1725 were from Web of Science, 854 were from PubMed, and 92 were from Cochrane Library. After removing 1396 duplicate publications, 2756 studies were screened by title, abstract, and/or full-text. Of these, 2736 studies were excluded: 986 reviews, 235 letters or case report studies, 196 non-human studies, 1305 non-PCOS or premenopausal studies, 11 clinical trials, and 3 studies with unavailable data. Accordingly, 20 articles were included in the meta-analysis.

3.2 Study Characteristics

The 20 articles included 6331 women (1042 with PCOS history and 5289 controls) aged between 40 and 89 years. Of these studies, 9 were case-control studies, 9 cohort studies and 2 were cross-sectional studies. The detailed features of the included studies are shown in Table 1 (Ref. [11,14,15,16,17,18,19,20,21,22,23,24,25,26,27,28,29,30,31,32]) and **Supplementary Tables 7,8,9**.

3.3 Study Quality

The quality scores of all studies are shown in **Supplementary Table 3**. All studies scored four stars or above, indicating medium or high quality. To facilitate interpretation of the clinical significance of the WMDs reported below, reference values and minimal clinically important differences were considered where available. For body mass index (BMI), a difference of $\geq 1.0 \text{ kg/m}^2$ is generally considered clinically meaningful in population-based studies [33].

For waist circumference, a difference of $\geq 5.0 \text{ cm}$ is associated with increased cardiometabolic risk [34]. For systolic blood pressure (SBP), a difference of $\geq 2.0 \text{ mmHg}$ corresponds to a meaningful reduction in cardiovascular risk at the population level [35]. For metabolic parameters, reference ranges were used to contextualize the observed differences: fasting blood glucose ($3.9\text{--}5.6 \text{ mmol/L}$), triglycerides ($<1.7 \text{ mmol/L}$), and LDL ($<3.0 \text{ mmol/L}$) [36]. These clinical benchmarks are used to guide interpretation of the pooled estimates.

3.4 Anthropometric and Clinical Parameters

3.4.1 BMI

Seventeen studies reported the BMI measurement of the participants [11,14,15,16,17,18,19,20,21,22,23,26,27,28,29,30,32]. Our meta-analysis showed that postmenopausal women with a PCOS history exhibited significantly higher BMI levels compared to those without (WMD = 2.75; 95% CI: 0.98 to 4.51; $I^2 = 96\%$, $p = 0.002$), as detailed in Table 2 and Fig. 2A.

3.4.2 Waist Circumference

Waist circumference was measured across 10 studies [11,16,17,18,19,20,21,22,28,29]. As shown in Table 2 and Fig. 2B, postmenopausal women with a PCOS history had a higher waist circumference than the controls (WMD = 7.85; 95% CI: 4.12 to 11.57; $I^2 = 73\%$, $p < 0.001$).

3.4.3 Waist/Hip Ratio

Waist/hip ratio (WHR) was analyzed in 11 studies [11,14,16,17,18,21,22,23,29,30,32], of which the pooled results showed that postmenopausal women with a PCOS history exhibited significantly higher WHR than those in the control group (WMD = 0.04; 95% CI: 0.01 to 0.06; $I^2 = 92\%$, $p = 0.008$), as shown in Table 2 and Fig. 2C.

3.4.4 SBP

SBP was measured in seven studies [11,14,15,16,22,30,32]. As shown in Table 2 and Fig. 2D, postmenopausal women with a PCOS history had higher SBP than those in the control group (WMD = 3.30; 95% CI: 1.27 to 5.32; $I^2 = 27\%$, $p = 0.001$).

3.4.5 Blood Glucose

Pooled data from 10 studies [11,14,15,16,19,21,22,28,30,32] showed that postmenopausal women with a PCOS history exhibited significantly higher blood glucose levels compared with the control group (WMD = 7.44, 95% CI: 2.59 to 12.30; $I^2 = 75\%$, $p = 0.003$), as shown in Table 2 and Fig. 3A.

3.4.6 Insulin and Homeostasis Model Assessment of IR

Five studies analyzed insulin levels [11,14,21,28,30], while eight studies evaluated homeostasis model assessment of insulin resistance (HOMA-IR) [11,14,19,

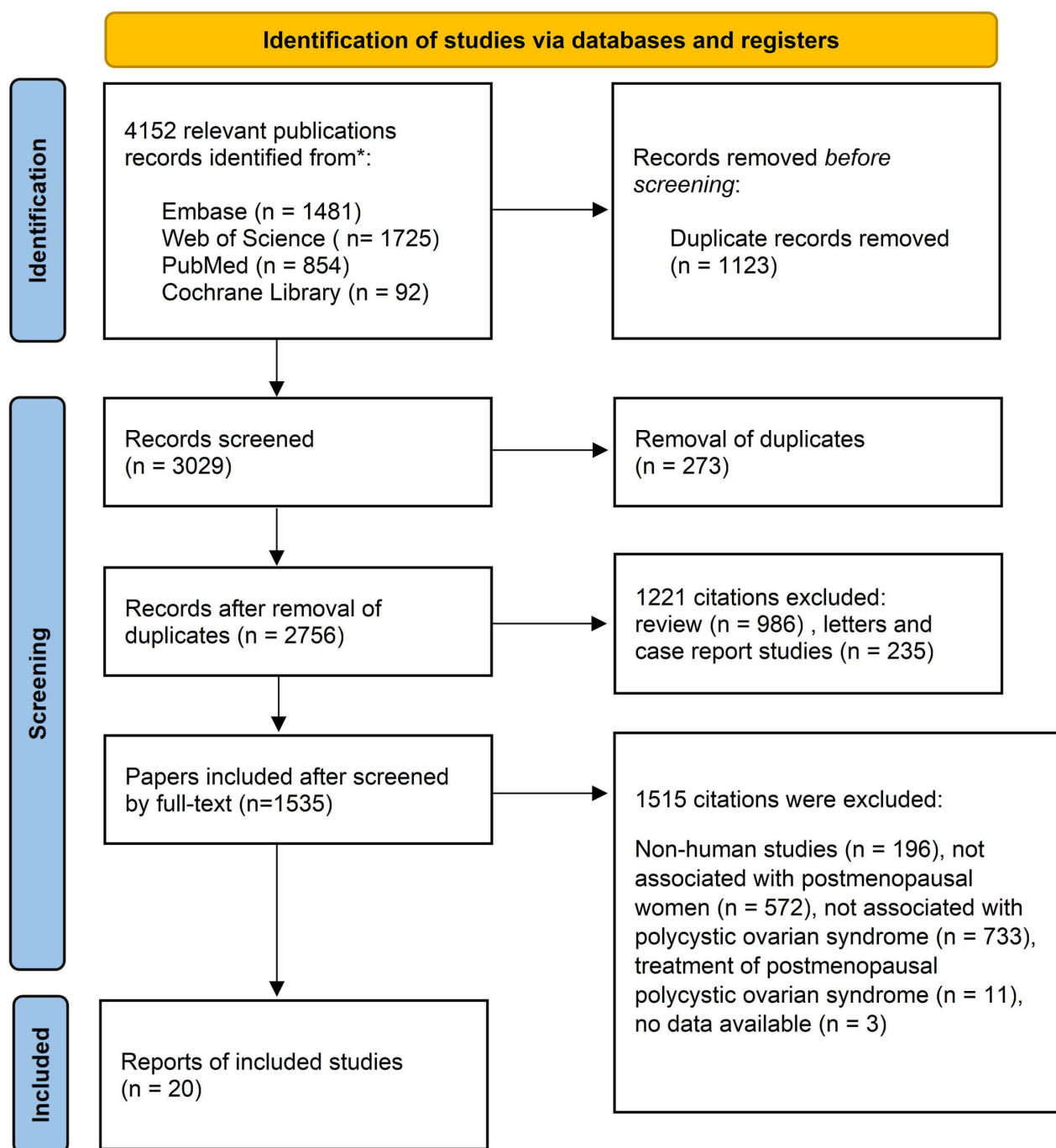


Fig. 1. PRISMA 2020 flow chart.

21,22,23,28,32]. Our meta-analysis illustrated that the postmenopausal women with a PCOS history had higher insulin levels (WMD = 4.27; 95% CI: 3.17 to 5.37; $I^2 = 0\%$, $p < 0.001$) and HOMA-IR (WMD = 0.89, 95% CI: 0.15 to 1.63; $I^2 = 91\%$, $p = 0.020$) compared to those without a history (Table 2, Fig. 3B,C).

3.4.7 Lipids

LDL levels were compared in six studies [11,14,16,19,22,32], while seven studies [11,14,16,19,22,30,32]

analyzed triglyceride levels between postmenopausal women with a PCOS history and the controls. The analysis showed that triglyceride levels (WMD = 0.25; 95% CI: 0.11 to 0.40; $I^2 = 55\%$, $p < 0.001$) and LDL levels (WMD = 5.14, 95% CI: 0.74 to 9.53; $I^2 = 49\%$, $p = 0.020$) were significantly higher in postmenopausal women with a PCOS history compared with controls (Table 2 and Fig. 3D,E).

Table 1. Characteristics of included studies.

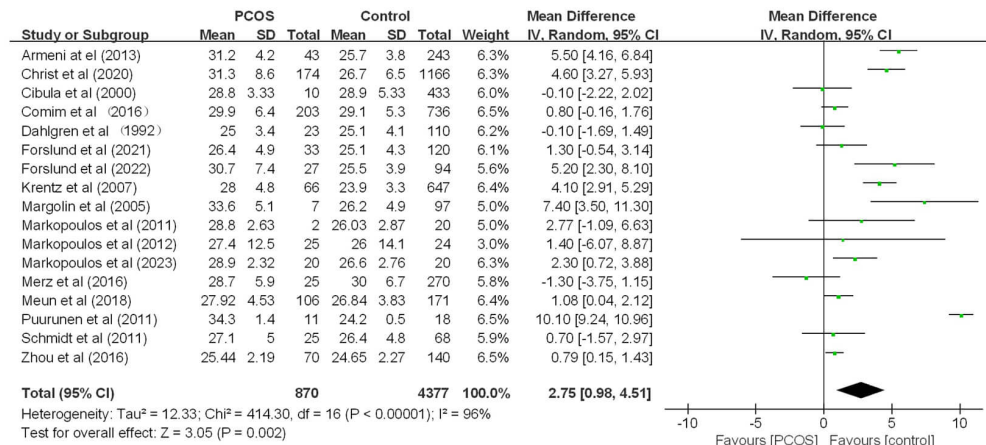
Author	Year	Country	Type	Sample size	NO. of postmenopausal women with PCOS history	Age (years)
Armeni et al. [14]	2013	Greece	Cross-sectional study	Total 286	43	48–63
Christ et al. [15]	2020	America	Cohort study	Total 1340	174	42–52
Cibula et al. [16]	2000	The Czech republic	Case-control study	Total 780	28	43–59
Comim et al. [26]	2016	The United Kingdom	Case-control study	Total 1057	221	59–75
Dahlgren et al. [27]	1992	Sweden	Cohort study	Total 165	33	40–59
Forslund et al. [17]	2021	Sweden	Cohort study	Total 153	33	40–59
Forslund et al. [18]	2022	Sweden	Cohort study	Total 121	27	52–58
Krentz et al. [19]	2007	America	Cross-sectional study	Total 713	66	51–89
Margolin et al. [20]	2005	Israel	Cohort study	Total 104	7	47–64
Markopoulos et al. [29]	2011	Greece	Case-control study	Total 40	20	50–65
Markopoulos et al. [21]	2012	Greece	Case-control study	Total 49	25	51–65
Markopoulos et al. [28]	2023	Greece	Case-control study	Total 40	20	51–63
Merz et al. [22]	2016	America	Cohort study	Total 195	25	62–74
Meun et al. [30]	2018	The Netherlands	Cohort study	Total 277	106	61–78
Puurunen et al. [23]	2011	Finland	Case-control study	Total 29	11	55–57
Schmidt et al. [11]	2011	Sweden	Cohort study	Total 127	32	61–79
Schmidt et al. [24]	2012	Sweden	Cohort study	Total 93	25	61–79
Talbott et al. [25]	2008	America	Case-control study	Total 99	34	50–54
Yin et al. [31]	2018	America	Case-control study	Total 453	42	45–54
Zhou et al. [32]	2016	China	Case-control study	Total 210	70	55–57

Table 2. Meta-analysis of anthropometric and clinical testing indicators in postmenopausal women with a history of PCOS.

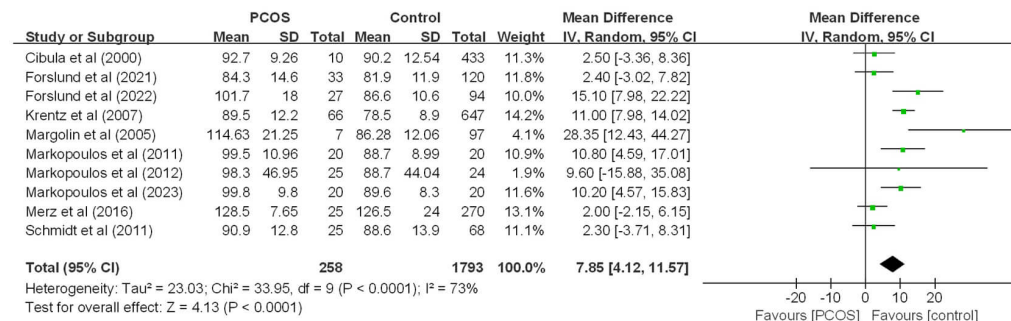
Group	No. of studies	No. of postmenopausal patients with PCOS history	No. of controls	WMD	<i>p</i> -value	<i>p</i> -value for heterogeneity	<i>I</i> ² (%)
BMI (kg/m ²)	17	870	4377	2.75 (0.98–4.51)	0.002	<0.001	96
Waist circumference (cm)	10	258	1793	7.85 (4.12–11.57)	<0.001	<0.001	73
Waist/hip ratio	11	395	1601	0.04 (0.01–0.06)	0.008	<0.001	92
SBP (mmHg)	7	453	2491	3.30 (1.27–5.32)	0.001	0.220	27
Blood glucose (mg/dL)	10	564	3182	7.44 (2.59–12.30)	0.003	<0.001	75
Insulin (μU/mL)	5	136	465	4.27 (3.17–5.37)	<0.001	0.450	0
IR	8	285	1430	0.89 (0.15–1.63)	0.020	<0.001	91
LDL (mg/dL)	6	239	1801	5.14 (0.74–9.53)	0.020	0.080	49
Triglyceride (mmol/L)	7	345	1972	0.25 (0.11–0.40)	<0.001	0.040	55
Total testosterone (nmol/L)	14	520	2152	0.40 (0.25–0.55)	<0.001	<0.001	81
FAI	9	335	899	1.86 (1.37–2.36)	<0.001	<0.001	83

BMI, body mass index; SBP, systolic blood pressure; LDL, low-density lipoprotein; FAI, free androgen index; WMDs, weighted mean differences; IR, insulin resistance.

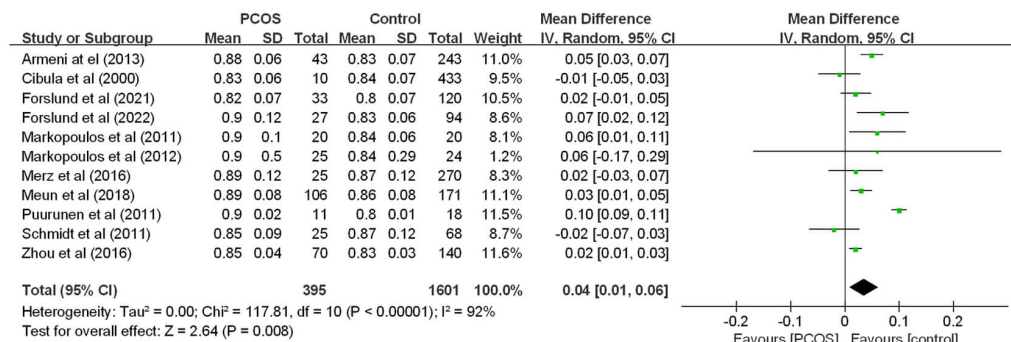
(A)



(B)



(C)



(D)

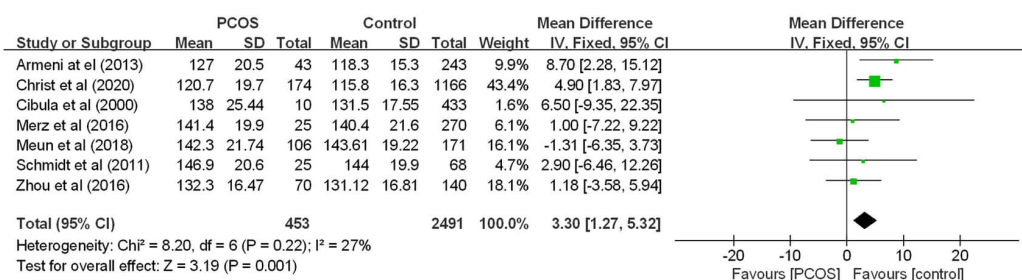


Fig. 2. Forest plot 1. (A) BMI (kg/m^2). (B) Waist circumference (cm). (C) Waist/hip ratio. (D) Systolic blood pressure (mmHg). WMDs with 95% CIs are shown for postmenopausal women with a history of PCOS compared with controls. A random-effects model was used for outcomes with significant heterogeneity ($I^2 > 50\%$), and a fixed-effects model was used for outcomes with $I^2 \leq 50\%$.

3.4.8 Total Testosterone

A meta-analysis of 14 studies [14,17,19,20,21,23,24,25,27,28,29,30,31,32] revealed that postmenopausal women with a history of PCOS had significantly higher

testosterone levels compared to those without (WMD = 0.40; 95% CI: 0.25 to 0.55; $I^2 = 81\%$, $p < 0.001$), as shown in Table 2 and Fig. 3F.

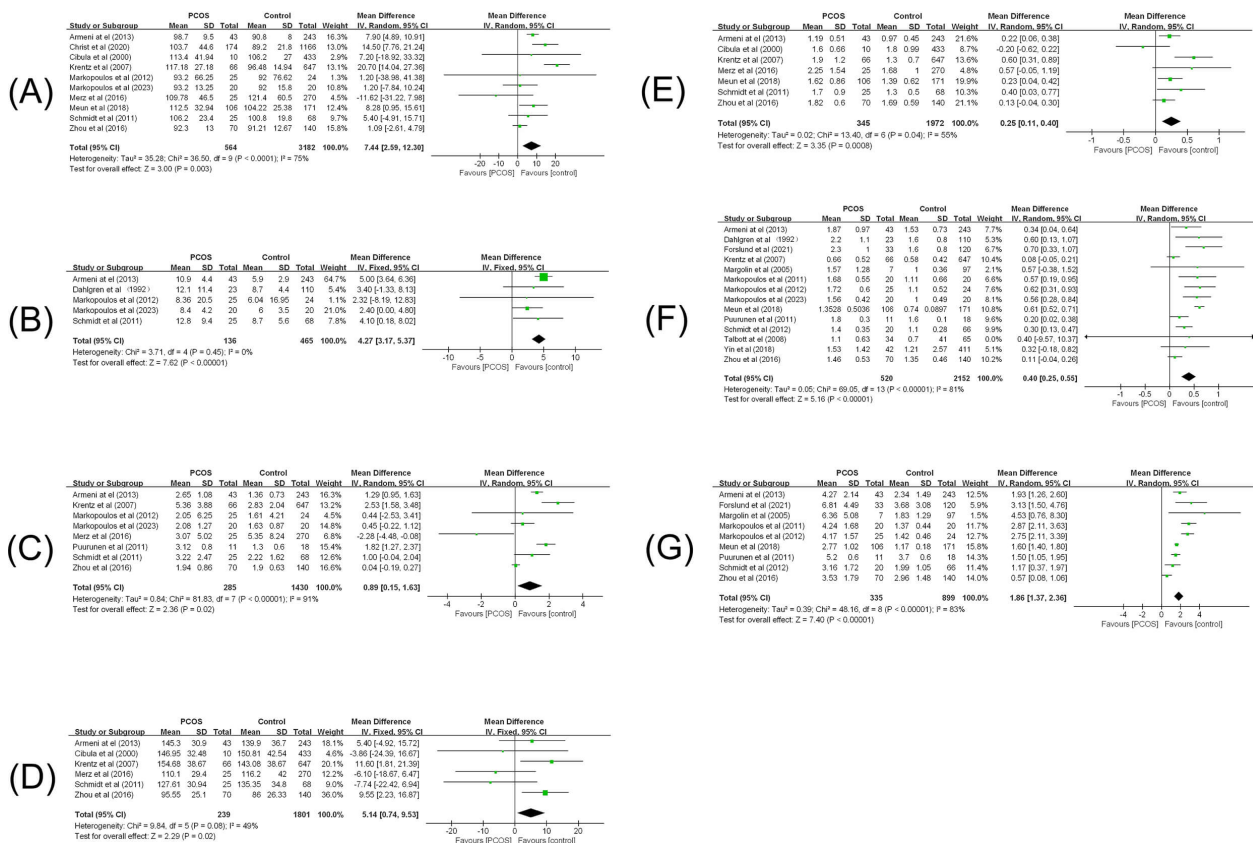


Fig. 3. Forest plot 2. (A) Blood Glucose (mg/dL). (B) Insulin (μU/mL). (C) IR. (D) LDL (mg/dL). (E) Triglyceride (mmol/L). (F) Total Testosterone (nmol/L). (G) FAI. WMDs with 95% CIs are shown for postmenopausal women with a history of PCOS compared with controls. A random-effects model was used for outcomes with significant heterogeneity ($I^2 > 50\%$), and a fixed-effects model was used for outcomes with $I^2 \leq 50\%$.

3.4.9 Free Androgen Index

Nine studies [14,17,20,21,23,24,29,30,32] reported the level of free androgen index (FAI). Compared with the controls, postmenopausal women with a PCOS history had significantly higher FAI (WMD = 1.86, 95% CI: 1.37 to 2.36; $I^2 = 83\%$, $p < 0.001$), as shown in Table 2 and Fig. 3G.

3.5 Disease Outcomes

3.5.1 Risk of Type 2 Diabetes

Nine studies regarding type 2 diabetes were included [11,16,18,20,22,23,26,27,30]. A history of PCOS was associated with a significantly higher pooled risk of type 2 diabetes in postmenopausal women compared with controls (OR = 3.08; 95% CI: 1.51 to 6.27; $I^2 = 78\%$, $p = 0.002$), as shown in Table 3 and Fig. 4A.

3.5.2 Risk of Metabolic Syndrome

Based on a meta-analysis of three studies [18,20,22], a history of PCOS significantly increased the risk of developing metabolic syndrome in postmenopausal women (OR = 3.93, 95% CI: 1.17 to 13.22; $I^2 = 67\%$, $p = 0.030$), as shown in Table 3 and Fig. 4B.

3.5.3 Risk of Hypertension

Eight studies reported the outcome of hypertension [11,16,18,20,22,27,30,32]. As shown in Table 3 and Fig. 4C, the risk of hypertension was higher in postmenopausal women with a PCOS history than in those without (OR = 1.72; 95% CI: 1.04 to 2.85; $I^2 = 69\%$, $p = 0.040$).

3.5.4 Risk of Cardiovascular Diseases

Cardiovascular diseases were reported in five studies [11,16,18,22,30], and no significant difference was observed between postmenopausal women with a PCOS history and controls (OR = 1.75; 95% CI: 0.83 to 3.66; $I^2 = 57\%$, $p = 0.140$), as shown in Table 3 and Fig. 4D.

3.6 Sensitivity Analysis

Leave-one-out sensitivity analyses showed that the pooled estimate for type 2 diabetes remained significant across all iterations (OR = 2.94–4.06). Findings for hypertension and cardiovascular diseases were not robust; results lost significance when omitting Forslund 2022 (hypertension) or Cibula 2000, Mez 2016, and Meun 2018 (cardiovascular diseases). For metabolic syndrome, all sensitivity analyses yielded non-significant estimates, reflecting the

Table 3. Meta-analysis of disease risk in postmenopausal women with a history of PCOS.

Group	No. of studies	No. of Postmenopausal patients with PCOS	No. of controls	OR	<i>p</i> -value	<i>p</i> -value for heterogeneity	<i>I</i> ² (%)
Diabetes	9	108	355	3.08 (1.51–6.27)	0.002	<0.001	78
Metabolic syndrome	3	34	188	3.93 (1.17–13.22)	0.030	0.050	67
Hypertension	8	169	711	1.72 (1.04–2.85)	0.040	0.002	69
Cardiovascular disease	5	34	198	1.75 (0.83–3.66)	0.140	0.050	57

OR, odds ratios.

limited number of included studies. Collectively, these sensitivity analyses demonstrate that type 2 diabetes findings are stable; conversely, the results for hypertension, cardiovascular diseases, and metabolic syndrome should be interpreted with caution given the impact of individual studies and limited statistical power.

3.7 Publication Bias

Publication bias was assessed visually using funnel plots (Figs. 5,6,7), and where appropriate, statistically using Egger's linear regression test and Begg's rank correlation test. For outcomes with ≥ 10 included studies, both tests were performed (Supplementary Table 10). No significant publication bias was detected for BMI (Egger's $p = 0.855$; Begg's $p = 0.621$), total testosterone (Egger's $p = 0.752$; Begg's $p = 0.477$), WHR (Egger's $p = 0.779$; Begg's $p = 0.586$), or blood glucose (Egger's $p = 0.339$; Begg's $p = 0.788$).

For outcomes with fewer than 10 included studies, formal statistical tests for publication bias were not performed due to insufficient statistical power, as recommended by the Cochrane Handbook [37]. In these cases, funnel plots were inspected visually, but the findings should be interpreted with caution, particularly for outcomes with very few studies (e.g., metabolic syndrome, which included only three studies). Visual inspection of funnel plots for these outcomes did not reveal obvious asymmetry; however, publication bias cannot be ruled out solely on the basis of funnel plot symmetry when the number of studies is small.

Collectively, these findings indicate that publication bias likely had a minimal impact on most outcomes, although potential bias cannot be excluded for outcomes with few studies.

3.8 Subgroup Analyses

Given the substantial heterogeneity observed in several primary meta-analyses ($I^2 > 75\%$), we performed subgroup analyses based on study design (cohort studies, case-control studies and cross-sectional studies) to explore potential sources of heterogeneity for these outcomes, includ-

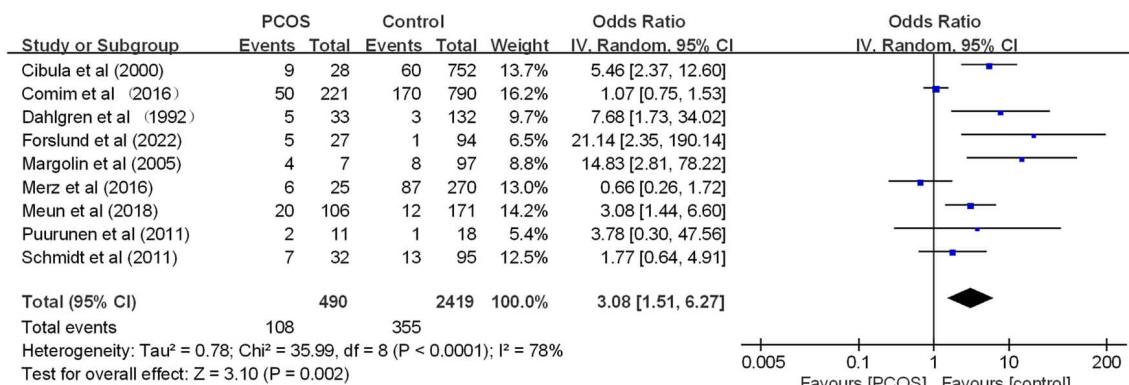
ing BMI, WHR, total testosterone, blood glucose, HOMA-IR, FAI, and type 2 diabetes risk.

Table 4 summarizes the results of subgroup analyses by study design. For most outcomes, the direction of effect remained consistent across subgroups. The test for subgroup differences revealed a significant difference only for total testosterone (p for interaction = 0.043). Both cohort studies (WMD = 0.53, 95% CI: 0.32 to 0.74) and case-control studies (WMD = 0.37, 95% CI: 0.16 to 0.57) showed significantly higher total testosterone in postmenopausal women with PCOS history, whereas cross-sectional studies ($n = 2$) did not show a significant difference (WMD = 0.17, 95% CI: -1.41 to 1.75), with a wide confidence interval. For other outcomes, including BMI, WHR, blood glucose, FAI, IR, and diabetes, no significant differences were observed between subgroups (p for interaction > 0.05 for all).

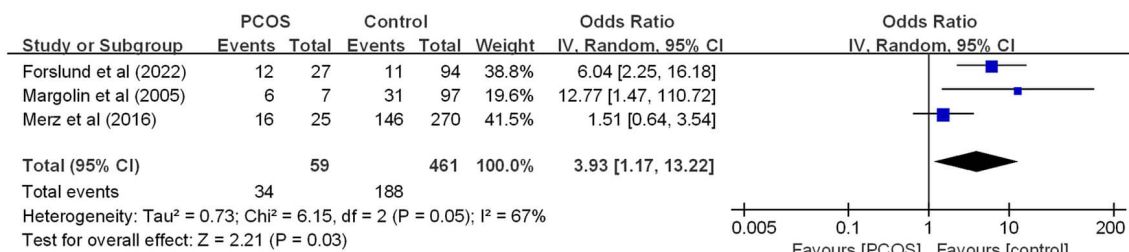
Notably, heterogeneity was reduced in certain subgroups. For blood glucose, heterogeneity was completely eliminated in case-control studies ($I^2 = 0\%$), whereas moderate heterogeneity persisted in cohort studies ($I^2 = 58.8\%$). For WHR, heterogeneity was lower in cohort studies ($I^2 = 48.3\%$) than in case-control studies ($I^2 = 96.7\%$). For FAI, heterogeneity was moderate in cohort studies ($I^2 = 55.9\%$) but remained high in case-control studies ($I^2 = 92.6\%$). For total testosterone, heterogeneity was moderate across subgroups (case-control: $I^2 = 63.6\%$; cohort: $I^2 = 61.5\%$; cross-sectional: $I^2 = 57.9\%$). However, for BMI, IR, and diabetes, heterogeneity remained high in all subgroups ($I^2 > 70\%$), suggesting that factors other than study design contribute to the heterogeneity of these outcomes.

For the outcome of type 2 diabetes risk, the association remained consistent across study designs, with no significant subgroup difference (p for interaction = 0.590). These findings suggest that study design may partially explain the heterogeneity observed in some primary analyses, particularly for total testosterone, whereas for other outcomes, study design did not appear to be a major source of heterogeneity.

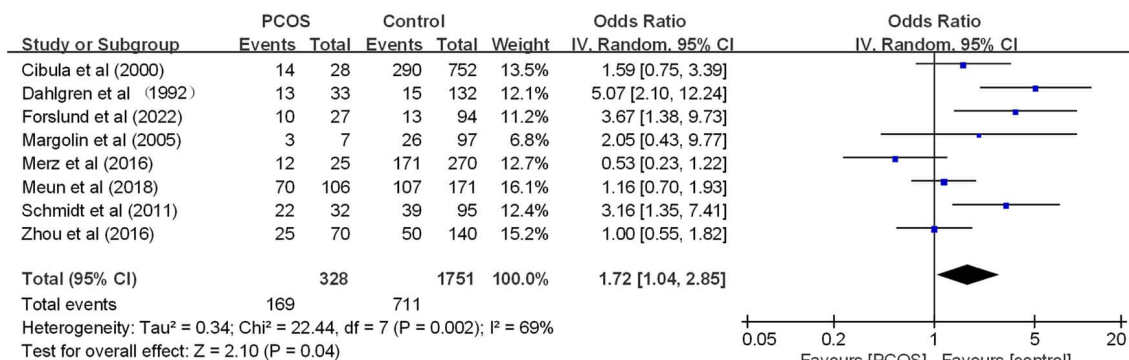
(A)



(B)



(C)



(D)

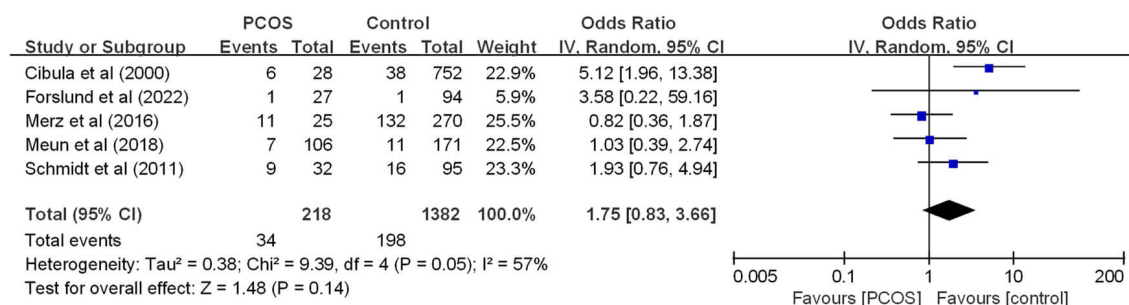


Fig. 4. Forest plot 3. (A) Diabetes. (B) Metabolic syndrome. (C) Hypertension. (D) Cardiovascular diseases. ORs with 95% CIs are shown for postmenopausal women with a history of PCOS compared with controls. A random-effects model was used for outcomes with significant heterogeneity ($I^2 > 50\%$), and a fixed-effects model was used for outcomes with $I^2 \leq 50\%$.

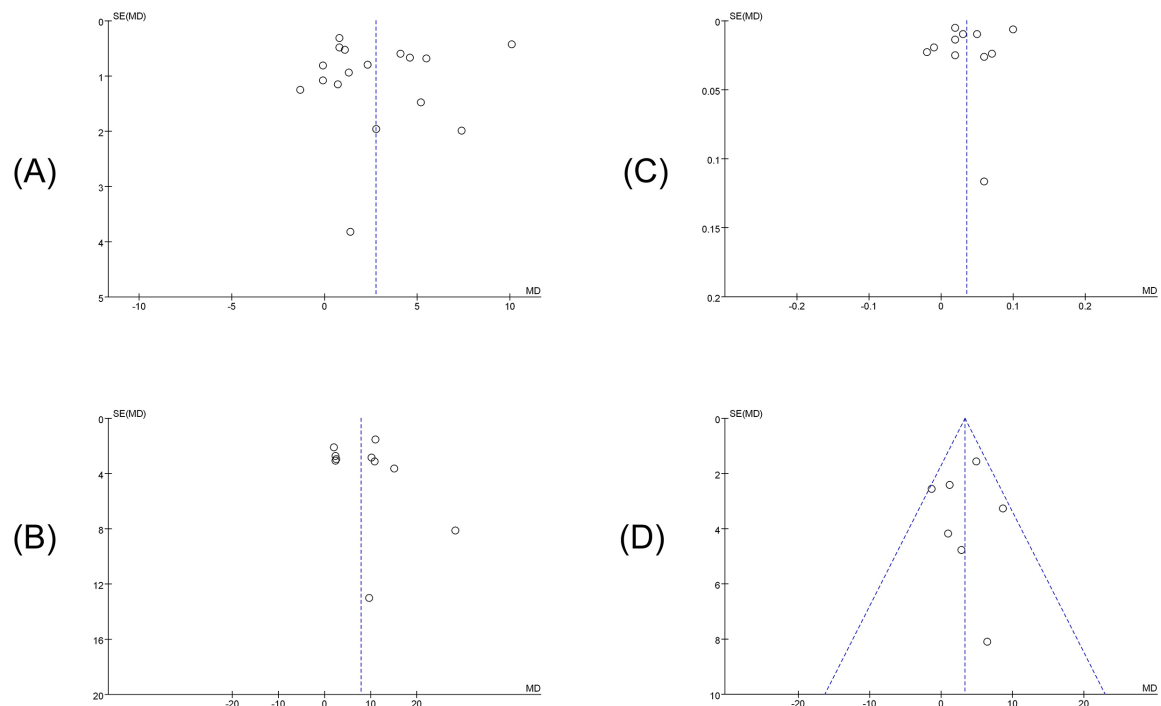


Fig. 5. Funnel plot 1. (A) BMI (kg/m²). (B) Waist circumference (cm). (C) WHR. (D) SBP (mmHg). Each dot represents an individual study. The vertical line indicates the pooled effect estimate, and the diagonal lines represent pseudo-95% CIs.

Table 4. Subgroup analyses by study design for selected outcomes.

Outcome	Subgroup	No. of studies	WMD/OR (95% CI)	<i>I</i> ² (%)	<i>p</i> for heterogeneity	<i>p</i> for interaction
BMI (kg/m ²)	Case-control	7	2.71 (−0.62 to 6.03)	98.2	<0.001	0.074
	Cohort	8	2.15 (−0.23 to 4.54)	84.5	<0.001	
	Cross-sectional	2	4.76 (−4.12 to 13.64)	57.4	0.126	
WHR	Case-control	5	0.04 (−0.02 to 0.10)	96.7	<0.001	0.271
	Cohort	5	0.02 (−0.01 to 0.06)	48.3	0.102	
	Cross-sectional	1	0.05 (0.03 to 0.07)	—	—	
Blood glucose (mg/dL)	Case-control	4	1.21 (−0.23 to 2.65)	0	0.977	0.237
	Cohort	4	7.54 (−6.28 to 21.36)	58.8	0.064	
	Cross-sectional	2	10.25 (−119.31 to 139.82)	96.9	<0.001	
Total testosterone (nmol/L)	Case-control	7	0.37 (0.16 to 0.57)	63.6	0.012	0.043
	Cohort	5	0.53 (0.32 to 0.74)	61.5	0.034	
	Cross-sectional	2	0.17 (−1.41 to 1.75)	57.9	0.123	
FAI	Case-control	4	1.89 (0.15 to 3.64)	92.6	<0.001	0.997
	Cohort	4	1.88 (0.21 to 3.56)	55.9	0.079	
	Cross-sectional	1	1.93 (1.26 to 2.60)	—	—	
IR	Case-control	4	0.73 (−0.63 to 2.09)	91.4	<0.001	0.218
	Cohort	2	−0.49 (−21.24 to 20.26)	85.7	0.008	
	Cross-sectional	2	1.83 (−5.98 to 9.63)	82.8	0.016	
Diabetes	Case-control	3	2.45 (0.24 to 25.57)	84.6	0.002	0.590
	Cohort	6	3.69 (0.96 to 14.25)	72.8	0.003	

WHR, Waist/hip ratio.

3.9 Certainty of Evidence

The certainty of evidence for each outcome was assessed using the GRADE framework. As summarized in **Supplementary Table 6**, the certainty varied across out-

comes. High certainty (⊕⊕⊕⊕) was achieved only for waist circumference, reflecting consistent findings across studies with no serious downgrading factors. Moderate certainty (⊕⊕⊕○) was observed for BMI, WHR, SBP, LDL,

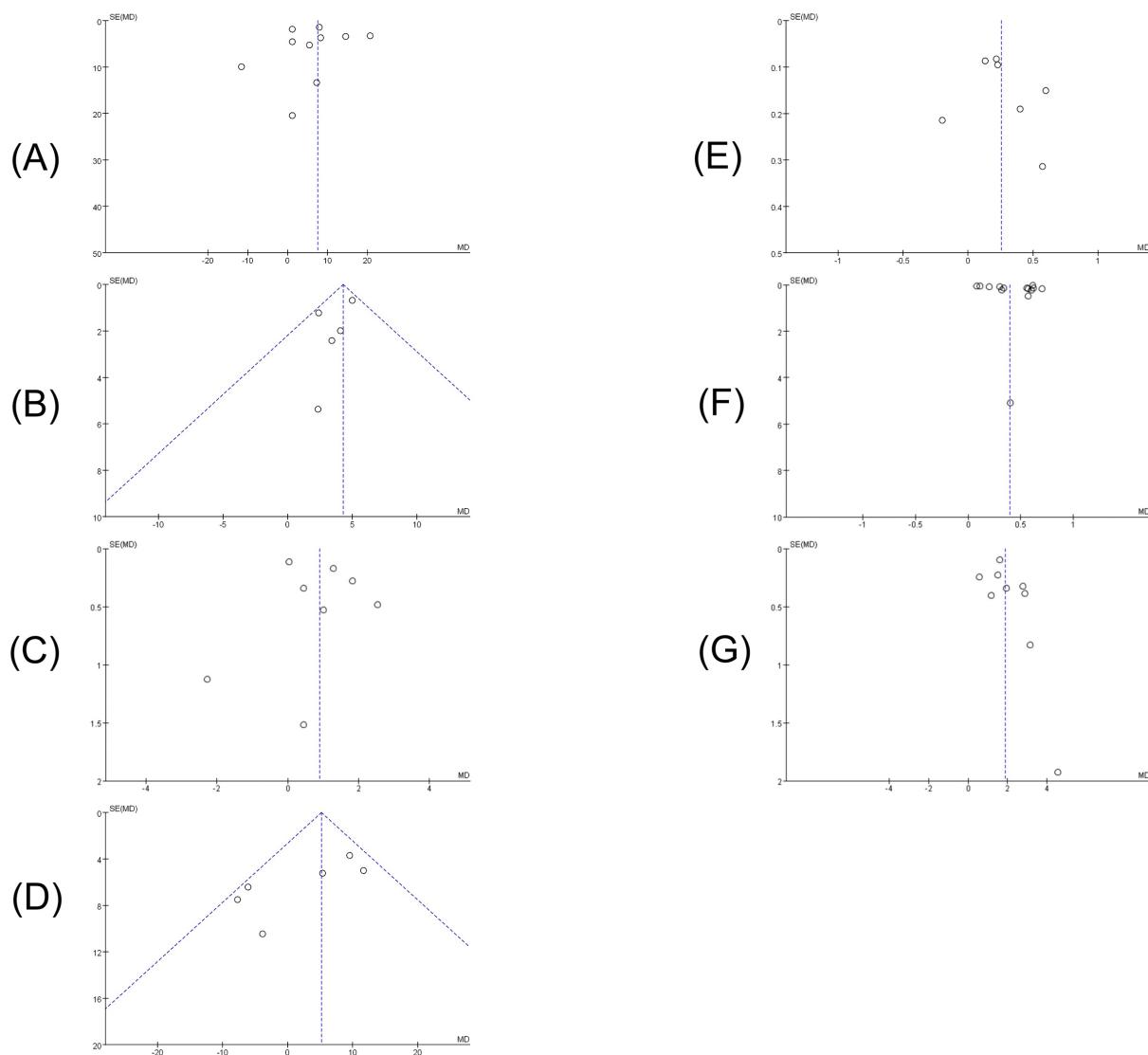


Fig. 6. Funnel plot 2. (A) Blood glucose (mg/dL). (B) Insulin ($\mu\text{U/mL}$). (C) IR. (D) LDL (mg/dL). (E) Triglyceride (mmol/L). (F) Total testosterone (nmol/L). (G) FAI. Each dot represents an individual study. The vertical line indicates the pooled effect estimate, and the diagonal lines represent pseudo-95% CIs.

total testosterone, type 2 diabetes, hypertension, and cardiovascular diseases. Downgrading was primarily due to substantial heterogeneity ($I^2 > 75\%$) for BMI, WHR, total testosterone, and type 2 diabetes, and due to imprecision (limited sample size or wide confidence intervals) for SBP, LDL, hypertension, and cardiovascular diseases.

Low certainty ($\oplus\oplus\circ\circ$) was assigned to blood glucose, insulin, IR, triglycerides, FAI, and metabolic syndrome. These outcomes were downgraded due to substantial heterogeneity combined with imprecision (limited sample size or wide confidence intervals). For metabolic syndrome, the low certainty was primarily due to the small number of included studies ($n = 3$) and wide CIs.

4. Discussion

Due to the increase in life expectancy, approximately one third of a woman's life occurs in the postmenopausal stage [38,39]. While some women experience the menopausal transition period without symptoms, most face increased health risks afterward, including cardiovascular disease, osteoporosis, diabetes, metabolic syndrome, and cancer [31,40,41]. PCOS is a chronic, multi-faceted hormonal disorder that impacts women throughout their lives. Besides causing infertility, PCOS is associated with higher risks of pregnancy complications, including miscarriage, hypertensive disorders, and gestational diabetes [42,43,44]. However, the effects of PCOS on the health outcomes of women at postmenopausal age have not been clearly elucidated.

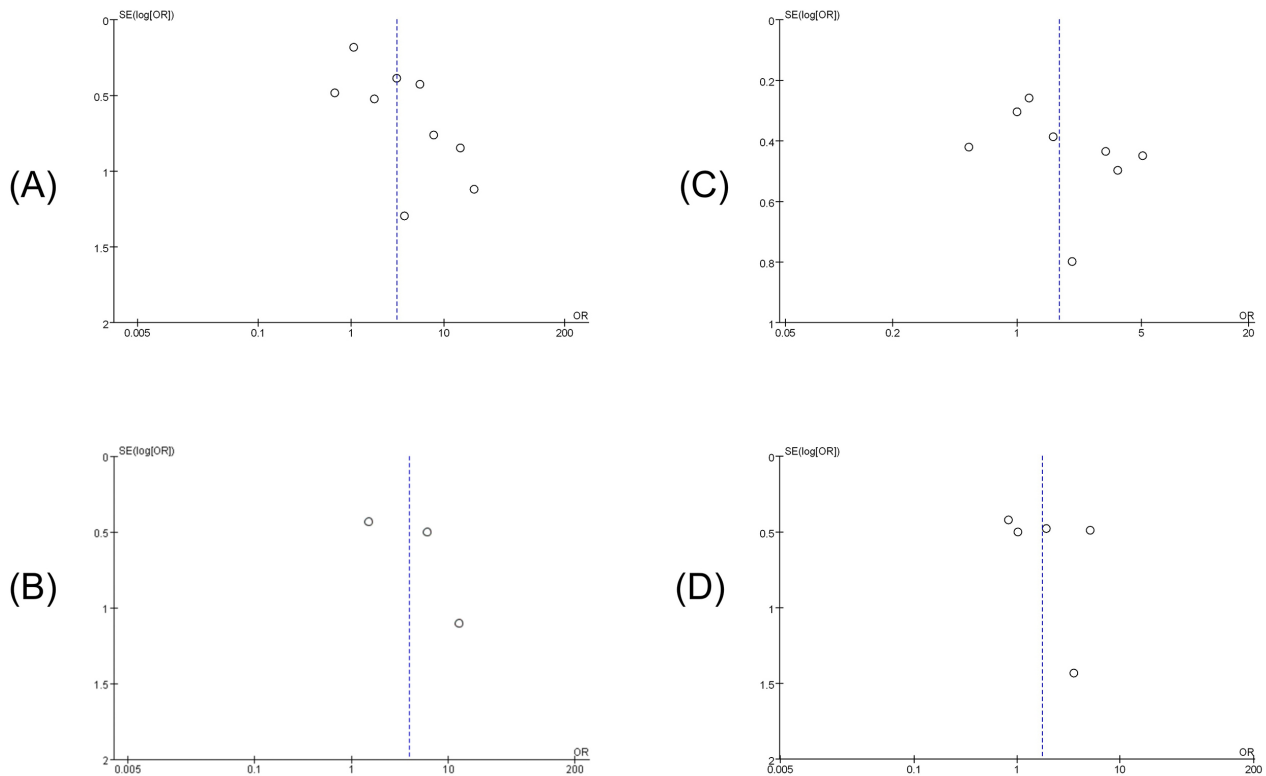


Fig. 7. Funnel plot 3. (A) Diabetes. (B) Metabolic Syndrome. (C) Hypertension. (D) Cardiovascular diseases. Each dot represents an individual study. The vertical line indicates the pooled effect estimate, and the diagonal lines represent pseudo-95% CIs.

This study provided a comprehensive literature review comparing health outcomes between postmenopausal women with and without a PCOS history, and found that the former group exhibited significantly higher metabolic and cardiovascular risk factors. Specifically, participants with a PCOS history presented with higher BMI, waist circumference, WHR, SBP, blood glucose, insulin, IR, triglycerides, LDL, total testosterone, and FAI. In addition, postmenopausal women with a PCOS history had an increased incidence of diabetes, metabolic syndrome, and hypertension. These findings indicate that postmenopausal women with a history of PCOS are face higher risks of obesity and associated health complications. Therefore, establishing long-term, proactive health management strategies for these women is essential.

4.1 Lipid Profile Alterations

Data regarding lipid changes in postmenopausal women with a PCOS history remains limited and inconsistent [38,45]. Although some studies indicate no significant differences in lipid profiles between groups [16], others have found higher triglycerides and lower HDL levels in women with PCOS [22,25]. In the present meta-analysis, both triglyceride and LDL levels were significantly elevated in the PCOS group compared with controls. This suggests that reducing triglyceride and LDL levels could represent a therapeutic target for mitigating adverse outcomes.

4.2 Androgen Profiles

Androgens play a central role in the development of metabolic and cardiovascular disorders in postmenopausal women. Although sex hormone-binding globulin (SHBG)-bound testosterone has limited biological activity, free testosterone is the primary bioactive androgen [46]. FAI is a reliable clinical marker of testosterone bioactivity [47]. Previous studies have yielded conflicting findings: some reported comparable FAI between postmenopausal women with and without PCOS [16], whereas others found significantly higher FAI in the PCOS group despite similar total testosterone levels [32]. The present meta-analysis demonstrated that both total testosterone and FAI were significantly higher in postmenopausal women with a PCOS history than in controls, indicating that hyper androgenism persists beyond menopause. In parallel, we observed higher BMI, waist circumference, and WHR in the PCOS group, which may reduce hepatic SHBG synthesis and consequently increase free testosterone. Therefore, weight management may help down regulate free androgen levels and potentially reduce disease risk in this population, alongside lifestyle modifications.

4.3 Glucose Metabolism and IR

IR is a well-recognized pathophysiological driver of PCOS [48,49]. IR and compensatory hyper insulinemia increase the risk of impaired glucose tolerance and type 2

diabetes; however, few studies have focused on these outcomes specifically in postmenopausal women with PCOS history. Earlier work, including a study by Cibula et al. [16] showed that women with PCOS had higher insulin levels and an increased prevalence of type 2 diabetes compared with controls, despite showing no significant differences in fasting glucose levels [25]. Aligning with these findings, our meta-analysis revealed that postmenopausal women with a PCOS history had elevated blood glucose, insulin, and HOMA-IR levels, along with a significantly increased prevalence of type 2 diabetes and metabolic syndrome. These findings suggest a heightened risk of glucose metabolism disorders in this population, although these estimates should be viewed with caution due to small sample sizes and significant regional or racial heterogeneity across studies.

4.4 Cardiovascular Risk

The association between PCOS and cardiovascular disease remains a subject of debate [50,51,52,53]. Some studies have reported that clinical features of PCOS are independent risk factors for cardiovascular disease in postmenopausal women [53,54,55], whereas others found no difference in the prevalence of stroke or myocardial infarction between older women with PCOS and age-matched controls [22,24]. Our study found a higher risk of hypertension but no significant increase in cardiovascular disease risk after menopause, which is consistent with some previous studies.

4.5 Clinical Relevance of the Findings

The clinical relevance of the observed differences warrants consideration. The pooled WMD of 2.75 kg/m² for BMI exceeds the commonly accepted threshold for a clinically meaningful difference (≥ 1.0 kg/m²), indicating that the higher BMI observed in postmenopausal women with PCOS history is not only statistically significant but also clinically relevant [33]. Similarly, the WMD of 7.85 cm for waist circumference surpasses the 5.0 cm threshold associated with increased cardiometabolic risk, suggesting a meaningful elevation in central adiposity [34]. For SBP, the observed WMD of 3.30 mmHg, while modest, corresponds to a population-level change in cardiovascular risk [35]. Regarding metabolic parameters, the WMD of 7.44 mg/dL (approximately 0.41 mmol/L) for blood glucose, though modest, may contribute to increased risk of progression to type 2 diabetes over time, particularly given the concurrent elevation in insulin and IR. The WMD of 0.25 mmol/L for triglycerides represents a clinically relevant difference, as it approaches the threshold distinguishing normal from borderline-high levels [56]. Collectively, these findings suggest that the adverse metabolic profile observed in postmenopausal women with a PCOS history carries meaningful clinical implications.

4.6 Subgroup Analyses and Sources of Heterogeneity

Substantial heterogeneity ($I^2 > 75\%$) was observed for several primary outcomes. To explore potential sources, we conducted subgroup analyses stratified by study design (case-control, cross-sectional or cohort). These analyses revealed that study design partially accounted for the heterogeneity, particularly for total testosterone and blood glucose. For total testosterone, a significant subgroup difference was observed (p for interaction = 0.043), with significant associations in case-control (WMD = 0.37, 95% CI: 0.16 to 0.57) and cohort studies (WMD = 0.53, 95% CI: 0.32 to 0.74), but not in cross-sectional studies (WMD = 0.17, 95% CI: -1.41 to 1.75). For blood glucose, heterogeneity was completely eliminated in case-control studies ($I^2 = 0\%$), whereas cohort ($I^2 = 58.8\%$) and cross-sectional studies ($I^2 = 96.9\%$) showed considerable heterogeneity, suggesting that study design is a key source of variability for this outcome. These findings support the robustness of the primary results while highlighting the importance of considering study design when interpreting the evidence.

4.7 Certainty of Evidence

The GRADE assessment revealed moderate to low certainty for most outcomes in this meta-analysis. High certainty ($\oplus\oplus\oplus\oplus$) was achieved only for waist circumference, indicating a well-established association with PCOS history. Moderate certainty ($\oplus\oplus\oplus\circ$) was achieved for BMI, WHR, SBP, LDL, total testosterone, type 2 diabetes, hypertension, and cardiovascular diseases, with downgrading primarily due to substantial heterogeneity or imprecision. Low certainty ($\oplus\oplus\circ\circ$) was observed for blood glucose, insulin, IR, triglycerides, FAI, and metabolic syndrome, reflecting the combined impact of heterogeneity, limited sample sizes, and wide CIs. These findings underscore the need for larger, well-designed studies to confirm the observed associations.

4.8 Study Limitations

This study had several limitations. First, as with any systematic review and meta-analysis, our findings are constrained by the quality and sample sizes of the included studies. All included studies were observational in design (case-control, cross-sectional or cohort), which limits causal inference and introduces potential for residual confounding. Second, substantial heterogeneity persisted for several outcomes despite subgroup analyses, and the small number of studies in some subgroups limited the ability to perform further stratified analyses. Third, for the outcome of type 2 diabetes, formal statistical tests for publication bias were not performed because fewer than 10 studies were included ($n = 9$). Visual inspection of the funnel plot did not reveal obvious asymmetry; however, publication bias cannot be ruled out given the small number of studies. This result should therefore be interpreted with caution. Fourth, variations in PCOS diagnostic criteria and outcome definitions

across studies may have contributed to heterogeneity. Fifth, the predominance of studies from Western populations limits the generalizability of our findings to other ethnic and geographic groups. Sixth, the pooled ORs for disease outcomes were calculated from crude event counts without adjustment for potential confounders (e.g., age, BMI, smoking, or hypertension). Although some original studies reported adjusted ORs, we chose to use crude data uniformly across all studies to maintain methodological consistency. This approach may have introduced residual confounding and should be considered when interpreting the magnitude of the associations. Future meta-analyses with access to individual participant data or consistently adjusted estimates would help to validate these findings. Finally, although we performed leave-one-out sensitivity analyses to assess the robustness of the pooled estimates for disease outcomes, the results for hypertension and cardiovascular diseases were sensitive to the exclusion of individual studies, and the number of studies for metabolic syndrome was too small to yield stable estimates. These findings highlight the need for larger, well-designed studies to confirm the observed associations for these outcomes.

5. Conclusions

To the best of our knowledge, this is the first systematic review and meta-analysis to investigate the relationship between health outcomes and PCOS history in postmenopausal women. Our findings suggest that postmenopausal women with a history of PCOS are significantly more likely to develop type 2 diabetes, metabolic syndrome, and hypertension, but not cardiovascular diseases, which may be influenced by concurrent obesity, dyslipidemia, and elevated FAI. Based on these findings, we recommend that clinical management for this population prioritize weight control, lipid lowering, and glycemic management. In addition, regular screening for diabetes, hypertension, and cardiovascular diseases should be considered in routine clinical practice for postmenopausal and perimenopausal women with a history of PCOS. Adoption of a healthy lifestyle may further reduce adverse outcomes in this population.

Availability of Data and Materials

All data and methods supporting the findings of this study are available from the corresponding authors upon reasonable request.

Author Contributions

WJZ and XCS designed the study and took responsibility for the integrity of the data and the accuracy of the data analysis. HRW and XGW contributed to statistical analysis and manuscript writing. MCL, JL and YXL completed the screening of the literature and contributed to drafting the manuscript. WJZ and XCS revised

the manuscript. All authors read and approved the final manuscript. All authors have participated sufficiently in the work and agreed to be accountable for all aspects of the work.

Ethics Approval and Consent to Participate

Not applicable.

Acknowledgment

Not applicable.

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Conflicts of Interest

The authors declare no conflicts of interest.

Supplementary Material

Supplementary material associated with this article can be found, in the online version, at <https://doi.org/10.31083/CEOG50107>.

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