

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

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<sup>21</sup> engagements, affiliated social media partnerships, and book publishing. James Steele provides research con-  
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<sup>28</sup> **Abstract**

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

<sup>45</sup> 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 population<sup>1</sup>. 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<sup>2–6</sup>, along with being a leading cause of anovulatory  
51 infertility in women<sup>7</sup>. 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<sup>8,9</sup>.

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<sup>10–12</sup> and a reduced resting energy expenditure (REE)<sup>13</sup>. Indeed, the study from Georgopoulos  
58 et al.<sup>13</sup> 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 resistant<sup>13</sup>. 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<sup>14,15</sup>. 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<sup>16–18</sup>.

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 PCOS<sup>19</sup>. 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 REE<sup>20</sup>. Recommendations such as this could influence the well documented  
75 prevalence of eating disorders in women with PCOS<sup>21,22</sup>. 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<sup>23,24</sup> and are routinely recommended<sup>25</sup>. 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<sup>26–29</sup>. 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 Therefore, to estimate and describe the magnitude of difference in REE between women with and without  
84 PCOS, we completed a systematic review and meta-analysis of studies reporting REE in these populations.

## 85 Materials and Methods

86 This systematic review and meta-analysis was pre-registered on PROSPERO ([CRD42024601434](#)) initially on  
87 the 3<sup>rd</sup> of December 2024 and performed in accordance with the Preferred Reporting Items for Systematic  
88 Reviews and Meta-Analyses (PRISMA) statement guidelines<sup>30</sup>. The PRISMA flow diagram reported below  
89 (Figure 1) was produced using the PRISMA2020 R package and Shiny app<sup>31</sup>. The primary aim of this review  
90 was to examine the descriptive question “Does resting energy expenditure (REE) differ between women with  
91 and without polycystic ovary syndrome (PCOS)?”. We summarise and describe the studies in addition to  
92 quantitatively synthesising their results via meta-analysis.

## 93 Search Strategy

94 PubMed, Web of Science, and MEDLINE databases were searched using the following Boolean search string:  
95 ((“Basal Metabolic Rate”[MeSH] OR “Energy Metabolism”[MeSH] OR “Resting Metabolic Rate” OR RMR

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

## 105 **Eligibility Criteria**

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

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

114 The condition being studied was PCOS and we included observational cross-sectional design studies, in  
115 addition to intervention studies where REE was reported for the population (and if present, the comparator  
116 i.e., women without PCOS) condition of interest. For clarity, studies of any design were included if they  
117 reported the REE using the methods indicated for a sample of adult women with PCOS and who were  
118 otherwise healthy. This included both studies with and without samples of healthy control women without  
119 PCOS. As detailed in the statistical analysis section below, a Bayesian model with informative priors based  
120 on normative data for REE in healthy women without PCOS was included to provide control information  
121 indirectly where this was missing. The use of such priors is an efficient tool for incorporating historical

<sup>122</sup> information on a particular population in a conservative manner<sup>32</sup>.

<sup>123</sup> Following the PICO framework our eligibility criteria can be defined as follows:

<sup>124</sup> • Population

<sup>125</sup> – Inclusion criteria:

<sup>126</sup> \* Women

<sup>127</sup> \* 18-65 y

<sup>128</sup> \* With or without insulin resistance (IR)

<sup>129</sup> • Intervention(s) or exposure(s)

<sup>130</sup> – Otherwise healthy women with PCOS

<sup>131</sup> • Comparator(s) or control(s)

<sup>132</sup> – Otherwise healthy control women without PCOS

<sup>133</sup> • Outcome

<sup>134</sup> – Inclusion criteria

<sup>135</sup> \* Resting energy expenditure (REE) measured via multiple methods including direct/indirect  
<sup>136</sup> calorimetry, doubly labelled water.

<sup>137</sup> – Exclusion criteria:

<sup>138</sup> \* Studies using invalid or non-standard methods for measuring REE (e.g., predicted REE from  
<sup>139</sup> body composition or accelerometer data)

## <sup>140</sup> Data extraction (selection and coding)

<sup>141</sup> Data was extracted by one investigator and checked in triplicate. Bibliometric data including authors,  
<sup>142</sup> journal, and article titles were extracted. Descriptive statistics for age, body mass, fat mass, fat free mass,  
<sup>143</sup> height, BMI, race, physical activity levels, country of investigation, information regarding glucose/insulin  
<sup>144</sup> regulation and insulin resistance status (where available), diagnostic criteria for PCOS, and measurement  
<sup>145</sup> method and device were extracted for each arm within each study in addition to sample size. Descriptive  
<sup>146</sup> characteristics were then tabulated across studies for reporting.

147 For each arm, and observation time point if multiple observations reported (e.g., before and after an in-  
148 tervention), depending on what was reported by the authors we extracted the means, medians, standard  
149 deviations, standard errors, lower and upper range values, and interquartile range for the unadjusted and/or  
150 body mass adjusted and/or fat free mass adjusted REE values. Where REE values adjusted for body mass  
151 and/or fat free mass were reported we used the reported body mass and/or fat free mass mean values for  
152 that arm to convert them to unadjusted REE values (i.e., multiplied them by body mass and/or fat free  
153 mass mean values). Where means and/or standard deviations were missing the latter were either calculated  
154 from standard errors and sample size, or all both were estimated from lower and upper range, interquartile  
155 range, median, and sample size depending on the available information using the methods of Wan et al.<sup>33</sup>.  
156 Further, where missing, height/body mass/BMI were estimated based on the reported means. The units  
157 of measurement for which REE was extracted and all REE values were converted to kcal/day. In one case<sup>34</sup>  
158 REE was reported relative to body mass and the unadjusted values were no longer available (confirmed  
159 by the authors). As such, in this case we used the mean body mass to convert back to estimated REE  
160 unadjusted.

### 161 **Studies with possible reporting errors**

162 During data extraction it was noted that several studies from the same lab/research group<sup>13,35-37</sup> contained  
163 a number of discrepancies that seemed to be possible reporting errors. This included, based on taking the  
164 authors results as written, standard errors that implied impossible or at least incredibly unlikely standard  
165 deviations, and discrepancies in sample size reporting throughout for most variables without explanation or  
166 where this was explained the sample sizes were discrepant with the text. Further, data was not reported for the  
167 healthy control women without PCOS in three of the studies<sup>35-37</sup>, and REE was reported as an “adjusted”  
168 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  
169 obtained by substituting the individual lean body mass, fat mass, gender, and age in the linear regression  
170 equation generated by the data of all patients. In correspondence with the senior author we were unable to  
171 clarify the reporting discrepancies as the person responsible for the data/results was no longer contactable.  
172 The original data were also no longer available and so we could not calculate the unadjusted REE.

173 Given these issues we decided to extract the results from these studies as reported and to conduct analyses  
174 both with and without their inclusion. Though not pre-registered, due to a lack of confidence in the reported  
175 results, we decided to include the analysis omitting these studies as our main models in the results reported  
176 below. The results of the analysis including them are reported in the sensitivity analysis section.

## 177 Statistical Analysis

178 Statistical analysis of the data extracted was be 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,  
179 transformations, analyses, plotting, and reporting are available in the corresponding GitHub repository  
180 [https://github.com/jamessteeleii/pcos\\_ree\\_meta](https://github.com/jamessteeleii/pcos_ree_meta). We cite all software and packages used in the analysis  
181 pipeline using the `grateful` package<sup>38</sup> which can be seen here: [https://github.com/jamessteeleii/pcos\\_ree\\_meta/blob/main/grateful-report.pdf](https://github.com/jamessteeleii/pcos_ree_meta/blob/main/grateful-report.pdf). The statistical analysis plan was linked in our pre-registration  
182 (PROSPERO: CRD42024601434) and available at the accompanying GitHub repository. Any deviations  
183 from the pre-registration are noted below.

186 Given our research question our analysis was aimed at parameter estimation<sup>39</sup> within a Bayesian meta-  
187 analytic framework<sup>40</sup>. For all analyses model parameter estimates and their precision, along with conclusions  
188 based upon them, are interpreted continuously and probabilistically, considering data quality, plausibility  
189 of effect, and previous literature, all within the context of each model. The `renv` package<sup>41</sup> was used for  
190 package version reproducibility and a function based analysis pipeline using the `targets` package<sup>42</sup> was  
191 employed (the analysis pipeline can be viewed by downloading the R Project and running the function  
192 `targets::tar_visnetwork()`). Effect sizes and their variances were all calculated using the `metafor` pack-  
193 ages `escalc()` function<sup>43</sup>. The main package `brms`<sup>44</sup> was used in fitting all the Bayesian meta-analysis  
194 models. Prior and posterior draws were taken using `marginaleffects`<sup>45</sup> and `tidybayes`<sup>46</sup> packages. All  
195 visualisations are created using `ggplot2`<sup>47</sup>, `tidybayes`, and the `patchwork`<sup>48</sup> packages.

196 **Main Pre-registered Models**

197 We adopted an arm-based multiple condition comparison (i.e., network) type model given that the studies  
198 included had arms of women with PCOS both with, and without, a non-PCOS control arm<sup>49</sup>, and also in  
199 some cases multiple observations of REE in the different arms included in the study (for example, where an  
200 intervention was conducted and pre- and post-intervention REE was reported). In typical contrast-based  
201 meta-analyses data is limited to the effect sizes for paired contrasts between arms and thus studies that  
202 include both arms (i.e., relative effects between non-PCOS control vs PCOS arms); however, in arm-based  
203 analyses the data are the absolute effects within each arm and information is borrowed across studies to  
204 enable both within condition absolute, and between condition relative contrasts to be estimated. We made  
205 use of historical information regarding REE in healthy control women without PCOS by setting informative  
206 priors based on meta-analysis of large scale studies reporting normative data for REE in this population.  
207 This was included to provide indirect control information where it was missing from particular studies. The  
208 use of historical priors like this is an efficient tool to incorporate historical information about a particular  
209 population in a conservative manner in meta-analyses<sup>32</sup>. From this model we focus on reporting the global  
210 grand mean estimate for the fixed between condition relative contrast for non-PCOS control vs PCOS arms  
211 as our primary estimand of interest (i.e.,  $\beta_1$  in both mean and standard deviation models). We examined  
212 both raw mean REE (i.e., the absolute mean REE in kcals per day for each arm) in addition to the between  
213 person standard deviation in REE (i.e., the absolute standard deviation in REE in kcals per day for each  
214 arm). Both models were multilevel in that they included nested random intercepts for both study and arm  
215 within study. In addition, and in deviation from our pre-registration, we also included lab as a random  
216 intercept as in some cases we had multiple studies from the same lab or research group. Lastly, the inclusion  
217 of a random intercept for each effect size was accidentally omitted from our pre-registration, and so this is  
218 also included in the model.

219 **Mean REE Model**

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

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

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

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

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

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

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

221 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  
 222 and  $\sigma_{ijkl}^2$  is the corresponding sampling error for that estimate. The random intercepts for the  $i$ th lab,  $j$ th  
 223 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  
 224 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  $\beta_0$  represents  
 225 the fixed effect estimate of REE for control conditions and  $\beta_1$  the fixed effect estimate for the offset from  
 226 this for the PCOS conditions (i.e., the difference between conditions). The estimated offset was allowed to  
 227 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  
 228 also modelled as correlated with the corresponding random intercepts with covariance  $\Sigma_{\text{lab}}$  and  $\Sigma_{\text{study}}$ , and  
 229  $\text{corr}_{\text{lab}}$  and  $\text{corr}_{\text{study}}$  correlation matrices.

230 The priors for this model were as follows:

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

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

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

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

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

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

231 where the prior for  $\beta_0$ , which corresponded to the model intercept and mean REE in the control condition,  
232 was set based on meta-analysis of the mean REEs for women from two large studies of healthy people<sup>50,51</sup>  
233 though set with a conservative degrees of freedom for the Student-*t* distribution. The random intercept  $\sigma_{0,\text{study}}^2$   
234 was set similarly to this. The prior for the fixed effect  $\beta_1$ , reflecting the difference between control and PCOS  
235 conditions was set based on a wide range of possible values considering the minimum and maximum  
236 values of the ranges reported in the two studies noted (i.e., 2492 - 908 = 1584). We then set a prior that  
237 permits values approximately across this range of values with the majority of it's mass centred around zero.  
238 The remaining random effects were set based on the default weakly regularising priors for `brms` and scaled  
239 to the expected response values using a half-student-*t* distribution with 3 degrees of freedom and  $\mu = 0$ , and  
240 both correlation matrices  $\text{corr}_{\text{lab}}$  and  $\text{corr}_{\text{study}}$  were set with an LKJcorr(1) distribution.

#### 241 Standard Deviation of REE Model

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

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

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

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

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

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

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

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

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

244 where  $\log(\hat{\theta}_{ijkl})$  is the  $l$ th natural logarithm of the standard deviation of REE estimate from the  $k$ th arm,  
245 for the  $j$ th study, conducted by the  $i$ th lab and  $\sigma_{ijkl}^2$  is the corresponding sampling error for that estimate.  
246 The random intercepts for the  $i$ th lab,  $j$ th study,  $k$ th arm, and  $l$ th standard deviation of REE estimate are  
247  $\alpha_{0,\text{lab}[i]}$ ,  $\alpha_{0,\text{study}[j]}$ ,  $\alpha_{0,\text{arm}[k]}$ , and  $\alpha_{0,\text{effect}[l]}$  respectively each with standard deviation of  $\sigma_{0,\text{lab}[i]}^2$ ,  $\sigma_{0,\text{study}[j]}^2$ ,  
248  $\sigma_{0,\text{arm}[k]}^2$ , and  $\sigma_{0,\text{effect}[l]}^2$ . The parameter  $\beta_0$  represents the fixed effect estimate of standard deviation of REE  
249 for control conditions and  $\beta_1$  the fixed effect estimate for the offset from this for the PCOS conditions (i.e.,  
250 the difference between conditions). The estimated offset was allowed to vary across both labs and studies  
251 each reflected by  $\alpha_{1,\text{lab}[i]}$  and  $\alpha_{1,\text{study}[j]}$  respectively, and these effects were also modelled as correlated with  
252 the corresponding random intercepts with covariance  $\Sigma_{\text{lab}}$  and  $\Sigma_{\text{study}}$ , and  $\text{corr}_{\text{lab}}$  and  $\text{corr}_{\text{study}}$  correlation  
253 matrices. Finally,  $\beta_2$  represents the fixed effect of the natural logarithm of the corresponding mean REE  
254 estimate  $\tilde{m}$  which is modelled as estimated with measurement error i.e.,  $m$  represents the point estimate for  
255 the  $l$ th natural logarithm of the mean REE estimate from the  $k$ th arm, for the  $j$ th study, conducted by the  
256  $i$ th lab and  $\sigma_{\log(y_{i,\text{mean},[\text{ijkl}]})}^2$  is the corresponding sampling error for that estimate.

257 The priors for this model were as follows:

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

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

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

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

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

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

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

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

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

258 where the prior for  $\beta_0$ , which corresponded to the model intercept and standard deviation of REE in the  
259 control condition, was again set based on meta-analysis of the standard deviation of REEs for women from  
260 two large studies of healthy people<sup>50,51</sup> though set with a conservative degrees of freedom for the Student-*t*  
261 distribution. The random intercept  $\sigma_{0,\text{study}}^2$  was set similarly to this. The prior for the fixed effect  $\beta_1$ , reflecting  
262 the difference between control and PCOS conditions was set based on a wide range of possible values. Given  
263 that in many cases of variables in the field there is an approximate relationship of ~1 for the natural logarithm  
264 of the standard deviation conditioned upon the natural logarithm of the mean<sup>52</sup> we set this prior to reflect  
265 the range of differences on the on the log scale (i.e.,  $\log(1584)$ ). We then set a prior that permits values  
266 approximately across this range of values with the majority of it's mass centred around zero. The prior  
267 for the fixed effect  $\beta_2$ , reflecting the relationship between the natural logarithm of the mean REE with the  
268 natural logarithm of the standard deviation of REE, was set it to be weakly informative centred on zero  
269 with a wide scale to indicate uncertainty in this outcome specifically despite the typical relationship close  
270 to ~1. Priors for the measurement error of the natural logarithm of the mean REE estimate  $\tilde{m}$  were again  
271 based upon meta-analysis of the aforementioned studies, though measurement error has to be positive, as  
272 does the corresponding standard deviation of this error, so we set these to conservative wide half-student-*t*

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

276 **Post-processing of models**

277 For both models we examined trace plots along with  $\hat{R}$  values to examine whether chains have converged, and  
278 posterior predictive checks for each model to understand the model implied distributions. From each model  
279 we took draws from the posterior distributions for the conditional absolute estimates for each condition (i.e.,  
280 controls and PCOS) by study incorporating random effects, the global grand mean absolute estimates for  
281 each condition ignoring random effects, and the global grand mean between condition relative contrast for  
282 controls vs PCOS conditions ignoring random effects. The between condition relative contrast for controls  
283 vs PCOS conditions corresponded to  $\beta_1$  in each model and was our primary estimand of interest; for the  
284 mean REE model this corresponded to the absolute difference in mean REE, and for the standard deviation  
285 of REE model this corresponded to the natural logarithm of the ratio of standard deviations of REE which  
286 was exponentiated (note, all log standard deviation of REE model estimates were exponentiated back to the  
287 original scale to aid interpretability). We present the full probability density functions for posterior visually,  
288 and also to calculate mean and 95% quantile intervals (QI: i.e., ‘credible’ or ‘compatibility’ intervals) for  
289 each estimate providing the most probable value of the parameter in addition to the range from 2.5% to  
290 97.5% percentiles given our priors and data.

291 **Sensitivity analyses**

292 **Pairwise contrast based models**

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

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

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

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

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

296 where  $\hat{\theta}_{ijkl}$  is the pairwise effect size, either the mean difference in REE or the log coefficient of variation ratio  
 297 (calculated as PCOS vs control), for the  $j$ th study, conducted by the  $i$ th lab and  $\sigma_{ijkl}^2$  is the corresponding  
 298 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  
 299  $\alpha_{0,\text{study}[j]}$  respectively each with standard deviation of  $\sigma_{0,\text{lab}[i]}^2$ ,  $\sigma_{0,\text{study}[j]}^2$ . The parameter  $\beta_0$  represents the  
 300 fixed effect estimate of the pairwise effect size i.e., the pooled estimate of the contrast between conditions.  
 301 The priors for  $\beta_0$  in these models were set as default weakly regularising which is set on an intercept that  
 302 results when internally centering all population-level predictors around zero to improve sampling efficiency  
 303 and scaled to the expected response values using a student- $t$  distribution; for the mean difference in REE  
 304 this was student- $t(3, 5.5, 50.2)$  and for the log coefficient of variance ratio this was student- $t(3, -0.1, 2.5)$ .  
 305 The random effects were set similarly scaled to the expected response values but using a half-student- $t$   
 306 distribution centred on zero. From these models we calculated the mean and 95% quantile intervals (i.e.,  
 307 ‘credible’ or ‘compatibility’ intervals) for the  $\beta_0$  (comparable to the  $\beta_1$  from the corresponding mean and  
 308 standard deviation of REE arm-based models) for each effect size providing the most probable value of the  
 309 parameter in addition to the range from 2.5% to 97.5% percentiles given our priors and data.

### 310 Additional sensitivity analyses

311 As noted in the section above, “*Studies with possible reporting errors*”, we opted to conduct analysis with  
 312 and without the inclusion of four studies with possible reporting errors we could not resolve<sup>13,35–37</sup>. Thus  
 313 the main models described above were run with and without these studies, the main results are presented  
 314 without them and the sensitivity results with them are reported separately below.  
 315 As a further sensitivity analysis, given the inclusion of some studies with interventions in women with

316 PCOS reporting REE at multiple timepoints such as mid- and post-intervention<sup>34,53,54</sup> but lacking of control  
317 women without PCOS, we opted to also conduct sensitivity analysis excluding these and only examining  
318 their baseline results in addition to the cross-sectional studies. As such, the main models described above  
319 were run without the follow-up (i.e., mid- or post-intervention timepoints) from these studies and only using  
320 the baseline results in addition to other cross-sectional studies.

321 Lastly, given the role of body mass and, in particular, highly metabolically active tissue on REE we included  
322 models adjusted for these group level characteristics where studies reported them. These models were  
323 essentially extensions of the main models noted above for BMI and fat-free mass where each was modelled  
324 with its corresponding sampling error similarly to how the log mean REE was modelled in the models for  
325 standard deviation of REE i.e., the mean BMI or fat-free mass estimates were modelled as estimated with  
326 measurement error. When extracting posterior distributions for the contrasts between conditions in these  
327 models both BMI and fat-free mass were adjusted to the median values seen in the control conditions i.e.,  
328 BMI = 26.03 kg/m<sup>2</sup> and fat-free mass = 48.8 kg.

## 329 Results

### 330 Systematic Review

331 Note, the numbers in this section exclude the four studies previously noted with possible reporting issues  
332 that were unresolved. These studies are however summarised in fully in the descriptive characteristics table  
333 in the online supplementary materials (see [Descriptives Table](#) where some of the possible reporting errors  
334 are seen in the standard deviations reported or calculated e.g., the standard deviation calculated for body  
335 mass of women with PCOS in Saltamavros et al.<sup>36</sup>. The PRISMA flow diagram (Figure 1) also includes all  
336 studies identified including the four with possible errors.

337 Our systematic review identified 13 studies from 12 lab/research groups including 24 arms (Control arms =  
338 9, PCOS arms = 15) and a total of 918 participants (Controls: minimum n = 9, median n = 29, maximum  
339 n = 54; PCOS: minimum n = 5, median n = 28, maximum n = 266). Descriptive characteristics of the arms  
340 and participants in these studies are reported fully in the online supplementary materials (see [Descriptives](#)

341 [Table](#)).

342 Studies were carried out in multiple countries: Brazil ( $k = 3$ ), and USA ( $k = 3$  studies), Australia, Cameroon,  
343 Canada, Italy, Sweden, Turkey, UK (all  $k = 1$  study). The four studies with noted reporting errors were  
344 carried out in Greece. A total of 11 studies used the Rotterdam criteria (or a modified version thereof) for  
345 diagnosing PCOS<sup>16,17,34,53–60</sup>. One study used the 1990 National Institutes of Health criteria<sup>61</sup>, and two  
346 studies<sup>18,55</sup> diagnosed PCOS via the presence of oligomenorrhea or amenorrhea alongside additional criteria  
347 including plasma androgen levels, hirsutism or polycystic ovaries on ultrasound scanning. All but one study<sup>61</sup>,  
348 which used doubly labelled water, measured REE using indirect calorimetry. The specific devices reported  
349 by these studies are included in the online supplementary materials (see [Descriptives Table](#)). We originally  
350 considered in our pre-registration that, given sufficient data, we would compare sub groups of women with  
351 PCOS who did and did not have accompanying insulin resistance. However, based on the metabolic health  
352 variables reported in studies with this information (see [Descriptives Table](#)) and, where available, considering  
353 primary criteria of either homeostatic model assessment of insulin resistance (HOMA-IR)  $\geq 2.5$  or secondary  
354 criteria including fasting insulin  $> 12\mu\text{U}/\text{mL}$  or fasting glucose  $\geq 100\text{mg}/\text{dL}$ , all groups of women with  
355 PCOS in the included studies would be considered to have insulin resistance. Mean age of women with  
356 PCOS in these studies ranged from 23 to 33 which was similar to the control women without PCOS ranging  
357 from 23 to 30. Across those studies where BMI was reported or it was possible to estimate the mean BMI of  
358 women with PCOS ranged from 26.4 to 39.9 was typically greater than women without PCOS which ranged  
359 20.5 to 27.9.

## 360 Mean REE Model Results

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

<sup>367</sup> visualisation of the posterior distribution and the conditional estimates by study, can be seen in Figure 2.

## <sup>368</sup> Standard Deviation of REE Model Results

<sup>369</sup> The main model for the between participant standard deviation of REE resulted in a posterior distribution  
<sup>370</sup> for the contrast ratio between control and PCOS conditions with a mean point estimate of 0.98 with a 95%  
<sup>371</sup> quantile interval ranging from 0.71 to 1.33 suggesting there is a 95% probability that the true ratio of standard  
<sup>372</sup> deviations lies between these values given our priors and the data from included studies. The corresponding  
<sup>373</sup> conditional estimates for the standard deviations of the control condition and PCOS condition respectively  
<sup>374</sup> were 238 kcal/day [95%QI:178 kcal/day to 312 kcal/day] and 229 kcal/day [95%QI:169 kcal/day to 303  
<sup>375</sup> kcal/day]. These results, including the full visualisation of the posterior distribution and the conditional  
<sup>376</sup> estimates by study, can be seen in Figure 3.

## <sup>377</sup> Sensitivity Analyses

### <sup>378</sup> Pairwise Models

<sup>379</sup> For mean REE the pairwise model resulted in qualitatively similar inferences suggesting little difference  
<sup>380</sup> between control and PCOS conditions with mean point estimate of 16 kcal/day with a 95% quantile interval  
<sup>381</sup> ranging from -36 kcal/day to 70 kcal/day. This was similar for the standard deviation of REE with the  
<sup>382</sup> pairwise model resulting in a contrast ratio between the control and PCOS conditions with a mean point  
<sup>383</sup> estimate of 0.9 and a 95% quantile interval ranging from 0.6 to 1.35.

### <sup>384</sup> Models including studies with possible reporting issues

<sup>385</sup> For mean REE the model including the studies noted with possible reporting issues that were not  
<sup>386</sup> resolved<sup>13,35-37</sup> still resulted in qualitatively similar inferences suggesting little difference between control  
<sup>387</sup> and PCOS conditions with mean point estimate of 15 kcal/day with a 95% quantile interval ranging from  
<sup>388</sup> -69 kcal/day to 95 kcal/day. This was similar for the standard deviation of REE with the model including  
<sup>389</sup> these studies resulting in a contrast ratio between the control and PCOS conditions with a mean point  
<sup>390</sup> estimate of 1.21 and a 95% quantile interval ranging from 0.68 to 2.13.

**391 Basline REE Measurement Models**

**392** For mean REE the model which included only baseline REE measurements from studies involving  
**393** interventions<sup>34,53,54</sup>, in addition to other cross-sectional studies, also resulted in qualitatively similar  
**394** inferences suggesting little difference between control and PCOS conditions with mean point estimate of 32  
**395** kcal/day with a 95% quantile interval ranging from -46 kcal/day to 118 kcal/day. This was also the case for  
**396** the standard deviation of REE with this model resulting in a contrast ratio between the control and PCOS  
**397** conditions with a mean point estimate of 0.97 and a 95% quantile interval ranging from 0.71 to 1.36.

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

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

**408 Discussion**

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

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

418 These findings challenge the widely held belief that PCOS is inherently associated with a slower  
419 metabolism<sup>@ 25</sup>, predisposing women with PCOS to weight gain. This belief largely stems from a single  
420 but influential 2009 study from Georgopoulos et al. that reported a significantly reduced BMR in women  
421 with PCOS<sup>13</sup>, which has been widely cited and reinforced in both academic and clinical contexts. However,  
422 as we have noted, this study along with others<sup>35-37</sup> have numerous reporting errors which led us to  
423 drop them from our present analysis (though sensitivity analysis including them did not alter our overall  
424 conclusions). The mistaken belief that REE is lower for those with PCOS may have mistakenly lead to  
425 recommendations centred on slightly more severe calorie restriction to achieve weight-loss goals, compared  
426 with recommendations for the general population, as primary management strategies for women with  
427 PCOS<sup>20</sup>. Recognising that there may be minimal differences in REE between women with PCOS and  
428 those without can inform both clinical and public practices, potentially leading to a shift in focus away  
429 from a requirement for more severe caloric restriction as a primary method of treatment towards more  
430 comprehensive, individualised, and psychologically safe approaches to care<sup>25</sup>.

431 The current study suggests that REE may not a barrier to weight regulation in PCOS given small group-  
432 level differences between women with PCOS and those without (1472 kcal/day [95%QI:1359 kcal/day to 1587  
433 kcal/day] versus 1442 kcal/day [95%QI:1334 kcal/day to 1553 kcal/day], respectively). If anything, REE  
434 may be slightly higher in women with PCOS compared to healthy women without PCOS. BMI of women  
435 with PCOS ranged from 26.4 to 39.9 was typically greater than women without PCOS which ranged 20.5 to  
436 27.9 and this may explain the slightly greater REE in the former group. However, for those studies where  
437 we could extract or estimate BMI, our additional exploratory models adjusted for this similarly showed  
438 little difference in REE (20 kcal/day [95%QI: -96 kcal/day to 140kcal/day]) between women with PCOS  
439 and those without. However, one of the studies included in our analysis<sup>62</sup> reported that, whilst there was  
440 little difference in unadjusted REE, when REE was adjusted for fat-free mass it was lower in women with  
441 PCOS suggesting the potential importance of fat-free tissue in energy regulation. Yet, for those studies  
442 where fat-free mass was reported, our additional exploratory models adjusted for this similarly showed little

443 difference in REE (-9 kcal/day [95%QI: -195 kcal/day to 168kcal/day]) between women with PCOS and  
444 those without. As such, even adjusted for both BMI and fat-free mass, there seems to be little difference in  
445 REE between women with, and without, PCOS. Yet, a recent systematic review of mechanisms for metabolic  
446 dysfunction has reported excess androgen drives metabolic issues within adipose tissue and muscle tissue  
447 contributing to complications like obesity and insulin resistance<sup>63</sup>. Taken together, these findings highlight  
448 that factors beyond REE and typical correlates of this including BMI or fat-free mass, such as hormonal and  
449 tissue-specific metabolic effects, may play a more significant role in weight regulation challenges in women  
450 with PCOS.

451 As noted, women with PCOS are more likely to engage in weight-loss attempts<sup>19</sup> and there could be con-  
452 cerns that this could inadvertently further foster the already well documented disordered eating in this  
453 population<sup>21,22,64,65</sup>. The pathways linking PCOS and disordered eating are multifactorial. Biological mech-  
454 anisms such as hyperandrogenism, hyperinsulinaemia, and altered ghrelin and leptin signalling can heighten  
455 hunger, carbohydrate cravings, and appetite variability<sup>66</sup>. Frequent hypoglycaemia and associated mood  
456 changes have also been observed, which can trigger compensatory eating or binge episodes<sup>66</sup>. These physi-  
457 ological processes interact with psychological and social stressors, including infertility concerns, conflicting  
458 nutrition advice, chronic dieting, and exposure to idealised body images on social media, which together  
459 compound vulnerability to disordered eating<sup>67</sup>. Moreover, eating disorders themselves can disrupt endocrine  
460 function, potentially worsening PCOS symptoms and creating a self-reinforcing cycle<sup>65,68</sup>.

461 Understanding the intertwined biological, psychological, and social influences suggests the importance of  
462 considering whether restrictive dietary advice is appropriate given it may exacerbate feelings of failure,  
463 hunger dysregulation, and shame<sup>69</sup>. These concerns, coupled with the lack of difference in REE between  
464 women with and without PCOS might suggest that energy restriction based dietary interventions for weight-  
465 loss may be unnecessary. But, there is also evidence supporting the effect of energy restricted dietary  
466 interventions for improving PCOS symptoms<sup>23,24</sup> and they are recommended in international guidelines<sup>25</sup>.  
467 Encouragingly though, these guidelines also recognise weight stigma as a determinant of health and call for its  
468 reduction across clinical and public health settings. Evidently the greater prevalence of women with PCOS  
469 falling into overweight and obese BMI categories compared to women without PCOS<sup>8,9</sup> is unlikely to be due

470 to differences in REE and so, despite the potential effectiveness of energy restriction dietary interventions,  
471 there is potential value in moving towards more weight-neutral, individualised, and empowering care following  
472 holistic guidelines recommending multiple approaches to management<sup>20,25</sup>.

## 473 **Strengths and Limitations**

474 The current study has multiple strengths stemming from its preregistered, comprehensive methodology and  
475 Bayesian statistical approach. This statistical framework allowed us to incorporate studies with and without  
476 control groups to better estimate REE in women with and without PCOS and to perform multiple sensitivity  
477 analyses that confirmed the stability of our findings. However, a limitation here is that variability in methods  
478 across studies, such as differences in PCOS diagnostic criteria and REE testing protocols, may have influenced  
479 results and the relatively small number of studies overall limits the extent to which we can explore these  
480 potential moderators. Furthermore, some studies controlled for body weight or body composition when  
481 reporting REE values, while others did not. We accounted for this by estimating or converting reported  
482 data to obtain unadjusted REE values across all groups, thereby reducing this variability and further as  
483 noted above provided estimates adjusted for BMI and fat-free mass both of which had little influence on our  
484 conclusions. Another limitation is that, due to fewer total control groups than PCOS groups, informed priors  
485 were required in several statistical models. However, in the context of Bayesian meta-analysis this can also  
486 be considered a strength. Additionally, some studies reported data inconsistencies that could not be clarified  
487 (e.g.,<sup>13,35–37</sup>) and were dropped from our main analysis though our conclusions again did not qualitative  
488 change when we conducted sensitivity analyses including these studies. Finally, most included studies were  
489 cross-sectional or baseline assessments within intervention trials, which limits causal inference. Indeed, we  
490 did not pre-register any kind of causal model (e.g., a directed acyclic graph) to inform our analysis approach  
491 for causal inference and as such have been explicit about the estimates presented as being descriptive.

## 492 **Conclusion**

493 In conclusion, the findings from this meta-analysis indicate that REE does not meaningfully differ between  
494 women with and without PCOS. Group-level differences in REE were small, insignificant, or not physiologi-

495 cally relevant. Additionally, variability in REE between individuals was also similar. These results suggest  
496 that a lower baseline REE is not associated with the weight-related challenges often associated with PCOS.  
497 These findings challenge the popular narrative that women with PCOS have a lower REE and may help  
498 better inform dietary interventions and nutritional support for these individuals. Future research should  
499 include more standardized REE measurement and reporting protocols, greater data transparency, consistent  
500 control and reporting of body weight or body composition, the presentation of both absolute and relative  
501 REE, and more precise characterization of PCOS phenotypes. Overall, these findings support the conclu-  
502 sion that PCOS is not negatively associated with REE and may help practitioners and researchers focus on  
503 individually targeted and holistic lifestyle interventions rather than negatively framed interventions based  
504 on unsupported assumptions regarding REE.

## 505 Data Availability

506 All code utilised for data preparation, transformations, analyses, plotting, and reporting are available in the  
507 corresponding GitHub repository [https://github.com/jamessteeleii/pcos\\_ree\\_meta](https://github.com/jamessteeleii/pcos_ree_meta).

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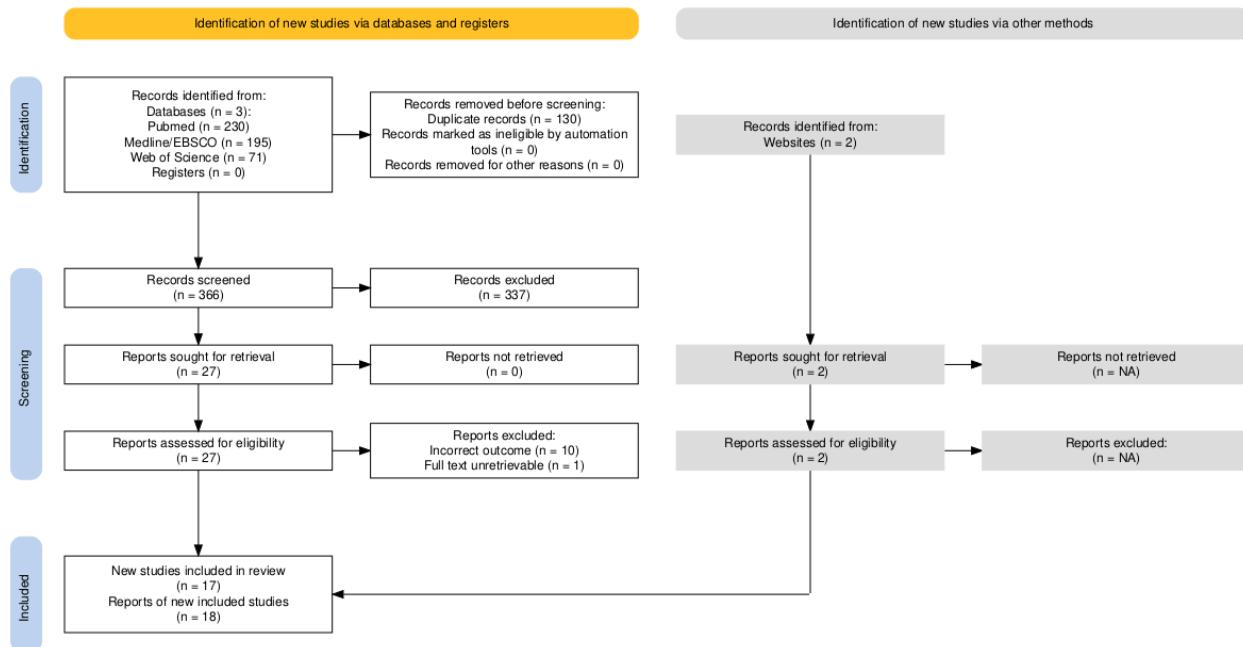


Figure 1: PRISMA 2020 flow diagram template for systematic reviews. Note that a “report” could be a journal article, preprint, conference abstract, study register entry, clinical study report, dissertation, unpublished manuscript, government report or any other document providing relevant information. 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

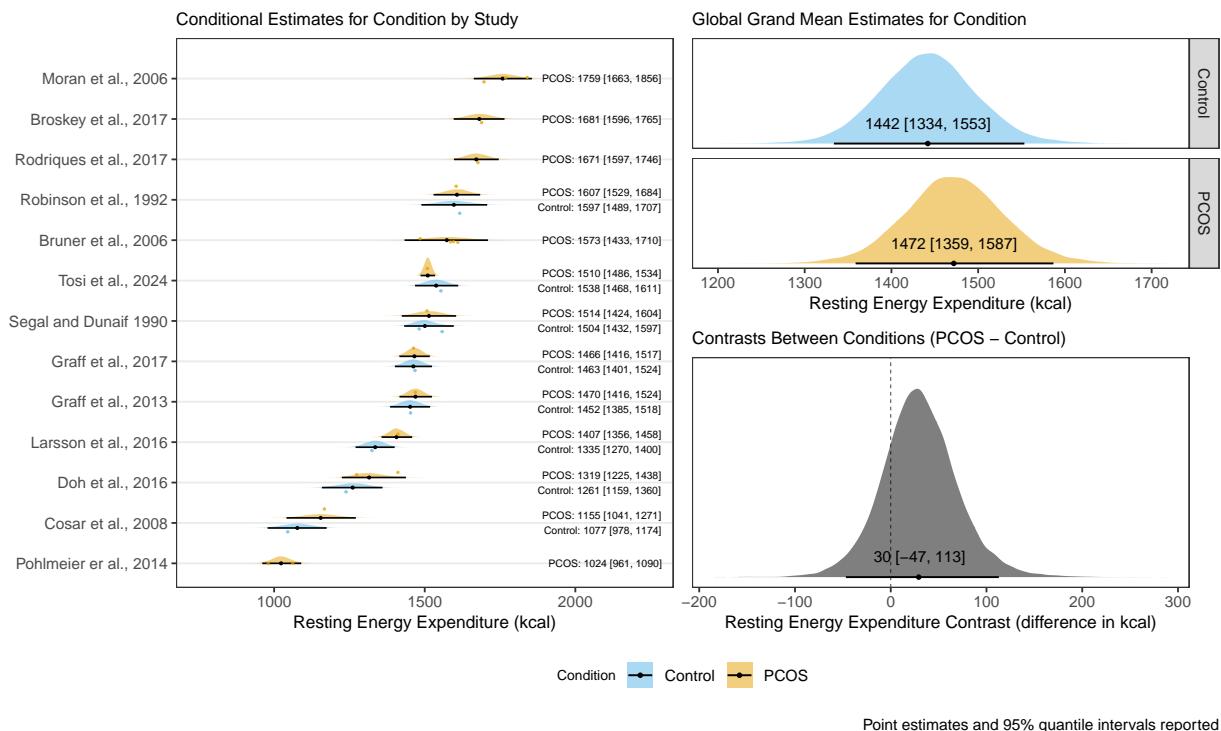


Figure 2: Posterior distribution, mean point estimates, and 95% quantile intervals for conditional estimates by study, global grand mean estimates by condition, and the contrast between conditions for mean resting energy expenditure of control women without PCOS and women with PCOS.

### Standard Deviation of Resting Energy Expenditure

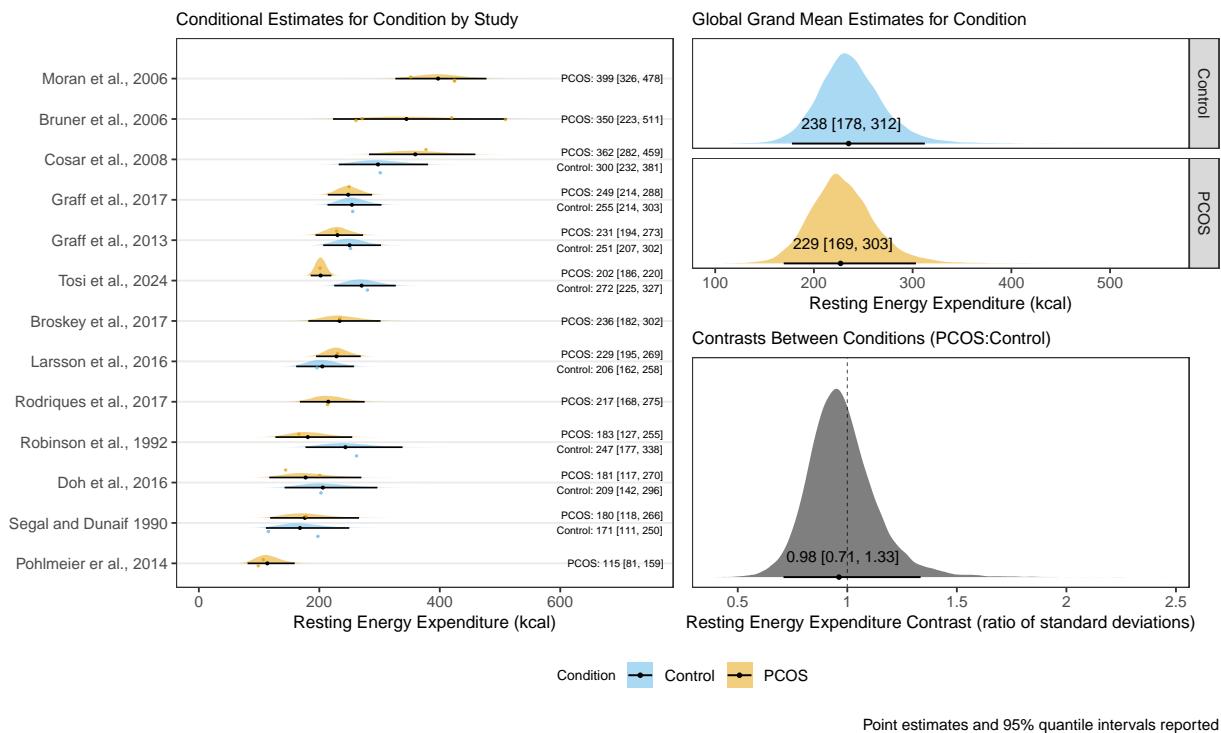


Figure 3: Posterior distribution, variance point estimates, and 95% quantile intervals for conditional estimates by study, global grand variance estimates by condition, and the contrast ratio between conditions for the standard deviation of resting energy expenditure of control women without PCOS and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