

¹ Resting energy expenditure of women with and without polycystic
² ovary syndrome: a systematic review and meta-analysis*

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²⁸ **Abstract**

²⁹ Context: Polycystic ovary syndrome (PCOS) is common in reproductive-age women, who often have higher
³⁰ BMI classification. This is assumed to stem from lower resting energy expenditure (REE), influencing
³¹ lifestyle intervention guidelines. However, evidence for reduced REE in women with PCOS compared with
³² those without is inconsistent. Objective: To systematically search and meta-analyse the existing literature
³³ to estimate and describe the difference in REE between women with and without PCOS. Data Sources: A
³⁴ systematic search was conducted using PubMed, Medline and Web of Science databases of published research
³⁵ from January 1990 to January 2025. Study Selection: Studies that measured REE in women living with
³⁶ PCOS, both with and without control arms of women without PCOS, were included. Data Extraction:
³⁷ Bibliometric, demographic, and REE data was extracted by one investigator and checked in triplicate. Data
³⁸ Synthesis: Thirteen studies were included in a Bayesian arm-based multiple condition comparison (i.e.,
³⁹ network) type meta-analysis model with informative priors to compare both mean REE, and between person
⁴⁰ variation in REE, between women with and without PCOS. Mean REE differed between groups by 30
⁴¹ kcal/day [95% quantile interval: -47 to 113 kcal/day] and the contrast ratio for between person standard
⁴² deviations was 0.98 [95% quantile interval: 0.71 to 1.33]. Conclusions: These findings indicate that REE does
⁴³ not meaningfully differ between women with and without PCOS. Group-level differences in resting energy
⁴⁴ expenditure are small, insignificant, or not physiologically relevant.

⁴⁵ Keywords: Polycystic ovary syndrome, Energy Metabolism, Resting Metabolic Rate, Basal Metabolism

46 Introduction

47 Polycystic ovary syndrome (PCOS) affects approximately 10% of women of reproductive age worldwide,
48 making it the most common endocrine disorder affecting this population¹. Due to several factors including
49 hyperandrogenism and alterations in insulin resistance, PCOS is believed to contribute to an increased risk of
50 diabetes, metabolic syndrome and cardiovascular disease^{2–6}, along with being a leading cause of anovulatory
51 infertility in women⁷. Furthermore, epidemiological data has consistently demonstrated that women with
52 PCOS are significantly more likely to suffer from overweight or obesity, compared to the general female
53 population, with estimates ranging from 38% to 88% of PCOS patients falling into overweight or obese body
54 mass index (BMI) categories^{8,9}.

55 The elevated incidence of overweight and obesity in PCOS is likely multifactorial with proposed mechanisms
56 including blunted postprandial appetite hormone responses leading to reduced satiety and increased food
57 cravings^{10–12} and a reduced resting energy expenditure (REE)¹³. Indeed, the study from Georgopoulos
58 et al.¹³ examined REE in women with and without PCOS using indirect calorimetry and reported that
59 resting energy expenditure was approximately ~400 kcal/day lower in women with PCOS. Notably, they
60 also reported that insulin resistance further reduced REE among women with PCOS, with insulin-resistant
61 women exhibiting an additional reduction nearly 500 kcal per day compared to women with PCOS who
62 were not insulin resistant¹³. Other studies similarly report lower REE in women with PCOS using indirect
63 methods (such as prediction from bioelectrical impedance analysis or accelerometer physical activity data),
64 also suggesting that factors including insulin resistance and BMI category influence REE in women with
65 PCOS^{14,15}. However, despite the widespread acceptance that women living with PCOS exhibit reduced
66 REE based on studies such as these, other research has reported little to no difference in REE between
67 women with and without PCOS^{16–18}.

68 The consequences of widespread acceptance that REE is substantially lower in women living with PCOS
69 should not be underestimated, particularly in light of the aforementioned incidence of overweight and obesity
70 in this population. Women with PCOS typically engage in more frequent weight-loss attempts than women
71 without PCOS¹⁹. From a physiological perspective, if women with PCOS do exhibit a lower REE, this

72 could imply a meaningful metabolic disadvantage that may influence dietary and nutritional guidance for
73 weight management; for example, recommending a slightly more severe energy restriction to overcome the
74 belief that they have a lower REE²⁰. Recommendations such as this could influence the well documented
75 prevalence of eating disorders in women with PCOS^{21,22}. Contrastingly, belief in a “slower metabolism”
76 could instead serve as a deterrent to energy restriction based weight-loss approaches for some women in line
77 with typical general population recommendations that are also recognised as efficacious for improving PCOS
78 symptoms^{23,24} and are routinely recommended^{25,26}. It has been well documented that women with PCOS
79 already experience higher rates of anxiety, depression, and lower quality of life (QOL) as a result of negative
80 body image and weight-related concerns^{27–30}. Clarifying the relationship between REE and PCOS may
81 therefore help guide more accurate clinical recommendations and empower both practitioners and women
82 with PCOS.

83 A recent scoping review has highlighted the inconsistency in research examining energy expenditure in
84 women with PCOS³¹. However, to our knowledge there has been no attempts to systematically review, and
85 quantitatively synthesise via meta-analysis, studies of REE in women with, and without, PCOS. Therefore,
86 to estimate and describe the magnitude of difference in REE between women with and without PCOS, we
87 completed a systematic review and meta-analysis of studies reporting REE in these populations.

88 Materials and Methods

89 This systematic review and meta-analysis was pre-registered on PROSPERO ([CRD42024601434](#)) initially on
90 the 3rd of December 2024 and performed in accordance with the Preferred Reporting Items for Systematic
91 Reviews and Meta-Analyses (PRISMA) statement guidelines³². The PRISMA flow diagram reported below
92 (Figure 1) was produced using the PRISMA2020 R package and Shiny app³³. The primary aim of this review
93 was to examine the descriptive question “Does resting energy expenditure (REE) differ between women with
94 and without polycystic ovary syndrome (PCOS)?”. We summarise and describe the studies in addition to
95 quantitatively synthesising their results via meta-analysis.

⁹⁶ **Search Strategy**

⁹⁷ PubMed, Web of Science, and MEDLINE databases were searched using the following Boolean search string:
⁹⁸ ((“Basal Metabolic Rate”[MeSH] OR “Energy Metabolism”[MeSH] OR “Resting Metabolic Rate” OR RMR
⁹⁹ OR “Resting Energy Expenditure” OR REE OR “Basal Metabolic Rate” OR BMR OR “resting energy” OR
¹⁰⁰ “basal energy expenditure”) AND (“Polycystic Ovary Syndrome”[MeSH] OR “Polycystic Ovary Syndrome”
¹⁰¹ OR PCOS OR “Polycystic Ovarian Disease” OR “Stein-Leventhal Syndrome”)). Searches were limited to
¹⁰² publications up until May 2025 when the search was completed, limited to English language articles, and
¹⁰³ Rayyan was used to manage the search and screening process. Two reviewers (RK and GK) independently
¹⁰⁴ screened all titles and abstracts against the predefined inclusion and exclusion criteria. Articles deemed
¹⁰⁵ potentially eligible by either reviewer were retrieved in full text. Full texts were then independently assessed
¹⁰⁶ by RK and LP to determine final eligibility. Any disagreements at either stage were resolved through
¹⁰⁷ discussion, and when consensus could not be reached, a third reviewer acted as an adjudicator.

¹⁰⁸ **Eligibility Criteria**

¹⁰⁹ Studies were included in the systematic review if 1) participants were confirmed as women with PCOS
¹¹⁰ between the ages of 18 to 65 years of age with or without insulin resistance; 2) otherwise healthy (e.g.,
¹¹¹ non-diabetic, no cardiovascular disease); 3) had a measure of REE measured via multiple methods including
¹¹² direct/indirect calorimetry, doubly labelled water; and 4) trials were not retracted at the time of this analysis.

¹¹³ Studies were excluded if they 1) used invalid or non-standard methods for measuring REE (e.g., predicted
¹¹⁴ REE from body composition or accelerometer data); 2) non-peer-reviewed journal articles (including grey
¹¹⁵ literature sources such as conference abstracts, theses and dissertations); and 3) were secondary analyses
¹¹⁶ with the same primary outcome data as another included study.

¹¹⁷ The condition being studied was PCOS and we included observational cross-sectional design studies, in
¹¹⁸ addition to intervention studies where REE was reported for the population (and if present, the comparator
¹¹⁹ i.e., women without PCOS) condition of interest. For clarity, studies of any design were included if they
¹²⁰ reported the REE using the methods indicated for a sample of adult women with PCOS and who were
¹²¹ otherwise healthy. This included both studies with and without samples of healthy control women without

122 PCOS. As detailed in the statistical analysis section below, a Bayesian model with informative priors based
123 on normative data for REE in healthy women without PCOS was included to provide control information
124 indirectly where this was missing. The use of such priors is an efficient tool for incorporating historical
125 information on a particular population in a conservative manner³⁴.

126 Following the PICO framework our eligibility criteria can be defined as follows:

127 • Population

128 – Inclusion criteria:

129 * Women

130 * 18-65 y

131 * With or without insulin resistance (IR)

132 • Intervention(s) or exposure(s)

133 – Otherwise healthy women with PCOS

134 • Comparator(s) or control(s)

135 – Otherwise healthy control women without PCOS

136 • Outcome

137 – Inclusion criteria

138 * Resting energy expenditure (REE) measured via multiple methods including direct/indirect
139 calorimetry, doubly labelled water.

140 – Exclusion criteria:

141 * Studies using invalid or non-standard methods for measuring REE (e.g., predicted REE from
142 body composition or accelerometer data)

143 **Data extraction (selection and coding)**

144 Data was extracted by one investigator and checked in triplicate. Bibliometric data including authors,
145 journal, and article titles were extracted. Descriptive statistics for age, body mass, fat mass, fat free mass,
146 height, BMI, race, physical activity levels, country of investigation, information regarding glucose/insulin
147 regulation and insulin resistance status (where available), diagnostic criteria for PCOS, and measurement

¹⁴⁸ method and device were extracted for each arm within each study in addition to sample size. Descriptive
¹⁴⁹ characteristics were then tabulated across studies for reporting.

¹⁵⁰ For each arm, and observation time point if multiple observations reported (e.g., before and after an in-
¹⁵¹ tervention), depending on what was reported by the authors we extracted the means, medians, standard
¹⁵² deviations, standard errors, lower and upper range values, and interquartile range for the unadjusted and/or
¹⁵³ body mass adjusted and/or fat free mass adjusted REE values. Where REE values adjusted for body mass
¹⁵⁴ and/or fat free mass were reported we used the reported body mass and/or fat free mass mean values for
¹⁵⁵ that arm to convert them to unadjusted REE values (i.e., multiplied them by body mass and/or fat free
¹⁵⁶ mass mean values). Where means and/or standard deviations were missing the latter were either calculated
¹⁵⁷ from standard errors and sample size, or all both were estimated from lower and upper range, interquartile
¹⁵⁸ range, median, and sample size depending on the available information using the methods of Wan et al.³⁵.
¹⁵⁹ Further, where missing, height/body mass/BMI were estimated based on the reported means. The units
¹⁶⁰ of measurement for which REE was extracted and all REE values were converted to kcal/day. In one case³⁶
¹⁶¹ REE was reported relative to body mass and the unadjusted values were no longer available (confirmed
¹⁶² by the authors). As such, in this case we used the mean body mass to convert back to estimated REE
¹⁶³ unadjusted.

¹⁶⁴ **Studies with possible reporting errors**

¹⁶⁵ During data extraction it was noted that several studies from the same lab/research group^{13,37-39} contained
¹⁶⁶ a number of discrepancies that seemed to be possible reporting errors. This included, based on taking the
¹⁶⁷ authors results as written, standard errors that implied impossible or at least incredibly unlikely standard
¹⁶⁸ deviations, and discrepancies in sample size reporting throughout for most variables without explanation or
¹⁶⁹ where this was explained the sample sizes were discrepant with the text. Further, data was not reported for the
¹⁷⁰ healthy control women without PCOS in three of the studies³⁷⁻³⁹, and REE was reported as an “adjusted”
¹⁷¹ value whereby $\text{REE}_{\text{adjusted}} = \text{REE}_{\text{group mean}} + (\text{REE}_{\text{adjusted}} - \text{REE}_{\text{predicted}})$ and the $\text{REE}_{\text{predicted}}$ was
¹⁷² obtained by substituting the individual lean body mass, fat mass, gender, and age in the linear regression
¹⁷³ equation generated by the data of all patients. In correspondence with the senior author we were unable to

174 clarify the reporting discrepancies as the person responsible for the data/results was no longer contactable.
175 The original data were also no longer available and so we could not calculate the unadjusted REE.
176 Given these issues we decided to extract the results from these studies as reported and to conduct analyses
177 both with and without their inclusion. Though not pre-registered, due to a lack of confidence in the reported
178 results, we decided to include the analysis omitting these studies as our main models in the results reported
179 below. The results of the analysis including them are reported in the sensitivity analysis section.

180 Statistical Analysis

181 Statistical analysis of the data extracted was performed in R, (v 4.3.3; R Core Team, <https://www.r-project.org/>) and RStudio (v 2023.06.1; Posit, <https://posit.co/>). All code utilised for data preparation,
182 transformations, analyses, plotting, and reporting are available in the corresponding GitHub repository
183 https://github.com/jamessteeleii/pcos_ree_meta. We cite all software and packages used in the analysis
184 pipeline using the `grateful` package⁴⁰ which can be seen here: https://github.com/jamessteeleii/pcos_ree_meta/blob/main/grateful-report.pdf. The statistical analysis plan was linked in our pre-registration
185 (PROSPERO: CRD42024601434) and available at the accompanying GitHub repository. Any deviations
186 from the pre-registration are noted below.

187 Given our research question our analysis was aimed at parameter estimation⁴¹ within a Bayesian meta-
188 analytic framework⁴². For all analyses model parameter estimates and their precision, along with conclusions
189 based upon them, are interpreted continuously and probabilistically, considering data quality, plausibility
190 of effect, and previous literature, all within the context of each model. The `renv` package⁴³ was used for
191 package version reproducibility and a function based analysis pipeline using the `targets` package⁴⁴ was
192 employed (the analysis pipeline can be viewed by downloading the R Project and running the function
193 `targets::tar_visnetwork()`). Effect sizes and their variances were all calculated using the `metafor` pack-
194 ages `escalc()` function⁴⁵. The main package `brms`⁴⁶ was used in fitting all the Bayesian meta-analysis
195 models. Prior and posterior draws were taken using `marginaleffects`⁴⁷ and `tidybayes`⁴⁸ packages. All
196 visualisations are created using `ggplot2`⁴⁹, `tidybayes`, and the `patchwork`⁵⁰ packages.

¹⁹⁹ **Main Pre-registered Models**

²⁰⁰ We adopted an arm-based multiple condition comparison (i.e., network) type model given that the studies
²⁰¹ included had arms of women with PCOS both with, and without, a non-PCOS control arm⁵¹, and also in
²⁰² some cases multiple observations of REE in the different arms included in the study (for example, where an
²⁰³ intervention was conducted and pre- and post-intervention REE was reported). In typical contrast-based
²⁰⁴ meta-analyses data is limited to the effect sizes for paired contrasts between arms and thus studies that
²⁰⁵ include both arms (i.e., relative effects between non-PCOS control vs PCOS arms); however, in arm-based
²⁰⁶ analyses the data are the absolute effects within each arm and information is borrowed across studies to
²⁰⁷ enable both within condition absolute, and between condition relative contrasts to be estimated. We made
²⁰⁸ use of historical information regarding REE in healthy control women without PCOS by setting informative
²⁰⁹ priors based on meta-analysis of large scale studies reporting normative data for REE in this population.
²¹⁰ This was included to provide indirect control information where it was missing from particular studies. The
²¹¹ use of historical priors like this is an efficient tool to incorporate historical information about a particular
²¹² population in a conservative manner in meta-analyses³⁴. From this model we focus on reporting the global
²¹³ grand mean estimate for the fixed between condition relative contrast for non-PCOS control vs PCOS arms
²¹⁴ as our primary estimand of interest (i.e., β_1 in both mean and standard deviation models). We examined
²¹⁵ both raw mean REE (i.e., the absolute mean REE in kcals per day for each arm) in addition to the between
²¹⁶ person standard deviation in REE (i.e., the absolute standard deviation in REE in kcals per day for each
²¹⁷ arm). Both models were multilevel in that they included nested random intercepts for both study and arm
²¹⁸ within study. In addition, and in deviation from our pre-registration, we also included lab as a random
²¹⁹ intercept as in some cases we had multiple studies from the same lab or research group. Lastly, the inclusion
²²⁰ of a random intercept for each effect size was accidentally omitted from our pre-registration, and so this is
²²¹ also included in the model.

²²² **Mean REE Model**

223 The main model for mean REE with cond representing the condition (either control or PCOS) was as follows:

$$\hat{\theta}_{ijkl} \sim \mathcal{N}(\mu_{ijkl}, \sigma_{ijkl}^2)$$

$$\mu_{ijkl} = \beta_0 + \beta_1 \text{cond}[ijkl] + \alpha_{0,\text{lab}[i]} + \alpha_{1,\text{lab}[i]} \text{cond} + \alpha_{0,\text{study}[j]} + \alpha_{1,\text{study}[j]} \text{cond} + \alpha_{0,\text{arm}[k]} + \alpha_{0,\text{effect}[l]}$$

$$\begin{pmatrix} \alpha_{0,\text{lab}[i]} \\ \alpha_{1,\text{lab}[i]} \end{pmatrix} \sim \mathcal{N}\left(\begin{pmatrix} 0 \\ 0 \end{pmatrix}, \Sigma_{\text{lab}}\right), \quad \Sigma_{\text{lab}} = \begin{pmatrix} \sigma_{0,\text{lab}}^2 & \rho_{\text{lab}} \sigma_{0,\text{lab}} \sigma_{1,\text{lab}} \\ \rho_{\text{lab}} \sigma_{0,\text{lab}} \sigma_{1,\text{lab}} & \sigma_{1,\text{lab}}^2 \end{pmatrix}, \text{ for lab } i = 1, \dots, I$$

$$\begin{pmatrix} \alpha_{0,\text{study}[j]} \\ \alpha_{1,\text{study}[j]} \end{pmatrix} \sim \mathcal{N}\left(\begin{pmatrix} 0 \\ 0 \end{pmatrix}, \Sigma_{\text{study}}\right), \quad \Sigma_{\text{study}} = \begin{pmatrix} \sigma_{0,\text{study}}^2 & \rho_{\text{study}} \sigma_{0,\text{study}} \sigma_{1,\text{study}} \\ \rho_{\text{study}} \sigma_{0,\text{study}} \sigma_{1,\text{study}} & \sigma_{1,\text{study}}^2 \end{pmatrix}, \text{ for study } j = 1, \dots, J$$

$$\alpha_{0,\text{arm}[k]} \sim \mathcal{N}(0, \sigma_{0,\text{arm}}^2), \text{ for arm } k = 1, \dots, K$$

$$\alpha_{0,\text{effect}[l]} \sim \mathcal{N}(0, \sigma_{0,\text{effect}}^2), \text{ for effect } l = 1, \dots, L$$

224 where $\hat{\theta}_{ijkl}$ is the l th mean REE estimate from the k th arm, for the j th study, conducted by the i th lab
 225 and σ_{ijkl}^2 is the corresponding sampling error for that estimate. The random intercepts for the i th lab, j th
 226 study, k th arm, and l th mean REE estimate are $\alpha_{0,\text{lab}[i]}$, $\alpha_{0,\text{study}[j]}$, $\alpha_{0,\text{arm}[k]}$, and $\alpha_{0,\text{effect}[l]}$ respectively
 227 each with standard deviation of $\sigma_{0,\text{lab}[i]}^2$, $\sigma_{0,\text{study}[j]}^2$, $\sigma_{0,\text{arm}[k]}^2$, and $\sigma_{0,\text{effect}[l]}^2$. The parameter β_0 represents
 228 the fixed effect estimate of REE for control conditions and β_1 the fixed effect estimate for the offset from
 229 this for the PCOS conditions (i.e., the difference between conditions). The estimated offset was allowed to
 230 vary across both labs and studies each reflected by $\alpha_{1,\text{lab}[i]}$ and $\alpha_{1,\text{study}[j]}$ respectively, and these effects were
 231 also modelled as correlated with the corresponding random intercepts with covariance Σ_{lab} and Σ_{study} , and
 232 corr_{lab} and corr_{study} correlation matrices.

233 The priors for this model were as follows:

$$\beta_0 \sim \text{Student-}t(3, 1441.81, 84.56)$$

$$\beta_1 \sim \text{Student-}t(3, 0, 200)$$

$$\sigma_{0,\text{study}}^2 \sim \text{Half-student-}t(3, 149.89, 82.91)$$

$$\sigma_{0,\text{lab}}^2, \sigma_{1,\text{lab}}^2, \sigma_{1,\text{study}}^2, \sigma_{0,\text{arm}}^2, \sigma_{0,\text{effect}}^2 \sim \text{Half-student-}t(3, 0, 112.4)$$

$$\text{corr}_{\text{lab}} \sim \text{LKJ}(1)$$

$$\text{corr}_{\text{study}} \sim \text{LKJ}(1)$$

234 where the prior for β_0 , which corresponded to the model intercept and mean REE in the control condition,
235 was set based on meta-analysis of the mean REEs for women from two large studies of healthy people^{52,53}
236 though set with a conservative degrees of freedom for the Student-*t* distribution. The random intercept $\sigma_{0,\text{study}}^2$
237 was set similarly to this. The prior for the fixed effect β_1 , reflecting the difference between control and PCOS
238 conditions was set based on a wide range of possible values considering the minimum and maximum
239 values of the ranges reported in the two studies noted (i.e., 2492 - 908 = 1584). We then set a prior that
240 permits values approximately across this range of values with the majority of it's mass centred around zero.
241 The remaining random effects were set based on the default weakly regularising priors for `brms` and scaled
242 to the expected response values using a half-student-*t* distribution with 3 degrees of freedom and $\mu = 0$, and
243 both correlation matrices corr_{lab} and $\text{corr}_{\text{study}}$ were set with an LKJcorr(1) distribution.

244 Standard Deviation of REE Model

245 The main model for the standard deviation of REE with cond representing the condition (either control or
246 PCOS) was as follows:

$$\log(\hat{\theta}_{ijkl}) \sim \mathcal{N}(\mu_{ijkl}, \sigma_{ijkl}^2)$$

$$\mu_{ijkl} = \beta_0 + \beta_1 \text{cond}_{[ijkl]} + \beta_2 \tilde{m}_{[ijkl]} + \alpha_{0,\text{lab}[i]} + \alpha_{1,\text{lab}[i]} \text{cond} + \alpha_{0,\text{study}[j]} + \alpha_{1,\text{study}[j]} \text{cond} + \alpha_{0,\text{arm}[k]} + \alpha_{0,\text{effect}[l]}$$

$$m = \log(y_{i,\text{mean},[\text{ijkl}]})$$

$$\tilde{m} \sim \mathcal{N}(m, \sigma_{\log(y_{i,\text{mean},[\text{ijkl}]})}^2)$$

$$\begin{pmatrix} \alpha_{0,\text{lab}[i]} \\ \alpha_{1,\text{lab}[i]} \end{pmatrix} \sim \mathcal{N}\left(\begin{pmatrix} 0 \\ 0 \end{pmatrix}, \Sigma_{\text{lab}}\right), \quad \Sigma_{\text{lab}} = \begin{pmatrix} \sigma_{0,\text{lab}}^2 & \rho_{\text{lab}} \sigma_{0,\text{lab}} \sigma_{1,\text{lab}} \\ \rho_{\text{lab}} \sigma_{0,\text{lab}} \sigma_{1,\text{lab}} & \sigma_{1,\text{lab}}^2 \end{pmatrix}, \text{ for lab } i = 1, \dots, I$$

$$\begin{pmatrix} \alpha_{0,\text{study}[j]} \\ \alpha_{1,\text{study}[j]} \end{pmatrix} \sim \mathcal{N}\left(\begin{pmatrix} 0 \\ 0 \end{pmatrix}, \Sigma_{\text{study}}\right), \quad \Sigma_{\text{study}} = \begin{pmatrix} \sigma_{0,\text{study}}^2 & \rho_{\text{study}} \sigma_{0,\text{study}} \sigma_{1,\text{study}} \\ \rho_{\text{study}} \sigma_{0,\text{study}} \sigma_{1,\text{study}} & \sigma_{1,\text{study}}^2 \end{pmatrix}, \text{ for study } j = 1, \dots, J$$

$$\alpha_{0,\text{arm}[k]} \sim \mathcal{N}(0, \sigma_{0,\text{arm}}^2), \text{ for arm } k = 1, \dots, K$$

$$\alpha_{0,\text{effect}[l]} \sim \mathcal{N}(0, \sigma_{0,\text{effect}}^2), \text{ for effect } l = 1, \dots, L$$

247 where $\log(\hat{\theta}_{ijkl})$ is the l th natural logarithm of the standard deviation of REE estimate from the k th arm,
 248 for the j th study, conducted by the i th lab and σ_{ijkl}^2 is the corresponding sampling error for that estimate.
 249 The random intercepts for the i th lab, j th study, k th arm, and l th standard deviation of REE estimate are
 250 $\alpha_{0,\text{lab}[i]}$, $\alpha_{0,\text{study}[j]}$, $\alpha_{0,\text{arm}[k]}$, and $\alpha_{0,\text{effect}[l]}$ respectively each with standard deviation of $\sigma_{0,\text{lab}[i]}^2$, $\sigma_{0,\text{study}[j]}^2$,
 251 $\sigma_{0,\text{arm}[k]}^2$, and $\sigma_{0,\text{effect}[l]}^2$. The parameter β_0 represents the fixed effect estimate of standard deviation of REE
 252 for control conditions and β_1 the fixed effect estimate for the offset from this for the PCOS conditions (i.e.,
 253 the difference between conditions). The estimated offset was allowed to vary across both labs and studies
 254 each reflected by $\alpha_{1,\text{lab}[i]}$ and $\alpha_{1,\text{study}[j]}$ respectively, and these effects were also modelled as correlated with
 255 the corresponding random intercepts with covariance Σ_{lab} and Σ_{study} , and corr_{lab} and $\text{corr}_{\text{study}}$ correlation
 256 matrices. Finally, β_2 represents the fixed effect of the natural logarithm of the corresponding mean REE
 257 estimate \tilde{m} which is modelled as estimated with measurement error i.e., m represents the point estimate for
 258 the l th natural logarithm of the mean REE estimate from the k th arm, for the j th study, conducted by the
 259 i th lab and $\sigma_{\log(y_{i,\text{mean},[\text{ijkl}]})}^2$ is the corresponding sampling error for that estimate.

260 The priors for this model were as follows:

$$\beta_0 \sim \text{Student-}t(3, 5.54, 0.80)$$

$$\beta_1 \sim \text{Student-}t(3, 0, 5.3)$$

$$\beta_2 \sim \text{Student-}t(3, 0, 2.5)$$

$$\text{mean}(\tilde{m}) \sim \text{Half-student-}t(3, 7.28, 0.62)$$

$$\text{sd}(\tilde{m}) \sim \text{Half-student-}t(3, 0, 5)$$

$$\sigma_{0,\text{study}}^2 \sim \text{Half-student-}t(3, 1.08, 1.06)$$

$$\sigma_{0,\text{lab}}^2, \sigma_{1,\text{lab}}^2, \sigma_{1,\text{study}}^2, \sigma_{0,\text{arm}}^2, \sigma_{0,\text{effect}}^2 \sim \text{Half-student-}t(3, 0, 2.5)$$

$$\text{corr}_{\text{lab}} \sim \text{LKJ}(1)$$

$$\text{corr}_{\text{study}} \sim \text{LKJ}(1)$$

261 where the prior for β_0 , which corresponded to the model intercept and standard deviation of REE in the
262 control condition, was again set based on meta-analysis of the standard deviation of REEs for women from
263 two large studies of healthy people^{52,53} though set with a conservative degrees of freedom for the Student-*t*
264 distribution. The random intercept $\sigma_{0,\text{study}}^2$ was set similarly to this. The prior for the fixed effect β_1 , reflecting
265 the difference between control and PCOS conditions was set based on a wide range of possible values. Given
266 that in many cases of variables in the field there is an approximate relationship of ~1 for the natural logarithm
267 of the standard deviation conditioned upon the natural logarithm of the mean⁵⁴ we set this prior to reflect
268 the range of differences on the on the log scale (i.e., $\log(1584)$). We then set a prior that permits values
269 approximately across this range of values with the majority of it's mass centred around zero. The prior
270 for the fixed effect β_2 , reflecting the relationship between the natural logarithm of the mean REE with the
271 natural logarithm of the standard deviation of REE, was set it to be weakly informative centred on zero
272 with a wide scale to indicate uncertainty in this outcome specifically despite the typical relationship close
273 to ~1. Priors for the measurement error of the natural logarithm of the mean REE estimate \tilde{m} were again
274 based upon meta-analysis of the aforementioned studies, though measurement error has to be positive, as
275 does the corresponding standard deviation of this error, so we set these to conservative wide half-student-*t*

276 distributions. The remaining random effects were set based on the default weakly regularising priors for
277 `brms` and scaled to the expected response values using a half-student-*t* distribution with 3 degrees of freedom
278 and $\mu = 0$, and both correlation matrices corr_{lab} and corr_{study} were set with an $\text{LKJcorr}(1)$ distribution.

279 **Post-processing of models**

280 For both models we examined trace plots along with \hat{R} values to examine whether chains have converged, and
281 posterior predictive checks for each model to understand the model implied distributions. From each model
282 we took draws from the posterior distributions for the conditional absolute estimates for each condition (i.e.,
283 controls and PCOS) by study incorporating random effects, the global grand mean absolute estimates for
284 each condition ignoring random effects, and the global grand mean between condition relative contrast for
285 controls vs PCOS conditions ignoring random effects. The between condition relative contrast for controls
286 vs PCOS conditions corresponded to β_1 in each model and was our primary estimand of interest; for the
287 mean REE model this corresponded to the absolute difference in mean REE, and for the standard deviation
288 of REE model this corresponded to the natural logarithm of the ratio of standard deviations of REE which
289 was exponentiated (note, all log standard deviation of REE model estimates were exponentiated back to the
290 original scale to aid interpretability). We present the full probability density functions for posterior visually,
291 and also to calculate mean and 95% quantile intervals (QI: i.e., ‘credible’ or ‘compatibility’ intervals) for
292 each estimate providing the most probable value of the parameter in addition to the range from 2.5% to
293 97.5% percentiles given our priors and data.

294 **Sensitivity analyses**

295 **Pairwise contrast based models**

296 By way of pre-registered sensitivity analysis we also conducted pairwise contrast based models where we
297 limited the included effects to those extracted from studies including only a directly comparable control and
298 PCOS arm at baseline. These models were both as follows:

$$\hat{\theta}_{ij} \sim \mathcal{N}(\mu_{ij}, \sigma_{ij}^2)$$

$$\mu_{ij} = \beta_0 + \alpha_{0,\text{lab}[i]} + \alpha_{0,\text{study}[j]}$$

$$\alpha_{0,\text{lab}[i]} \sim \mathcal{N}(0, \sigma_{0,\text{lab}}^2), \text{ for lab } i = 1, \dots, I$$

$$\alpha_{0,\text{study}[j]} \sim \mathcal{N}(0, \sigma_{0,\text{study}}^2), \text{ for study } j = 1, \dots, J$$

299 where $\hat{\theta}_{ijkl}$ is the pairwise effect size, either the mean difference in REE or the log coefficient of variation ratio
 300 (calculated as PCOS vs control), for the j th study, conducted by the i th lab and σ_{ijkl}^2 is the corresponding
 301 sampling error for that effect size estimate. The random intercepts for the i th lab, j th study are $\alpha_{0,\text{lab}[i]}$ and
 302 $\alpha_{0,\text{study}[j]}$ respectively each with standard deviation of $\sigma_{0,\text{lab}[i]}^2$, $\sigma_{0,\text{study}[j]}^2$. The parameter β_0 represents the
 303 fixed effect estimate of the pairwise effect size i.e., the pooled estimate of the contrast between conditions.
 304 The priors for β_0 in these models were set as default weakly regularising which is set on an intercept that
 305 results when internally centering all population-level predictors around zero to improve sampling efficiency
 306 and scaled to the expected response values using a student- t distribution; for the mean difference in REE
 307 this was student- $t(3, 5.5, 50.2)$ and for the log coefficient of variance ratio this was student- $t(3, -0.1, 2.5)$.
 308 The random effects were set similarly scaled to the expected response values but using a half-student- t
 309 distribution centred on zero. From these models we calculated the mean and 95% quantile intervals (i.e.,
 310 ‘credible’ or ‘compatibility’ intervals) for the β_0 (comparable to the β_1 from the corresponding mean and
 311 standard deviation of REE arm-based models) for each effect size providing the most probable value of the
 312 parameter in addition to the range from 2.5% to 97.5% percentiles given our priors and data.

313 Additional sensitivity analyses

314 As noted in the section above, “*Studies with possible reporting errors*”, we opted to conduct analysis with
 315 and without the inclusion of four studies with possible reporting errors we could not resolve^{13,37–39}. Thus
 316 the main models described above were run with and without these studies, the main results are presented
 317 without them and the sensitivity results with them are reported separately below.
 318 As a further sensitivity analysis, given the inclusion of some studies with interventions in women with

319 PCOS reporting REE at multiple timepoints such as mid- and post-intervention^{36,55,56} but lacking of control
320 women without PCOS, we opted to also conduct sensitivity analysis excluding these and only examining
321 their baseline results in addition to the cross-sectional studies. As such, the main models described above
322 were run without the follow-up (i.e., mid- or post-intervention timepoints) from these studies and only using
323 the baseline results in addition to other cross-sectional studies.

324 Lastly, given the role of body mass and, in particular, highly metabolically active tissue on REE we included
325 models adjusted for these group level characteristics where studies reported them. These models were
326 essentially extensions of the main models noted above for BMI and fat-free mass where each was modelled
327 with its corresponding sampling error similarly to how the log mean REE was modelled in the models for
328 standard deviation of REE i.e., the mean BMI or fat-free mass estimates were modelled as estimated with
329 measurement error. When extracting posterior distributions for the contrasts between conditions in these
330 models both BMI and fat-free mass were adjusted to the median values seen in the control conditions i.e.,
331 BMI = 26.03 kg/m² and fat-free mass = 48.8 kg.

332 Results

333 Systematic Review

334 Note, the numbers in this section exclude the four studies previously noted with possible reporting issues
335 that were unresolved. These studies are however summarised in fully in the descriptive characteristics table
336 in the online supplementary materials (see [Descriptives Table](#) where some of the possible reporting errors
337 are seen in the standard deviations reported or calculated e.g., the standard deviation calculated for body
338 mass of women with PCOS in Saltamavros et al.³⁸. The PRISMA flow diagram (Figure 1) also includes all
339 studies identified including the four with possible errors.

340 Our systematic review identified 13 studies from 12 lab/research groups including 24 arms (Control arms =
341 9, PCOS arms = 15) and a total of 918 participants (Controls: minimum n = 9, median n = 29, maximum
342 n = 54; PCOS: minimum n = 5, median n = 28, maximum n = 266). Descriptive characteristics of the arms
343 and participants in these studies are reported fully in the online supplementary materials (see [Descriptives](#)

344 [Table](#)).

345 Studies were carried out in multiple countries: Brazil ($k = 3$), and USA ($k = 3$ studies), Australia, Cameroon,
346 Canada, Italy, Sweden, Turkey, UK (all $k = 1$ study). The four studies with noted reporting errors were
347 carried out in Greece. A total of 11 studies used the Rotterdam criteria (or a modified version thereof) for
348 diagnosing PCOS^{16,17,36,55–62}. One study used the 1990 National Institutes of Health criteria⁶³, and two
349 studies^{18,57} diagnosed PCOS via the presence of oligomenorrhea or amenorrhea alongside additional criteria
350 including plasma androgen levels, hirsutism or polycystic ovaries on ultrasound scanning. All but one study⁶³,
351 which used doubly labelled water, measured REE using indirect calorimetry. The specific devices reported
352 by these studies are included in the online supplementary materials (see [Descriptives Table](#)). We originally
353 considered in our pre-registration that, given sufficient data, we would compare sub groups of women with
354 PCOS who did and did not have accompanying insulin resistance. However, based on the metabolic health
355 variables reported in studies with this information (see [Descriptives Table](#)) and, where available, considering
356 primary criteria of either homeostatic model assessment of insulin resistance (HOMA-IR) ≥ 2.5 or secondary
357 criteria including fasting insulin $> 12\mu\text{U}/\text{mL}$ or fasting glucose $\geq 100\text{mg}/\text{dL}$, all groups of women with
358 PCOS in the included studies would be considered to have insulin resistance. Mean age of women with
359 PCOS in these studies ranged from 23 to 33 which was similar to the control women without PCOS ranging
360 from 23 to 30. Across those studies where BMI was reported or it was possible to estimate the mean BMI of
361 women with PCOS ranged from 26.4 to 39.9 was typically greater than women without PCOS which ranged
362 20.5 to 27.9.

363 Mean REE Model Results

364 The main model for mean REE resulted in a posterior distribution for the contrast between control and
365 PCOS conditions with a mean point estimate of 30 kcal/day with a 95% quantile interval ranging from -47
366 kcal/day to 113 kcal/day suggesting there is a 95% probability that the true difference lies between these
367 values given our priors and the data from included studies. The corresponding conditional estimates for
368 the control condition and PCOS condition respectively were 1442 kcal/day [95%QI:1334 kcal/day to 1553
369 kcal/day] and 1472 kcal/day [95%QI:1359 kcal/day to 1587 kcal/day]. These results, including the full

³⁷⁰ visualisation of the posterior distribution and the conditional estimates by study, can be seen in Figure 2.

³⁷¹ **Standard Deviation of REE Model Results**

³⁷² The main model for the between participant standard deviation of REE resulted in a posterior distribution
³⁷³ for the contrast ratio between control and PCOS conditions with a mean point estimate of 0.98 with a 95%
³⁷⁴ quantile interval ranging from 0.71 to 1.33 suggesting there is a 95% probability that the true ratio of standard
³⁷⁵ deviations lies between these values given our priors and the data from included studies. The corresponding
³⁷⁶ conditional estimates for the standard deviations of the control condition and PCOS condition respectively
³⁷⁷ were 238 kcal/day [95%QI:178 kcal/day to 312 kcal/day] and 229 kcal/day [95%QI:169 kcal/day to 303
³⁷⁸ kcal/day]. These results, including the full visualisation of the posterior distribution and the conditional
³⁷⁹ estimates by study, can be seen in Figure 3.

³⁸⁰ **Sensitivity Analyses**

³⁸¹ **Pairwise Models**

³⁸² For mean REE the pairwise model resulted in qualitatively similar inferences suggesting little difference
³⁸³ between control and PCOS conditions with mean point estimate of 16 kcal/day with a 95% quantile interval
³⁸⁴ ranging from -36 kcal/day to 70 kcal/day. This was similar for the standard deviation of REE with the
³⁸⁵ pairwise model resulting in a contrast ratio between the control and PCOS conditions with a mean point
³⁸⁶ estimate of 0.9 and a 95% quantile interval ranging from 0.6 to 1.35.

³⁸⁷ **Models including studies with possible reporting issues**

³⁸⁸ For mean REE the model including the studies noted with possible reporting issues that were not
³⁸⁹ resolved^{13,37-39} still resulted in qualitatively similar inferences suggesting little difference between control
³⁹⁰ and PCOS conditions with mean point estimate of 15 kcal/day with a 95% quantile interval ranging from
³⁹¹ -69 kcal/day to 95 kcal/day. This was similar for the standard deviation of REE with the model including
³⁹² these studies resulting in a contrast ratio between the control and PCOS conditions with a mean point
³⁹³ estimate of 1.21 and a 95% quantile interval ranging from 0.68 to 2.13.

394 **Baseline REE Measurement Models**

395 For mean REE the model which included only baseline REE measurements from studies involving
396 interventions^{36,55,56}, in addition to other cross-sectional studies, also resulted in qualitatively similar
397 inferences suggesting little difference between control and PCOS conditions with mean point estimate of 32
398 kcal/day with a 95% quantile interval ranging from -46 kcal/day to 118 kcal/day. This was also the case for
399 the standard deviation of REE with this model resulting in a contrast ratio between the control and PCOS
400 conditions with a mean point estimate of 0.97 and a 95% quantile interval ranging from 0.71 to 1.36.

401 **BMI and Fat-Free Mass Adjusted Models**

402 For mean REE the model adjusted for BMI, also resulted in qualitatively similar inferences suggesting little
403 difference between control and PCOS conditions with mean point estimate of 20 kcal/day with a 95% quantile
404 interval ranging from -96 kcal/day to 140 kcal/day. This was also the case for the standard deviation of
405 REE with this model resulting in a contrast ratio between the control and PCOS conditions with a mean
406 point estimate of 0.91 and a 95% quantile interval ranging from 0.56 to 1.41. This was similar for fat-free
407 mass adjusted models too showing little difference in mean REE between control and PCOS conditions with
408 mean point estimate of -9 kcal/day with a 95% quantile interval ranging from -195 kcal/day to 168 kcal/day
409 and a contrast ratio between the control and PCOS conditions with a mean point estimate of 0.79 and a
410 95% quantile interval ranging from 0.31 to 1.94.

411 **Discussion**

412 This study sought to estimate and describe the magnitude of difference in REE between women with and
413 without PCOS. Most studies identified in the systematic review, and included in the meta-analysis, used
414 indirect calorimetry as the primary measure of REE and assessed women with PCOS who were insulin
415 resistant and categorised as being in overweight or obese BMI categories compared to healthy controls. Our
416 results indicate there is only a small magnitude of difference in REE (30 kcal/day [95%QI: -47 kcal/day to
417 113kcal/day]) between women with PCOS and those without. Further, there is little difference in between
418 person variation between the groups based on the ratio of standard deviations (0.98 [95%QI: 0.71 to 1.33])

419 suggesting that, despite individual differences in REE, PCOS is not systematically associated with lesser or
420 greater individual variability.

421 These findings challenge the widely held belief that PCOS is inherently associated with a slower metabolism²⁶,
422 predisposing women with PCOS to weight gain. This belief largely stems from a single but influential 2009
423 study from Georgopoulos et al. that reported a significantly reduced BMR in women with PCOS¹³, which
424 has been widely cited and reinforced in both academic and clinical contexts. However, as we have noted, this
425 study along with others³⁷⁻³⁹ have numerous reporting errors which led us to drop them from our present
426 analysis (though sensitivity analysis including them did not alter our overall conclusions). The mistaken belief
427 that REE is lower for those with PCOS may have mistakenly lead to recommendations centred on slightly
428 more severe calorie restriction to achieve weight-loss goals, compared with recommendations for the general
429 population, as primary management strategies for women with PCOS²⁰. Recognising that there may be
430 minimal differences in REE between women with PCOS and those without can inform both clinical and public
431 practices, potentially leading to a shift in focus away from a requirement for more severe caloric restriction
432 as a primary method of treatment towards more comprehensive, individualised, and psychologically safe
433 approaches to care^{25,26}.

434 The current study suggests that REE may not a barrier to weight regulation in PCOS given small group-
435 level differences between women with PCOS and those without (1472 kcal/day [95%QI:1359 kcal/day to 1587
436 kcal/day] versus 1442 kcal/day [95%QI:1334 kcal/day to 1553 kcal/day], respectively). If anything, REE
437 may be slightly higher in women with PCOS compared to healthy women without PCOS. BMI of women
438 with PCOS ranged from 26.4 to 39.9 was typically greater than women without PCOS which ranged 20.5 to
439 27.9 and this may explain the slightly greater REE in the former group. However, for those studies where
440 we could extract or estimate BMI, our additional exploratory models adjusted for this similarly showed
441 little difference in REE (20 kcal/day [95%QI: -96 kcal/day to 140kcal/day]) between women with PCOS
442 and those without. However, one of the studies included in our analysis⁶⁴ reported that, whilst there was
443 little difference in unadjusted REE, when REE was adjusted for fat-free mass it was lower in women with
444 PCOS suggesting the potential importance of fat-free tissue in energy regulation. Yet, for those studies
445 where fat-free mass was reported, our additional exploratory models adjusted for this similarly showed little

446 difference in REE (-9 kcal/day [95%QI: -195 kcal/day to 168kcal/day]) between women with PCOS and
447 those without. As such, even adjusted for both BMI and fat-free mass, there seems to be little difference in
448 REE between women with, and without, PCOS. Yet, a recent systematic review of mechanisms for metabolic
449 dysfunction has reported excess androgen drives metabolic issues within adipose tissue and muscle tissue
450 contributing to complications like obesity and insulin resistance⁶⁵. Taken together, these findings highlight
451 that factors beyond REE and typical correlates of this including BMI or fat-free mass, such as hormonal and
452 tissue-specific metabolic effects, may play a more significant role in weight regulation challenges in women
453 with PCOS.

454 As noted, women with PCOS are more likely to engage in weight-loss attempts¹⁹ and there could be con-
455 cerns that this could inadvertently further foster the already well documented disordered eating in this
456 population^{21,22,66,67}. The pathways linking PCOS and disordered eating are multifactorial. Biological mech-
457 anisms such as hyperandrogenism, hyperinsulinaemia, and altered ghrelin and leptin signalling can heighten
458 hunger, carbohydrate cravings, and appetite variability⁶⁸. Frequent hypoglycaemia and associated mood
459 changes have also been observed, which can trigger compensatory eating or binge episodes⁶⁸. These physi-
460 ological processes interact with psychological and social stressors, including infertility concerns, conflicting
461 nutrition advice, chronic dieting, and exposure to idealised body images on social media, which together
462 compound vulnerability to disordered eating⁶⁹. Moreover, eating disorders themselves can disrupt endocrine
463 function, potentially worsening PCOS symptoms and creating a self-reinforcing cycle^{67,70}.

464 Understanding the intertwined biological, psychological, and social influences suggests the importance of
465 considering whether restrictive dietary advice is appropriate given it may exacerbate feelings of failure,
466 hunger dysregulation, and shame⁷¹. These concerns, coupled with the lack of difference in REE between
467 women with and without PCOS might suggest that energy restriction based dietary interventions for weight-
468 loss may be unnecessary. But, there is also evidence supporting the effect of energy restricted dietary
469 interventions for improving PCOS symptoms^{23,24} and they are recommended in international guidelines
470 [moranEvidenceSummariesRecommendations2020;²⁶]. Encouragingly though, these guidelines also recognise
471 weight stigma as a determinant of health and call for its reduction across clinical and public health settings.
472 Evidently the greater prevalence of women with PCOS falling into overweight and obese BMI categories

473 compared to women without PCOS^{8,9} is unlikely to be due to differences in REE and so, despite the potential
474 effectiveness of energy restriction dietary interventions, there is potential value in moving towards more
475 weight-neutral, individualised, and empowering care following holistic guidelines recommending multiple
476 approaches to management [moranEvidenceSummariesRecommendations2020;^{26,20}].

477 **Strengths and Limitations**

478 The current study has multiple strengths stemming from its preregistered, comprehensive methodology and
479 Bayesian statistical approach. This statistical framework allowed us to incorporate studies with and without
480 control groups to better estimate REE in women with and without PCOS and to perform multiple sensitivity
481 analyses that confirmed the stability of our findings. However, a limitation here is that variability in methods
482 across studies, such as differences in PCOS diagnostic criteria and REE testing protocols, may have influenced
483 results and the relatively small number of studies overall limits the extent to which we can explore these
484 potential moderators. Furthermore, some studies controlled for body weight or body composition when
485 reporting REE values, while others did not. We accounted for this by estimating or converting reported
486 data to obtain unadjusted REE values across all groups, thereby reducing this variability and further as
487 noted above provided estimates adjusted for BMI and fat-free mass both of which had little influence on our
488 conclusions. Another limitation is that, due to fewer total control groups than PCOS groups, informed priors
489 were required in several statistical models. However, in the context of Bayesian meta-analysis this can also
490 be considered a strength. Additionally, some studies reported data inconsistencies that could not be clarified
491 (e.g.,^{13,37–39}) and were dropped from our main analysis though our conclusions again did not qualitative
492 change when we conducted sensitivity analyses including these studies. Finally, most included studies were
493 cross-sectional or baseline assessments within intervention trials, which limits causal inference. Indeed, we
494 did not pre-register any kind of causal model (e.g., a directed acyclic graph) to inform our analysis approach
495 for causal inference and as such have been explicit about the estimates presented as being descriptive.

⁴⁹⁶ Conclusion

⁴⁹⁷ In conclusion, the findings from this meta-analysis indicate that REE does not meaningfully differ between
⁴⁹⁸ women with and without PCOS. Group-level differences in REE were small, insignificant, or not physiologi-
⁴⁹⁹ cally relevant. Additionally, variability in REE between individuals was also similar. These results suggest
⁵⁰⁰ that a lower baseline REE is not associated with the weight-related challenges often associated with PCOS.
⁵⁰¹ These findings challenge the popular narrative that women with PCOS have a lower REE and may help
⁵⁰² better inform dietary interventions and nutritional support for these individuals. Future research should
⁵⁰³ include more standardized REE measurement and reporting protocols, greater data transparency, consistent
⁵⁰⁴ control and reporting of body weight or body composition, the presentation of both absolute and relative
⁵⁰⁵ REE, and more precise characterization of PCOS phenotypes. Overall, these findings support the conclu-
⁵⁰⁶ sion that PCOS is not negatively associated with REE and may help practitioners and researchers focus on
⁵⁰⁷ individually targeted and holistic lifestyle interventions rather than negatively framed interventions based
⁵⁰⁸ on unsupported assumptions regarding REE.

⁵⁰⁹ Data Availability

⁵¹⁰ All code utilised for data preparation, transformations, analyses, plotting, and reporting are available in the
⁵¹¹ corresponding GitHub repository https://github.com/jamessteeleii/pcos_ree_meta.

⁵¹² Contributions

⁵¹³ Gregory Nuckols and Leigh Peele conceived the idea for the project. All authors contributed to the design
⁵¹⁴ of the project and methods. Richie Kirwan and Leigh Peele conducted the systematic search and screening.
⁵¹⁵ James Steele performed the data extraction, conducted the statistical analyses, and produced the data
⁵¹⁶ visualisations. All authors contributed to drafting the initial manuscript. All authors contributed to editing
⁵¹⁷ the manuscript. All authors read and approved the final manuscript.

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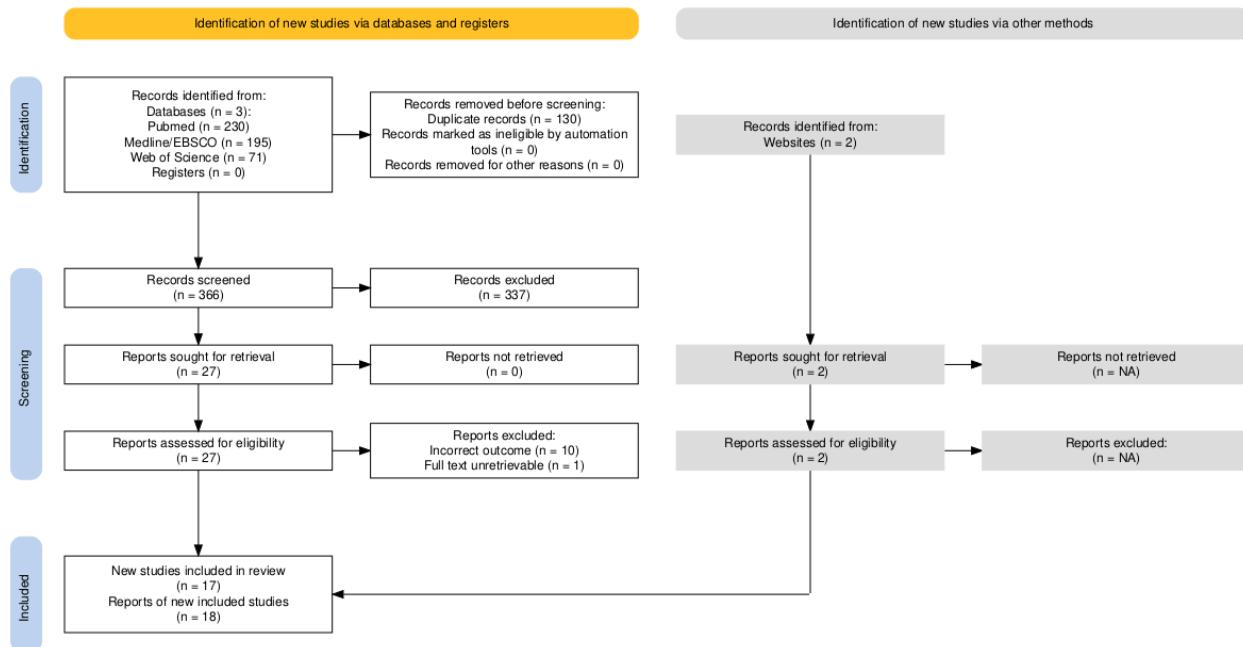


Figure 1: PRISMA 2020 flow diagram template for systematic reviews. Note that a “report” could be a journal article, preprint, conference abstract, study register entry, clinical study report, dissertation, unpublished manuscript, government report or any other document providing relevant information. The website noted was a prior narrative review on this topic by some of the authors (<https://macrofactorapp.com/pCos-BMR/>) which identified two studies not found in our systematic database search.

Mean Resting Energy Expenditure

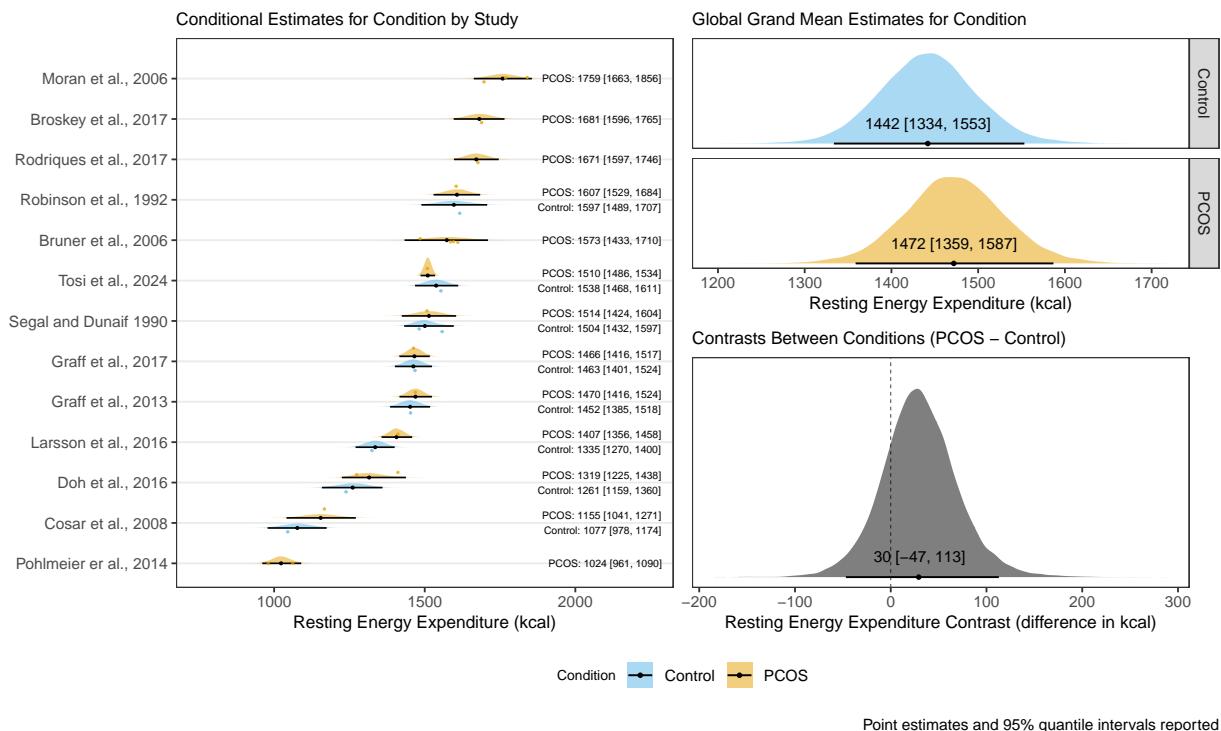


Figure 2: Posterior distribution, mean point estimates, and 95% quantile intervals for conditional estimates by study, global grand mean estimates by condition, and the contrast between conditions for mean resting energy expenditure of control women without PCOS and women with PCOS.

Standard Deviation of Resting Energy Expenditure

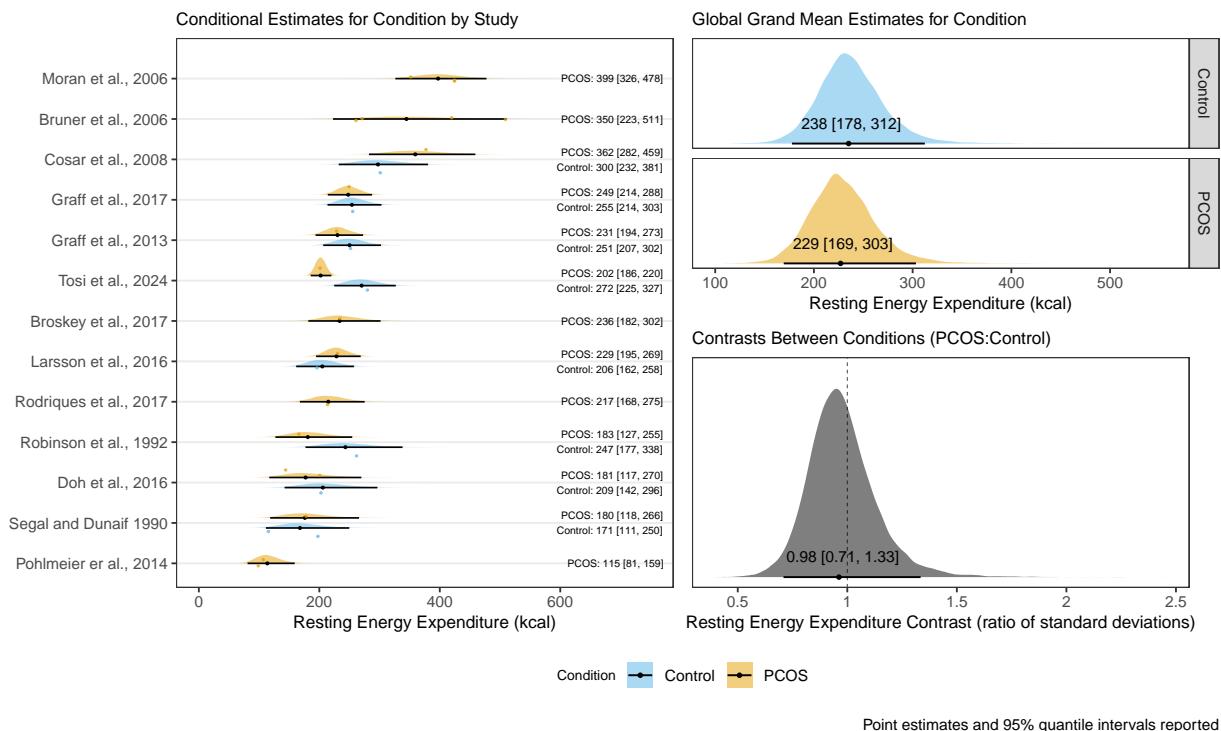


Figure 3: Posterior distribution, variance point estimates, and 95% quantile intervals for conditional estimates by study, global grand variance estimates by condition, and the contrast ratio between conditions for the standard deviation of resting energy expenditure of control women without PCOS and women with PCOS.