

<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>32</sup> **Abstract**

<sup>33</sup> Context: Polycystic ovary syndrome (PCOS) is common in reproductive-age women, who often have higher  
<sup>34</sup> BMI classification. This is assumed to stem from lower resting energy expenditure (REE), influencing  
<sup>35</sup> lifestyle intervention guidelines. However, evidence for reduced REE in women with PCOS compared with  
<sup>36</sup> those without is inconsistent. Objective: To systematically search and meta-analyse the existing literature  
<sup>37</sup> to estimate and describe the difference in REE between women with and without PCOS. Data Sources: A  
<sup>38</sup> systematic search was conducted using PubMed, Medline and Web of Science databases of published research  
<sup>39</sup> from January 1990 to January 2025. Study Selection: Studies that measured REE in women living with  
<sup>40</sup> PCOS, both with and without control arms of women without PCOS, were included. Data Extraction:  
<sup>41</sup> Bibliometric, demographic, and REE data was extracted by one investigator and checked in triplicate. Data  
<sup>42</sup> Synthesis: Thirteen studies were included in a Bayesian arm-based multiple condition comparison (i.e.,  
<sup>43</sup> network) type meta-analysis model with informative priors to compare both mean REE, and between person  
<sup>44</sup> variation in REE, between women with and without PCOS. Mean REE differed between groups by 30  
<sup>45</sup> kcal/day [95% quantile interval: -47 to 113 kcal/day] and the contrast ratio for between person standard  
<sup>46</sup> deviations was 0.98 [95% quantile interval: 0.71 to 1.33]. Conclusions: These findings indicate that REE does  
<sup>47</sup> not meaningfully differ between women with and without PCOS. Group-level differences in resting energy  
<sup>48</sup> expenditure are small, insignificant, or not physiologically relevant.

## 49 Introduction

50 Polycystic ovary syndrome (PCOS) affects approximately 10% of women of reproductive age worldwide,  
51 making it the most common endocrine disorder affecting this population<sup>1</sup>. Due to several factors including  
52 hyperandrogenism and alterations in insulin resistance, PCOS is believed to contribute to an increased risk of  
53 diabetes, metabolic syndrome and cardiovascular disease<sup>2-6</sup>, along with being a leading cause of anovulatory  
54 infertility in women<sup>7</sup>. Furthermore, epidemiological data has consistently demonstrated that women with  
55 PCOS are significantly more likely to suffer from overweight or obesity, compared to the general female  
56 population, with estimates ranging from 38% to 88% of PCOS patients falling into overweight or obese body  
57 mass index (BMI) categories<sup>8,9</sup>.

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

71 The consequences of widespread acceptance that REE is substantially lower in women living with PCOS  
72 should not be underestimated, particularly in light of the aforementioned incidence of overweight and obesity  
73 in this population. Women with PCOS typically engage in more frequent weight-loss attempts than women  
74 without PCOS<sup>19</sup>. From a physiological perspective, if women with PCOS do exhibit a lower REE, this

75 could imply a meaningful metabolic disadvantage that may influence dietary and nutritional guidance for  
76 weight management; for example, recommending a slightly more severe energy restriction to overcome the  
77 belief that they have a lower REE<sup>20</sup>. Recommendations such as this could influence the well documented  
78 prevalence of eating disorders in women with PCOS<sup>21,22</sup>. Contrastingly, belief in a “slower metabolism”  
79 could instead serve as a deterrent to energy restriction based weight-loss approaches for some women in line  
80 with typical general population recommendations that are also recognised as efficacious for improving PCOS  
81 symptoms<sup>23,24</sup> and are routinely recommended<sup>25,26</sup>. It has been well documented that women with PCOS  
82 already experience higher rates of anxiety, depression, and lower quality of life (QOL) as a result of negative  
83 body image and weight-related concerns<sup>27–30</sup>. Clarifying the relationship between REE and PCOS may  
84 therefore help guide more accurate clinical recommendations and empower both practitioners and women  
85 with PCOS.

86 A recent scoping review has highlighted the inconsistency in research examining energy expenditure in  
87 women with PCOS<sup>31</sup>. However, to our knowledge there has been no attempts to systematically review, and  
88 quantitatively synthesise via meta-analysis, studies of REE in women with, and without, PCOS. Therefore,  
89 to estimate and describe the magnitude of difference in REE between women with and without PCOS, we  
90 completed a systematic review and meta-analysis of studies reporting REE in these populations.

## 91 Materials and Methods

92 This systematic review and meta-analysis was pre-registered on PROSPERO ([CRD42024601434](#)) initially on  
93 the 3<sup>rd</sup> of December 2024 and performed in accordance with the Preferred Reporting Items for Systematic  
94 Reviews and Meta-Analyses (PRISMA) statement guidelines<sup>32</sup>. The PRISMA flow diagram reported below  
95 (Figure 1) was produced using the PRISMA2020 R package and Shiny app<sup>33</sup>. The primary aim of this review  
96 was to examine the descriptive question “Does resting energy expenditure (REE) differ between women with  
97 and without polycystic ovary syndrome (PCOS)?”. We summarise and describe the studies in addition to  
98 quantitatively synthesising their results via meta-analysis.

<sup>99</sup> **Search Strategy**

<sup>100</sup> PubMed, Web of Science, and MEDLINE databases were searched using the following Boolean search string:  
<sup>101</sup> ((“Basal Metabolic Rate”[MeSH] OR “Energy Metabolism”[MeSH] OR “Resting Metabolic Rate” OR RMR  
<sup>102</sup> OR “Resting Energy Expenditure” OR REE OR “Basal Metabolic Rate” OR BMR OR “resting energy” OR  
<sup>103</sup> “basal energy expenditure”) AND (“Polycystic Ovary Syndrome”[MeSH] OR “Polycystic Ovary Syndrome”  
<sup>104</sup> OR PCOS OR “Polycystic Ovarian Disease” OR “Stein-Leventhal Syndrome”)). Searches were limited to  
<sup>105</sup> publications up until May 2025 when the search was completed, limited to English language articles, and  
<sup>106</sup> Rayyan was used to manage the search and screening process. Two reviewers (RK and GK) independently  
<sup>107</sup> screened all titles and abstracts against the predefined inclusion and exclusion criteria. Articles deemed  
<sup>108</sup> potentially eligible by either reviewer were retrieved in full text. Full texts were then independently assessed  
<sup>109</sup> by RK and LP to determine final eligibility. Any disagreements at either stage were resolved through  
<sup>110</sup> discussion, and when consensus could not be reached, a third reviewer acted as an adjudicator.

<sup>111</sup> **Eligibility Criteria**

<sup>112</sup> Studies were included in the systematic review if 1) participants were confirmed as women with PCOS  
<sup>113</sup> between the ages of 18 to 65 years of age with or without insulin resistance; 2) otherwise healthy (e.g.,  
<sup>114</sup> non-diabetic, no cardiovascular disease); 3) had a measure of REE measured via multiple methods including  
<sup>115</sup> direct/indirect calorimetry, doubly labelled water; and 4) trials were not retracted at the time of this analysis.

<sup>116</sup> Studies were excluded if they 1) used invalid or non-standard methods for measuring REE (e.g., predicted  
<sup>117</sup> REE from body composition or accelerometer data); 2) non-peer-reviewed journal articles (including grey  
<sup>118</sup> literature sources such as conference abstracts, theses and dissertations); and 3) were secondary analyses  
<sup>119</sup> with the same primary outcome data as another included study.

<sup>120</sup> The condition being studied was PCOS and we included observational cross-sectional design studies, in  
<sup>121</sup> addition to intervention studies where REE was reported for the population (and if present, the comparator  
<sup>122</sup> i.e., women without PCOS) condition of interest. For clarity, studies of any design were included if they  
<sup>123</sup> reported the REE using the methods indicated for a sample of adult women with PCOS and who were  
<sup>124</sup> otherwise healthy. This included both studies with and without samples of healthy control women without

125 PCOS. As detailed in the statistical analysis section below, a Bayesian model with informative priors based  
126 on normative data for REE in healthy women without PCOS was included to provide control information  
127 indirectly where this was missing. The use of such priors is an efficient tool for incorporating historical  
128 information on a particular population in a conservative manner<sup>34</sup>.

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

130 • Population

131 – Inclusion criteria:

132 \* Women

133 \* 18-65 y

134 \* With or without insulin resistance (IR)

135 • Intervention(s) or exposure(s)

136 – Otherwise healthy women with PCOS

137 • Comparator(s) or control(s)

138 – Otherwise healthy control women without PCOS

139 • Outcome

140 – Inclusion criteria

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

143 – Exclusion criteria:

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

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

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

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

153 For each arm, and observation time point if multiple observations reported (e.g., before and after an in-  
154 tervention), depending on what was reported by the authors we extracted the means, medians, standard  
155 deviations, standard errors, lower and upper range values, and interquartile range for the unadjusted and/or  
156 body mass adjusted and/or fat free mass adjusted REE values. Where REE values adjusted for body mass  
157 and/or fat free mass were reported we used the reported body mass and/or fat free mass mean values for  
158 that arm to convert them to unadjusted REE values (i.e., multiplied them by body mass and/or fat free  
159 mass mean values). Where means and/or standard deviations were missing the latter were either calculated  
160 from standard errors and sample size, or all both were estimated from lower and upper range, interquartile  
161 range, median, and sample size depending on the available information using the methods of Wan et al.<sup>35</sup>.  
162 Further, where missing, height/body mass/BMI where estimated based on the reported means. The units  
163 of measurement for which REE was extracted and all REE values were converted to kcal/day. In one case<sup>36</sup>  
164 REE was reported relative to body mass and the unadjusted values were no longer available (confirmed  
165 by the authors). As such, in this case we used the mean body mass to convert back to estimated REE  
166 unadjusted.

167 **Studies with possible reporting errors**

168 During data extraction it was noted that several studies from the same lab/research group<sup>13,37-39</sup> contained  
169 a number of discrepancies that seemed to be possible reporting errors. This included, based on taking the  
170 authors results as written, standard errors that implied impossible or at least incredibly unlikely standard  
171 deviations, and discrepancies in sample size reporting throughout for most variables without explanation or  
172 where this was explained the sample sizes were discrepant with the text. Further, data was not reported for the  
173 healthy control women without PCOS in three of the studies<sup>37-39</sup>, and REE was reported as an “adjusted”  
174 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  
175 obtained by substituting the individual lean body mass, fat mass, gender, and age in the linear regression  
176 equation generated by the data of all patients. In correspondence with the senior author we were unable to

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

## 183 Statistical Analysis

184 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,  
185 transformations, analyses, plotting, and reporting are available in the corresponding GitHub repository  
186 [https://github.com/jamessteeleii/pcos\\_ree\\_meta](https://github.com/jamessteeleii/pcos_ree_meta). We cite all software and packages used in the analysis  
187 pipeline using the `grateful` package<sup>40</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  
188 (PROSPERO: CRD42024601434) and available at the accompanying GitHub repository. Any deviations  
189 from the pre-registration are noted below.

190 Given our research question our analysis was aimed at parameter estimation<sup>41</sup> within a Bayesian meta-  
191 analytic framework<sup>42</sup>. For all analyses model parameter estimates and their precision, along with conclusions  
192 based upon them, are interpreted continuously and probabilistically, considering data quality, plausibility  
193 of effect, and previous literature, all within the context of each model. The `renv` package<sup>43</sup> was used for  
194 package version reproducibility and a function based analysis pipeline using the `targets` package<sup>44</sup> was  
195 employed (the analysis pipeline can be viewed by downloading the R Project and running the function  
196 `targets::tar_visnetwork()`). Effect sizes and their variances were all calculated using the `metafor` pack-  
197 ages `escalc()` function<sup>45</sup>. The main package `brms`<sup>46</sup> was used in fitting all the Bayesian meta-analysis  
198 models. Prior and posterior draws were taken using `marginaleffects`<sup>47</sup> and `tidybayes`<sup>48</sup> packages. All  
199 visualisations are created using `ggplot2`<sup>49</sup>, `tidybayes`, and the `patchwork`<sup>50</sup> packages.

202 **Main Pre-registered Models**

203 We adopted an arm-based multiple condition comparison (i.e., network) type model given that the studies  
204 included had arms of women with PCOS both with, and without, a non-PCOS control arm<sup>51</sup>, and also in  
205 some cases multiple observations of REE in the different arms included in the study (for example, where an  
206 intervention was conducted and pre- and post-intervention REE was reported). In typical contrast-based  
207 meta-analyses data is limited to the effect sizes for paired contrasts between arms and thus studies that  
208 include both arms (i.e., relative effects between non-PCOS control vs PCOS arms); however, in arm-based  
209 analyses the data are the absolute effects within each arm and information is borrowed across studies to  
210 enable both within condition absolute, and between condition relative contrasts to be estimated. We made  
211 use of historical information regarding REE in healthy control women without PCOS by setting informative  
212 priors based on meta-analysis of large scale studies reporting normative data for REE in this population.  
213 This was included to provide indirect control information where it was missing from particular studies. The  
214 use of historical priors like this is an efficient tool to incorporate historical information about a particular  
215 population in a conservative manner in meta-analyses<sup>34</sup>. From this model we focus on reporting the global  
216 grand mean estimate for the fixed between condition relative contrast for non-PCOS control vs PCOS arms  
217 as our primary estimand of interest (i.e.,  $\beta_1$  in both mean and standard deviation models). We examined  
218 both raw mean REE (i.e., the absolute mean REE in kcals per day for each arm) in addition to the between  
219 person standard deviation in REE (i.e., the absolute standard deviation in REE in kcals per day for each  
220 arm). Both models were multilevel in that they included nested random intercepts for both study and arm  
221 within study. In addition, and in deviation from our pre-registration, we also included lab as a random  
222 intercept as in some cases we had multiple studies from the same lab or research group. Lastly, the inclusion  
223 of a random intercept for each effect size was accidentally omitted from our pre-registration, and so this is  
224 also included in the model.

225 **Mean REE Model**

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

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

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

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

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

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

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

227 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  
 228 and  $\sigma_{ijkl}^2$  is the corresponding sampling error for that estimate. The random intercepts for the  $i$ th lab,  $j$ th  
 229 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  
 230 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  
 231 the fixed effect estimate of REE for control conditions and  $\beta_1$  the fixed effect estimate for the offset from  
 232 this for the PCOS conditions (i.e., the difference between conditions). The estimated offset was allowed to  
 233 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  
 234 also modelled as correlated with the corresponding random intercepts with covariance  $\Sigma_{\text{lab}}$  and  $\Sigma_{\text{study}}$ , and  
 235  $\text{corr}_{\text{lab}}$  and  $\text{corr}_{\text{study}}$  correlation matrices.

236 The priors for this model were as follows:

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

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

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

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

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

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

237 where the prior for  $\beta_0$ , which corresponded to the model intercept and mean REE in the control condition,  
238 was set based on meta-analysis of the mean REEs for women from two large studies of healthy people<sup>52,53</sup>  
239 though set with a conservative degrees of freedom for the Student-*t* distribution. The random intercept  $\sigma_{0,\text{study}}^2$   
240 was set similarly to this. The prior for the fixed effect  $\beta_1$ , reflecting the difference between control and PCOS  
241 conditions was set based on a wide range of possible values considering the minimum and maximum  
242 values of the ranges reported in the two studies noted (i.e., 2492 - 908 = 1584). We then set a prior that  
243 permits values approximately across this range of values with the majority of it's mass centred around zero.  
244 The remaining random effects were set based on the default weakly regularising priors for `brms` and scaled  
245 to the expected response values using a half-student-*t* distribution with 3 degrees of freedom and  $\mu = 0$ , and  
246 both correlation matrices  $\text{corr}_{\text{lab}}$  and  $\text{corr}_{\text{study}}$  were set with an LKJcorr(1) distribution.

#### 247 Standard Deviation of REE Model

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

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

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

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

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

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

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

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

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

where  $\log(\hat{\theta}_{ijkl})$  is the  $l$ th natural logarithm of the standard deviation of REE estimate from the  $k$ th arm,  
 for the  $j$ th study, conducted by the  $i$ th lab and  $\sigma_{ijkl}^2$  is the corresponding sampling error for that estimate.  
 The random intercepts for the  $i$ th lab,  $j$ th study,  $k$ th arm, and  $l$ th standard deviation of 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 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 the fixed effect estimate of standard deviation of REE  
 for control conditions and  $\beta_1$  the fixed effect estimate for the offset from this for the PCOS conditions (i.e.,  
 the difference between conditions). The estimated offset was allowed to 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 also modelled as correlated with  
 the corresponding random intercepts with covariance  $\Sigma_{\text{lab}}$  and  $\Sigma_{\text{study}}$ , and  $\text{corr}_{\text{lab}}$  and  $\text{corr}_{\text{study}}$  correlation  
 matrices. Finally,  $\beta_2$  represents the fixed effect of the natural logarithm of the corresponding mean REE  
 estimate  $\tilde{m}$  which is modelled as estimated with measurement error i.e.,  $m$  represents the point estimate for  
 the  $l$ th natural logarithm of the mean REE estimate from the  $k$ th arm, for the  $j$ th study, conducted by the  
 $i$ th lab and  $\sigma_{\log(y_{i,\text{mean},[\text{ijkl}]})}^2$  is the corresponding sampling error for that estimate.

263 The priors for this model were as follows:

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

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

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

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

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

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

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

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

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

264 where the prior for  $\beta_0$ , which corresponded to the model intercept and standard deviation of REE in the  
265 control condition, was again set based on meta-analysis of the standard deviation of REEs for women from  
266 two large studies of healthy people<sup>52,53</sup> though set with a conservative degrees of freedom for the Student-*t*  
267 distribution. The random intercept  $\sigma_{0,\text{study}}^2$  was set similarly to this. The prior for the fixed effect  $\beta_1$ , reflecting  
268 the difference between control and PCOS conditions was set based on a wide range of possible values. Given  
269 that in many cases of variables in the field there is an approximate relationship of ~1 for the natural logarithm  
270 of the standard deviation conditioned upon the natural logarithm of the mean<sup>54</sup> we set this prior to reflect  
271 the range of differences on the on the log scale (i.e.,  $\log(1584)$ ). We then set a prior that permits values  
272 approximately across this range of values with the majority of it's mass centred around zero. The prior  
273 for the fixed effect  $\beta_2$ , reflecting the relationship between the natural logarithm of the mean REE with the  
274 natural logarithm of the standard deviation of REE, was set it to be weakly informative centred on zero  
275 with a wide scale to indicate uncertainty in this outcome specifically despite the typical relationship close  
276 to ~1. Priors for the measurement error of the natural logarithm of the mean REE estimate  $\tilde{m}$  were again  
277 based upon meta-analysis of the aforementioned studies, though measurement error has to be positive, as  
278 does the corresponding standard deviation of this error, so we set these to conservative wide half-student-*t*

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

282 **Post-processing of models**

283 For both models we examined trace plots along with  $\hat{R}$  values to examine whether chains have converged, and  
284 posterior predictive checks for each model to understand the model implied distributions. From each model  
285 we took draws from the posterior distributions for the conditional absolute estimates for each condition (i.e.,  
286 controls and PCOS) by study incorporating random effects, the global grand mean absolute estimates for  
287 each condition ignoring random effects, and the global grand mean between condition relative contrast for  
288 controls vs PCOS conditions ignoring random effects. The between condition relative contrast for controls  
289 vs PCOS conditions corresponded to  $\beta_1$  in each model and was our primary estimand of interest; for the  
290 mean REE model this corresponded to the absolute difference in mean REE, and for the standard deviation  
291 of REE model this corresponded to the natural logarithm of the ratio of standard deviations of REE which  
292 was exponentiated (note, all log standard deviation of REE model estimates were exponentiated back to the  
293 original scale to aid interpretability). We present the full probability density functions for posterior visually,  
294 and also to calculate mean and 95% quantile intervals (QI: i.e., ‘credible’ or ‘compatibility’ intervals) for  
295 each estimate providing the most probable value of the parameter in addition to the range from 2.5% to  
296 97.5% percentiles given our priors and data.

297 **Sensitivity analyses**

298 **Pairwise contrast based models**

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

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

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

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

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

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

### 316 Additional sensitivity analyses

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

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

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

## 335 Results

### 336 Systematic Review

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

343 Our systematic review identified 13 studies from 12 lab/research groups including 24 arms (Control arms =  
344 9, PCOS arms = 15) and a total of 918 participants (Controls: minimum n = 9, median n = 29, maximum  
345 n = 54; PCOS: minimum n = 5, median n = 28, maximum n = 266). Descriptive characteristics of the arms  
346 and participants in these studies are reported fully in the online supplementary materials (see [Descriptives](#)

347 [Table](#)).

348 Studies were carried out in multiple countries: Brazil ( $k = 3$ ), and USA ( $k = 3$  studies), Australia, Cameroon,  
349 Canada, Italy, Sweden, Turkey, UK (all  $k = 1$  study). The four studies with noted reporting errors were  
350 carried out in Greece. A total of 11 studies used the Rotterdam criteria (or a modified version thereof) for  
351 diagnosing PCOS<sup>16,17,36,55–62</sup>. One study used the 1990 National Institutes of Health criteria<sup>63</sup>, and two  
352 studies<sup>18,57</sup> diagnosed PCOS via the presence of oligomenorrhea or amenorrhea alongside additional criteria  
353 including plasma androgen levels, hirsutism or polycystic ovaries on ultrasound scanning. All but one study<sup>63</sup>,  
354 which used doubly labelled water, measured REE using indirect calorimetry. The specific devices reported  
355 by these studies are included in the online supplementary materials (see [Descriptives Table](#)). We originally  
356 considered in our pre-registration that, given sufficient data, we would compare sub groups of women with  
357 PCOS who did and did not have accompanying insulin resistance. However, based on the metabolic health  
358 variables reported in studies with this information (see [Descriptives Table](#)) and, where available, considering  
359 primary criteria of either homeostatic model assessment of insulin resistance (HOMA-IR)  $\geq 2.5$  or secondary  
360 criteria including fasting insulin  $> 12\mu\text{U}/\text{mL}$  or fasting glucose  $\geq 100\text{mg}/\text{dL}$ , all groups of women with  
361 PCOS in the included studies would be considered to have insulin resistance. Mean age of women with  
362 PCOS in these studies ranged from 23 to 33 which was similar to the control women without PCOS ranging  
363 from 23 to 30. Across those studies where BMI was reported or it was possible to estimate the mean BMI of  
364 women with PCOS ranged from 26.4 to 39.9 was typically greater than women without PCOS which ranged  
365 20.5 to 27.9.

## 366 Mean REE Model Results

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

<sup>373</sup> visualisation of the posterior distribution and the conditional estimates by study, can be seen in Figure 2.

### <sup>374</sup> Standard Deviation of REE Model Results

<sup>375</sup> The main model for the between participant standard deviation of REE resulted in a posterior distribution  
<sup>376</sup> for the contrast ratio between control and PCOS conditions with a mean point estimate of 0.98 with a 95%  
<sup>377</sup> quantile interval ranging from 0.71 to 1.33 suggesting there is a 95% probability that the true ratio of standard  
<sup>378</sup> deviations lies between these values given our priors and the data from included studies. The corresponding  
<sup>379</sup> conditional estimates for the standard deviations of the control condition and PCOS condition respectively  
<sup>380</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>381</sup> kcal/day]. These results, including the full visualisation of the posterior distribution and the conditional  
<sup>382</sup> estimates by study, can be seen in Figure 3.

### <sup>383</sup> Sensitivity Analyses

#### <sup>384</sup> Pairwise Models

<sup>385</sup> For mean REE the pairwise model resulted in qualitatively similar inferences suggesting little difference  
<sup>386</sup> between control and PCOS conditions with mean point estimate of 16 kcal/day with a 95% quantile interval  
<sup>387</sup> ranging from -36 kcal/day to 70 kcal/day. This was similar for the standard deviation of REE with the  
<sup>388</sup> pairwise model resulting in a contrast ratio between the control and PCOS conditions with a mean point  
<sup>389</sup> estimate of 0.9 and a 95% quantile interval ranging from 0.6 to 1.35.

#### <sup>390</sup> Models including studies with possible reporting issues

<sup>391</sup> For mean REE the model including the studies noted with possible reporting issues that were not  
<sup>392</sup> resolved<sup>13,37-39</sup> still resulted in qualitatively similar inferences suggesting little difference between control  
<sup>393</sup> and PCOS conditions with mean point estimate of 15 kcal/day with a 95% quantile interval ranging from  
<sup>394</sup> -69 kcal/day to 95 kcal/day. This was similar for the standard deviation of REE with the model including  
<sup>395</sup> these studies resulting in a contrast ratio between the control and PCOS conditions with a mean point  
<sup>396</sup> estimate of 1.21 and a 95% quantile interval ranging from 0.68 to 2.13.

<sup>397</sup> **Baseline REE Measurement Models**

<sup>398</sup> For mean REE the model which included only baseline REE measurements from studies involving  
<sup>399</sup> interventions<sup>36,55,56</sup>, in addition to other cross-sectional studies, also resulted in qualitatively similar  
<sup>400</sup> inferences suggesting little difference between control and PCOS conditions with mean point estimate of 32  
<sup>401</sup> kcal/day with a 95% quantile interval ranging from -46 kcal/day to 118 kcal/day. This was also the case for  
<sup>402</sup> the standard deviation of REE with this model resulting in a contrast ratio between the control and PCOS  
<sup>403</sup> conditions with a mean point estimate of 0.97 and a 95% quantile interval ranging from 0.71 to 1.36.

<sup>404</sup> **BMI and Fat-Free Mass Adjusted Models**

<sup>405</sup> For mean REE the model adjusted for BMI, also resulted in qualitatively similar inferences suggesting little  
<sup>406</sup> difference between control and PCOS conditions with mean point estimate of 20 kcal/day with a 95% quantile  
<sup>407</sup> interval ranging from -96 kcal/day to 140 kcal/day. This was also the case for the standard deviation of  
<sup>408</sup> REE with this model resulting in a contrast ratio between the control and PCOS conditions with a mean  
<sup>409</sup> point estimate of 0.91 and a 95% quantile interval ranging from 0.56 to 1.41. This was similar for fat-free  
<sup>410</sup> mass adjusted models too showing little difference in mean REE between control and PCOS conditions with  
<sup>411</sup> mean point estimate of -9 kcal/day with a 95% quantile interval ranging from -195 kcal/day to 168 kcal/day  
<sup>412</sup> and a contrast ratio between the control and PCOS conditions with a mean point estimate of 0.79 and a  
<sup>413</sup> 95% quantile interval ranging from 0.31 to 1.94.

<sup>414</sup> **Discussion**

<sup>415</sup> This study sought to estimate and describe the magnitude of difference in REE between women with and  
<sup>416</sup> without PCOS. Most studies identified in the systematic review, and included in the meta-analysis, used  
<sup>417</sup> indirect calorimetry as the primary measure of REE and assessed women with PCOS who were insulin  
<sup>418</sup> resistant and categorised as being in overweight or obese BMI categories compared to healthy controls. Our  
<sup>419</sup> results indicate there is only a small magnitude of difference in REE (30 kcal/day [95%QI: -47 kcal/day to  
<sup>420</sup> 113kcal/day]) between women with PCOS and those without. Further, there is little difference in between  
<sup>421</sup> person variation between the groups based on the ratio of standard deviations (0.98 [95%QI: 0.71 to 1.33])

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

424 These findings challenge the widely held belief that PCOS is inherently associated with a slower metabolism<sup>26</sup>,  
425 predisposing women with PCOS to weight gain. This belief largely stems from a single but influential 2009  
426 study from Georgopoulos et al. that reported a significantly reduced BMR in women with PCOS<sup>13</sup>, which  
427 has been widely cited and reinforced in both academic and clinical contexts. However, as we have noted, this  
428 study along with others<sup>37-39</sup> have numerous reporting errors which led us to drop them from our present  
429 analysis (though sensitivity analysis including them did not alter our overall conclusions). The mistaken belief  
430 that REE is lower for those with PCOS may have mistakenly lead to recommendations centred on slightly  
431 more severe calorie restriction to achieve weight-loss goals, compared with recommendations for the general  
432 population, as primary management strategies for women with PCOS<sup>20</sup>. Recognising that there may be  
433 minimal differences in REE between women with PCOS and those without can inform both clinical and public  
434 practices, potentially leading to a shift in focus away from a requirement for more severe caloric restriction  
435 as a primary method of treatment towards more comprehensive, individualised, and psychologically safe  
436 approaches to care<sup>25,26</sup>.

437 The current study suggests that REE may not a barrier to weight regulation in PCOS given small group-  
438 level differences between women with PCOS and those without (1472 kcal/day [95%QI:1359 kcal/day to 1587  
439 kcal/day] versus 1442 kcal/day [95%QI:1334 kcal/day to 1553 kcal/day], respectively). If anything, REE  
440 may be slightly higher in women with PCOS compared to healthy women without PCOS. BMI of women  
441 with PCOS ranged from 26.4 to 39.9 was typically greater than women without PCOS which ranged 20.5 to  
442 27.9 and this may explain the slightly greater REE in the former group. However, for those studies where  
443 we could extract or estimate BMI, our additional exploratory models adjusted for this similarly showed  
444 little difference in REE (20 kcal/day [95%QI: -96 kcal/day to 140kcal/day]) between women with PCOS  
445 and those without. However, one of the studies included in our analysis<sup>64</sup> reported that, whilst there was  
446 little difference in unadjusted REE, when REE was adjusted for fat-free mass it was lower in women with  
447 PCOS suggesting the potential importance of fat-free tissue in energy regulation. Yet, for those studies  
448 where fat-free mass was reported, our additional exploratory models adjusted for this similarly showed little

449 difference in REE (-9 kcal/day [95%QI: -195 kcal/day to 168kcal/day]) between women with PCOS and  
450 those without. As such, even adjusted for both BMI and fat-free mass, there seems to be little difference in  
451 REE between women with, and without, PCOS. Yet, a recent systematic review of mechanisms for metabolic  
452 dysfunction has reported excess androgen drives metabolic issues within adipose tissue and muscle tissue  
453 contributing to complications like obesity and insulin resistance<sup>65</sup>. Taken together, these findings highlight  
454 that factors beyond REE and typical correlates of this including BMI or fat-free mass, such as hormonal and  
455 tissue-specific metabolic effects, may play a more significant role in weight regulation challenges in women  
456 with PCOS.

457 As noted, women with PCOS are more likely to engage in weight-loss attempts<sup>19</sup> and there could be con-  
458 cerns that this could inadvertently further foster the already well documented disordered eating in this  
459 population<sup>21,22,66,67</sup>. The pathways linking PCOS and disordered eating are multifactorial. Biological mech-  
460 anisms such as hyperandrogenism, hyperinsulinaemia, and altered ghrelin and leptin signalling can heighten  
461 hunger, carbohydrate cravings, and appetite variability<sup>68</sup>. Frequent hypoglycaemia and associated mood  
462 changes have also been observed, which can trigger compensatory eating or binge episodes<sup>68</sup>. These physi-  
463 ological processes interact with psychological and social stressors, including infertility concerns, conflicting  
464 nutrition advice, chronic dieting, and exposure to idealised body images on social media, which together  
465 compound vulnerability to disordered eating<sup>69</sup>. Moreover, eating disorders themselves can disrupt endocrine  
466 function, potentially worsening PCOS symptoms and creating a self-reinforcing cycle<sup>67,70</sup>.

467 Understanding the intertwined biological, psychological, and social influences suggests the importance of  
468 considering whether restrictive dietary advice is appropriate given it may exacerbate feelings of failure,  
469 hunger dysregulation, and shame<sup>71</sup>. These concerns, coupled with the lack of difference in REE between  
470 women with and without PCOS might suggest that energy restriction based dietary interventions for weight-  
471 loss may be unnecessary. But, there is also evidence supporting the effect of energy restricted dietary  
472 interventions for improving PCOS symptoms<sup>23,24</sup> and they are recommended in international guidelines  
473 [moranEvidenceSummariesRecommendations2020;<sup>26</sup>]. Encouragingly though, these guidelines also recognise  
474 weight stigma as a determinant of health and call for its reduction across clinical and public health settings.  
475 Evidently the greater prevalence of women with PCOS falling into overweight and obese BMI categories

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

## 480 Strengths and Limitations

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

<sup>499</sup> **Conclusion**

<sup>500</sup> In conclusion, the findings from this meta-analysis indicate that REE does not meaningfully differ between  
<sup>501</sup> women with and without PCOS. Group-level differences in REE were small, insignificant, or not physiologi-  
<sup>502</sup> cally relevant. Additionally, variability in REE between individuals was also similar. These results suggest  
<sup>503</sup> that a lower baseline REE is not associated with the weight-related challenges often associated with PCOS.  
<sup>504</sup> These findings challenge the popular narrative that women with PCOS have a lower REE and may help  
<sup>505</sup> better inform dietary interventions and nutritional support for these individuals. Future research should  
<sup>506</sup> include more standardized REE measurement and reporting protocols, greater data transparency, consistent  
<sup>507</sup> control and reporting of body weight or body composition, the presentation of both absolute and relative  
<sup>508</sup> REE, and more precise characterization of PCOS phenotypes. Overall, these findings support the conclu-  
<sup>509</sup> sion that PCOS is not negatively associated with REE and may help practitioners and researchers focus on  
<sup>510</sup> individually targeted and holistic lifestyle interventions rather than negatively framed interventions based  
<sup>511</sup> on unsupported assumptions regarding REE.

<sup>512</sup> **Data Availability**

<sup>513</sup> All code utilised for data preparation, transformations, analyses, plotting, and reporting are available in the  
<sup>514</sup> corresponding GitHub repository [https://github.com/jamessteeleii/pcos\\_ree\\_meta](https://github.com/jamessteeleii/pcos_ree_meta).

<sup>515</sup> **Contributions**

<sup>516</sup> Gregory Nuckols and Leigh Peele conceived the idea for the project. All authors contributed to the design  
<sup>517</sup> of the project and methods. Richie Kirwan and Leigh Peele conducted the systematic search and screening.  
<sup>518</sup> James Steele performed the data extraction, conducted the statistical analyses, and produced the data  
<sup>519</sup> visualisations. All authors contributed to drafting the initial manuscript. All authors contributed to editing  
<sup>520</sup> the manuscript. All authors read and approved the final manuscript.

521    **References**

- 522    1. Salari N, Nankali A, Ghanbari A, et al. Global prevalence of polycystic ovary syndrome in women worldwide: A comprehensive systematic review and meta-analysis. *Archives of Gynecology and Obstetrics*. 2024;310(3):1303-1314. doi:[10.1007/s00404-024-07607-x](https://doi.org/10.1007/s00404-024-07607-x)
- 523    2. Glintborg D, Rubin KH, Nybo M, Abrahamsen B, Andersen M. Cardiovascular disease in a nationwide population of Danish women with polycystic ovary syndrome. *Cardiovascular Diabetology*. 2018;17(1):37. doi:[10.1186/s12933-018-0680-5](https://doi.org/10.1186/s12933-018-0680-5)
- 524    3. Glintborg D, Kolster ND, Ravn P, Andersen MS. Prospective Risk of Type 2 Diabetes in Normal Weight Women with Polycystic Ovary Syndrome. *Biomedicines*. 2022;10(6):1455. doi:[10.3390/biomedicines10061455](https://doi.org/10.3390/biomedicines10061455)
- 525    4. Persson S, Elenis E, Turkmen S, Kramer MS, Yong EL, Poromaa IS. Higher risk of type 2 diabetes in women with hyperandrogenic polycystic ovary syndrome. *Fertility and Sterility*. 2021;116(3):862-871. doi:[10.1016/j.fertnstert.2021.04.018](https://doi.org/10.1016/j.fertnstert.2021.04.018)
- 526    5. Glintborg D, Ollila MM, Møller JJK, et al. Prospective risk of Type 2 diabetes in 99 892 Nordic women with polycystic ovary syndrome and 446 055 controls: National cohort study from Denmark, Finland, and Sweden. *Human Reproduction (Oxford, England)*. 2024;39(8):1823-1834. doi:[10.1093/humrep/deae124](https://doi.org/10.1093/humrep/deae124)
- 527    6. Lim SS, Kakoly NS, Tan JWJ, et al. Metabolic syndrome in polycystic ovary syndrome: A systematic review, meta-analysis and meta-regression. *Obesity Reviews: An Official Journal of the International Association for the Study of Obesity*. 2019;20(2):339-352. doi:[10.1111/obr.12762](https://doi.org/10.1111/obr.12762)

- 528 7. Joham AE, Teede HJ, Ranasinha S, Zoungas S, Boyle J. Prevalence of infertility and use of fertility treatment in women with polycystic ovary syndrome: Data from a large community-based cohort study. *Journal of Women's Health (2002)*. 2015;24(4):299-307. doi:[10.1089/jwh.2014.5000](https://doi.org/10.1089/jwh.2014.5000)
- 529 8. Barber TM. Why are women with polycystic ovary syndrome obese? *British Medical Bulletin*. 2022;143(1):4-15. doi:[10.1093/bmb/ldac007](https://doi.org/10.1093/bmb/ldac007)
- 530 9. Barber TM, Franks S. Obesity and polycystic ovary syndrome. *Clinical Endocrinology*. 2021;95(4):531-541. doi:[10.1111/cen.14421](https://doi.org/10.1111/cen.14421)
- 531 10. Hirschberg AL, Naessén S, Stridsberg M, Byström B, Holtet J. Impaired cholecystokinin secretion and disturbed appetite regulation in women with polycystic ovary syndrome. *Gynecological Endocrinology: The Official Journal of the International Society of Gynecological Endocrinology*. 2004;19(2):79-87. doi:[10.1080/09513590400002300](https://doi.org/10.1080/09513590400002300)
- 532 11. Moran LJ, Noakes M, Clifton PM, et al. Ghrelin and measures of satiety are altered in polycystic ovary syndrome but not differentially affected by diet composition. *The Journal of Clinical Endocrinology and Metabolism*. 2004;89(7):3337-3344. doi:[10.1210/jc.2003-031583](https://doi.org/10.1210/jc.2003-031583)
- 533 12. Stefanaki K, Karagiannakis DS, Peppa M, et al. Food Cravings and Obesity in Women with Polycystic Ovary Syndrome: Pathophysiological and Therapeutic Considerations. *Nutrients*. 2024;16(7):1049. doi:[10.3390/nu16071049](https://doi.org/10.3390/nu16071049)
- 534 13. Georgopoulos NA, Saltamavros AD, Vervita V, et al. Basal metabolic rate is decreased in women with polycystic ovary syndrome and biochemical hyperandrogenemia and is associated with insulin resistance. *Fertility and Sterility*. 2009;92(1):250-255. doi:[10.1016/j.fertnstert.2008.04.067](https://doi.org/10.1016/j.fertnstert.2008.04.067)

- 535 14. Romualdi D, Versace V, Tagliaferri V, et al. The resting metabolic rate in women with polycystic ovary syndrome and its relation to the hormonal milieu, insulin metabolism, and body fat distribution: A cohort study. *Journal of Endocrinological Investigation*. 2019;42(9):1089-1097. doi:[10.1007/s40618-019-01029-2](https://doi.org/10.1007/s40618-019-01029-2)
- 536 15. Churchill SJ, Wang ET, Bhushan G, et al. Basal metabolic rate in women with PCOS compared to eumenorrheic controls. *Clinical Endocrinology*. 2015;83(3):384-388. doi:[10.1111/cen.12740](https://doi.org/10.1111/cen.12740)
- 537 16. Graff SK, Mario FM, Magalhães JA, Moraes RS, Spritzer PM. Saturated Fat Intake Is Related to Heart Rate Variability in Women with Polycystic Ovary Syndrome. *Annals of Nutrition & Metabolism*. 2017;71(3-4):224-233. doi:[10.1159/000484325](https://doi.org/10.1159/000484325)
- 538 17. Larsson I, Hulthén L, Landén M, Pålsson E, Janson P, Stener-Victorin E. Dietary intake, resting energy expenditure, and eating behavior in women with and without polycystic ovary syndrome. *Clinical Nutrition (Edinburgh, Scotland)*. 2016;35(1):213-218. doi:[10.1016/j.clnu.2015.02.006](https://doi.org/10.1016/j.clnu.2015.02.006)
- 539 18. Segal KR, Dunaif A. Resting metabolic rate and postprandial thermogenesis in polycystic ovarian syndrome. *International Journal of Obesity*. 1990;14(7):559-567.
- 540 19. Pesonen E, Nurkkala M, Niemelä M, et al. Polycystic ovary syndrome is associated with weight-loss attempts and perception of overweight independent of BMI: A population-based cohort study. *Obesity (Silver Spring, Md)*. 2023;31(4):1108-1120. doi:[10.1002/oby.23681](https://doi.org/10.1002/oby.23681)
- 541 20. Ozgen Saydam B, Yildiz BO. Weight management strategies for patients with PCOS: Current perspectives. *Expert Review of Endocrinology & Metabolism*. 2021;16(2):49-62. doi:[10.1080/17446651.2021.1896966](https://doi.org/10.1080/17446651.2021.1896966)

- 542 21. Lalonde-Bester S, Malik M, Masoumi R, et al. Prevalence and Etiology of Eating Disorders in Polycystic Ovary Syndrome: A Scoping Review. *Advances in Nutrition (Bethesda, Md)*. 2024;15(4):100193. doi:[10.1016/j.advnut.2024.100193](https://doi.org/10.1016/j.advnut.2024.100193)
- 543 22. Jeanes YM, Reeves S, Gibson EL, Piggott C, May VA, Hart KH. Binge eating behaviours and food cravings in women with Polycystic Ovary Syndrome. *Appetite*. 2017;109:24-32. doi:[10.1016/j.appet.2016.11.010](https://doi.org/10.1016/j.appet.2016.11.010)
- 544 23. Alenezi SA, Elkmeshi N, Alanazi A, Alanazi ST, Khan R, Amer S. The Impact of Diet-Induced Weight Loss on Inflammatory Status and Hyperandrogenism in Women with Polycystic Ovarian Syndrome (PCOS)-A Systematic Review and Meta-Analysis. *Journal of Clinical Medicine*. 2024;13(16):4934. doi:[10.3390/jcm13164934](https://doi.org/10.3390/jcm13164934)
- 545 24. Holte J, Bergh T, Berne C, Wide L, Lithell H. Restored insulin sensitivity but persistently increased early insulin secretion after weight loss in obese women with polycystic ovary syndrome. *The Journal of Clinical Endocrinology and Metabolism*. 1995;80(9):2586-2593. doi:[10.1210/jcem.80.9.7673399](https://doi.org/10.1210/jcem.80.9.7673399)
- 546 25. Moran LJ, Tassone EC, Boyle J, et al. Evidence summaries and recommendations from the international evidence-based guideline for the assessment and management of polycystic ovary syndrome: Lifestyle management. *Obesity Reviews*. 2020;21(10):e13046. doi:[10.1111/obr.13046](https://doi.org/10.1111/obr.13046)
- 547 26. Teede HJ, Tay CT, Laven JJE, et al. Recommendations From the 2023 International Evidence-based Guideline for the Assessment and Management of Polycystic Ovary Syndrome. *The Journal of Clinical Endocrinology and Metabolism*. 2023;108(10):2447-2469. doi:[10.1210/clinem/dgad463](https://doi.org/10.1210/clinem/dgad463)
- 548 27. Himelein MJ, Thatcher SS. Depression and body image among women with polycystic ovary syndrome. *Journal of Health Psychology*. 2006;11(4):613-625. doi:[10.1177/1359105306065021](https://doi.org/10.1177/1359105306065021)

- 549 28. Hofmann K, Decrinis C, Bitterlich N, Tropschuh K, Stute P, Bachmann A. Body image and mental health in women with polycystic ovary syndrome-a cross-sectional study. *Archives of Gynecology and Obstetrics*. 2025;312(1):177-190. doi:[10.1007/s00404-024-07913-4](https://doi.org/10.1007/s00404-024-07913-4)
- 550 29. Geller S, Levy S, Avitsur R. Body image, illness perception, and psychological distress in women coping with polycystic ovary syndrome (PCOS). *Health Psychology Open*. 2025;12:20551029251327441. doi:[10.1177/20551029251327441](https://doi.org/10.1177/20551029251327441)
- 551 30. Davitadze M, Malhotra K, Khalil H, et al. Body image concerns in women with polycystic ovary syndrome: A systematic review and meta-analysis. *European Journal of Endocrinology*. 2023;189(2):R1-R9. doi:[10.1093/ejendo/lvad110](https://doi.org/10.1093/ejendo/lvad110)
- 552 31. Nguo K, McGowan M, Cowan S, et al. Exploring the physiological factors relating to energy balance in women with polycystic ovary syndrome: A scoping review. *Nutrition Reviews*. 2025;83(1):160-174. doi:[10.1093/nutrit/nuad169](https://doi.org/10.1093/nutrit/nuad169)
- 553 32. Page MJ, McKenzie JE, Bossuyt PM, et al. The PRISMA 2020 statement: An updated guideline for reporting systematic reviews. *BMJ*. 2021;372:n71. doi:[10.1136/bmj.n71](https://doi.org/10.1136/bmj.n71)
- 554 33. Haddaway NR, Page MJ, Pritchard CC, McGuinness LA. PRISMA2020: An R package and Shiny app for producing PRISMA 2020-compliant flow diagrams, with interactivity for optimised digital transparency and Open Synthesis. *Campbell Systematic Reviews*. 2022;18(2):e1230. doi:[10.1002/cds.1230](https://doi.org/10.1002/cds.1230)
- 555 34. Weber S, Li Y, Iii JWS, Kakizume T, Schmidli H. Applying Meta-Analytic-Predictive Priors with the R Bayesian Evidence Synthesis Tools. *Journal of Statistical Software*. 2021;100:1-32. doi:[10.18637/jss.v100.i19](https://doi.org/10.18637/jss.v100.i19)

- 556 35. Wan X, Wang W, Liu J, Tong T. Estimating the sample mean and standard deviation from the sample size, median, range and/or interquartile range. *BMC Medical Research Methodology*. 2014;14(1):135. doi:[10.1186/1471-2288-14-135](https://doi.org/10.1186/1471-2288-14-135)
- 557 36. Pohlmeier AM, Phy JL, Watkins P, et al. Effect of a low-starch/low-dairy diet on fat oxidation in overweight and obese women with polycystic ovary syndrome. *Applied Physiology, Nutrition, and Metabolism = Physiologie Appliquée, Nutrition Et Métabolisme*. 2014;39(11):1237-1244. doi:[10.1139/apnm-2014-0073](https://doi.org/10.1139/apnm-2014-0073)
- 558 37. Kritikou S, Saltamavros AD, Adonakis G, et al. The  $\alpha$ 2B and  $\beta$ 3 adrenergic receptor genes polymorphisms in women with polycystic ovarian syndrome (PCOS) and their association with insulin resistance and basal metabolic rate (BMR). *Review of Clinical Pharmacology and Pharmacokinetics – International Edition*. 2006;2006(2).
- 559 38. Saltamavros AD, Adonakis G, Kritikou S, et al. Alpha 2 beta adrenoreceptor 301-303 deletion polymorphism in polycystic ovary syndrome. *Clinical Autonomic Research: Official Journal of the Clinical Autonomic Research Society*. 2007;17(2):112-114. doi:[10.1007/s10286-007-0403-6](https://doi.org/10.1007/s10286-007-0403-6)
- 560 39. Koika V, Marioli DJ, Saltamavros AD, et al. Association of the Pro12Ala polymorphism in peroxisome proliferator-activated receptor Gamma2 with decreased basic metabolic rate in women with polycystic ovary syndrome. *European Journal of Endocrinology*. 2009;161(2):317-322. doi:[10.1530/EJE-08-1014](https://doi.org/10.1530/EJE-08-1014)
- 561 40. Rodriguez-Sanchez F, cre, cph, Jackson CP, Hutchins SD, Clawson JM. Grateful: Facilitate Citation of R Packages. Published online October 2023.
- 562 41. Cumming G. The New Statistics: Why and How. *Psychological Science*. 2014;25(1):7-29. doi:[10.1177/0956797613504966](https://doi.org/10.1177/0956797613504966)

- 563 42. Kruschke JK, Liddell TM. The Bayesian New Statistics: Hypothesis testing, estimation, meta-analysis, and power analysis from a Bayesian perspective. *Psychonomic Bulletin & Review*. 2018;25:178-206.
- 564 43. Ushey K, cre, Wickham H, Software P, PBC. Renv: Project Environments. Published online September 2023.
- 565 44. Landau WM, Warkentin MT, Edmondson M, Oliver S, Mahr T, Company EL and. Targets: Dynamic Function-Oriented 'Make'-Like Declarative Pipelines. Published online October 2023.
- 566 45. Viechtbauer W. Metafor: Meta-Analysis Package for R. Published online September 2023.
- 567 46. Bürkner PC, Gabry J, Weber S, et al. Brms: Bayesian Regression Models using 'Stan'. Published online September 2023.
- 568 47. Arel-Bundock V, cre, cph, Diniz MA, Greifer N, Bacher E. MarginalEffects: Predictions, Comparisons, Slopes, Marginal Means, and Hypothesis Tests. Published online October 2023.
- 569 48. Kay M, Mastny T. Tidybayes: Tidy Data and 'Geoms' for Bayesian Models. Published online August 2023.
- 570 49. Wickham H, Chang W, Henry L, et al. Ggplot2: Create Elegant Data Visualisations Using the Grammar of Graphics. Published online October 2023.
- 571 50. Pedersen TL. Patchwork: The Composer of Plots. Published online August 2023.

- 572 51. Hong H, Chu H, Zhang J, Carlin BP. A Bayesian missing data framework for generalized  
multiple outcome mixed treatment comparisons. *Research Synthesis Methods*. 2016;7(1):6-22.  
doi:[10.1002/jrsm.1153](https://doi.org/10.1002/jrsm.1153)
- 573 52. Velasquez JR. The Use of Ammonia Inhalants Among Athletes. *Strength & Conditioning Journal*.  
2011;33(2):33. doi:[10.1519/SSC.0b013e3181fd5c9b](https://doi.org/10.1519/SSC.0b013e3181fd5c9b)
- 574 53. Pavlidou E, Papadopoulou SK, Seroglou K, Giagnis C. Revised Harris-Benedict Equation: New  
Human Resting Metabolic Rate Equation. *Metabolites*. 2023;13(2):189. doi:[10.3390/metabo13020189](https://doi.org/10.3390/metabo13020189)
- 575 54. Steele J, Fisher, Smith, Schoenfeld, Yang, and Nakagawa S. Meta-analysis of variation in sport and ex-  
ercise science: Examples of application within resistance training research. *Journal of Sports Sciences*.  
2023;41(17):1617-1634. doi:[10.1080/02640414.2023.2286748](https://doi.org/10.1080/02640414.2023.2286748)
- 576 55. Moran LJ, Noakes M, Clifton PM, Wittert GA, Williams G, Norman RJ. Short-term meal replace-  
ments followed by dietary macronutrient restriction enhance weight loss in polycystic ovary syndrome.  
*The American Journal of Clinical Nutrition*. 2006;84(1):77-87. doi:[10.1093/ajcn/84.1.77](https://doi.org/10.1093/ajcn/84.1.77)
- 577 56. Bruner B, Chad K, Chizen D. Effects of exercise and nutritional counseling in women with polycystic  
ovary syndrome. *Applied Physiology, Nutrition, and Metabolism = Physiologie Appliquée, Nutrition  
Et Métabolisme*. 2006;31(4):384-391. doi:[10.1139/h06-007](https://doi.org/10.1139/h06-007)
- 578 57. Robinson S, Chan SP, Spacey S, Anyaoku V, Johnston DG, Franks S. Postprandial thermogenesis  
is reduced in polycystic ovary syndrome and is associated with increased insulin resistance. *Clinical  
Endocrinology*. 1992;36(6):537-543. doi:[10.1111/j.1365-2265.1992.tb02262.x](https://doi.org/10.1111/j.1365-2265.1992.tb02262.x)

- 579 58. Cosar E, Köken G, Sahin FK, et al. Resting metabolic rate and exercise capacity in women  
with polycystic ovary syndrome. *International Journal of Gynaecology and Obstetrics: The Of-*  
*ficial Organ of the International Federation of Gynaecology and Obstetrics.* 2008;101(1):31-34.  
doi:[10.1016/j.ijgo.2007.10.011](https://doi.org/10.1016/j.ijgo.2007.10.011)
- 580 59. Graff SK, Mário FM, Alves BC, Spritzer PM. Dietary glycemic index is associated with less favorable  
anthropometric and metabolic profiles in polycystic ovary syndrome women with different phenotypes.  
*Fertility and Sterility.* 2013;100(4):1081-1088. doi:[10.1016/j.fertnstert.2013.06.005](https://doi.org/10.1016/j.fertnstert.2013.06.005)
- 581 60. Doh E, Mbanya A, Kemfang-Ngowa JD, et al. The Relationship between Adiposity and Insulin Sensi-  
tivity in African Women Living with the Polycystic Ovarian Syndrome: A Clamp Study. *International*  
*Journal of Endocrinology.* 2016;2016:9201701. doi:[10.1155/2016/9201701](https://doi.org/10.1155/2016/9201701)
- 582 61. Rodrigues AM dos S, Costa ABP, Campos DL, et al. Low validity of predictive equations for cal-  
culating resting energy expenditure in overweight and obese women with polycystic ovary syndrome.  
*Journal of Human Nutrition and Dietetics.* 2018;31(2):266-275. doi:[10.1111/jhn.12498](https://doi.org/10.1111/jhn.12498)
- 583 62. Tosi F, Villani M, Migazzi M, et al. Insulin-Mediated Substrate Use in Women With Different  
Phenotypes of PCOS: The Role of Androgens. *The Journal of Clinical Endocrinology and Metabolism.*  
2021;106(9):e3414-e3425. doi:[10.1210/clinem/dgab380](https://doi.org/10.1210/clinem/dgab380)
- 584 63. Broskey NT, Klempel MC, Gilmore LA, et al. Assessing Energy Requirements in Women With  
Polycystic Ovary Syndrome: A Comparison Against Doubly Labeled Water. *The Journal of Clinical*  
*Endocrinology and Metabolism.* 2017;102(6):1951-1959. doi:[10.1210/jc.2017-00459](https://doi.org/10.1210/jc.2017-00459)
- 585 64. Tosi F, Rosmini F, Gremes V, et al. Resting energy expenditure in women with polycystic ovary syn-  
drome. *Human Reproduction (Oxford, England).* 2024;39(8):1794-1803. doi:[10.1093/humrep/deae129](https://doi.org/10.1093/humrep/deae129)

- 586 65. Sanchez-Garrido MA, Tena-Sempere M. Metabolic dysfunction in polycystic ovary syndrome: Pathogenic role of androgen excess and potential therapeutic strategies. *Molecular Metabolism*. 2020;35:100937. doi:[10.1016/j.molmet.2020.01.001](https://doi.org/10.1016/j.molmet.2020.01.001)
- 587 66. Lee I, Cooney LG, Saini S, et al. Increased risk of disordered eating in polycystic ovary syndrome. *Fertility and Sterility*. 2017;107(3):796-802. doi:[10.1016/j.fertnstert.2016.12.014](https://doi.org/10.1016/j.fertnstert.2016.12.014)
- 588 67. Bernadett M, Szemán-N A. [Prevalence of eating disorders among women with polycystic ovary syndrome]. *Psychiatria Hungarica: A Magyar Pszichiatriai Tarsaság Tudományos Folyoirata*. 2016;31(2):136-145.
- 589 68. Barry JA, Bouloux P, Hardiman PJ. The impact of eating behavior on psychological symptoms typical of reactive hypoglycemia. A pilot study comparing women with polycystic ovary syndrome to controls. *Appetite*. 2011;57(1):73-76. doi:[10.1016/j.appet.2011.03.003](https://doi.org/10.1016/j.appet.2011.03.003)
- 590 69. Steegers-Theunissen RPM, Wiegel RE, Jansen PW, Laven JSE, Sinclair KD. Polycystic Ovary Syndrome: A Brain Disorder Characterized by Eating Problems Originating during Puberty and Adolescence. *International Journal of Molecular Sciences*. 2020;21(21):8211. doi:[10.3390/ijms21218211](https://doi.org/10.3390/ijms21218211)
- 591 70. Cooney LG, Gyorfi K, Sanneh A, et al. Increased Prevalence of Binge Eating Disorder and Bulimia Nervosa in Women With Polycystic Ovary Syndrome: A Systematic Review and Meta-Analysis. *The Journal of Clinical Endocrinology and Metabolism*. 2024;109(12):3293-3305. doi:[10.1210/clinem/dgae462](https://doi.org/10.1210/clinem/dgae462)
- 592 71. Pirotta S, Barillaro M, Brennan L, et al. Disordered Eating Behaviours and Eating Disorders in Women in Australia with and without Polycystic Ovary Syndrome: A Cross-Sectional Study. *Journal of Clinical Medicine*. 2019;8(10):1682. doi:[10.3390/jcm8101682](https://doi.org/10.3390/jcm8101682)

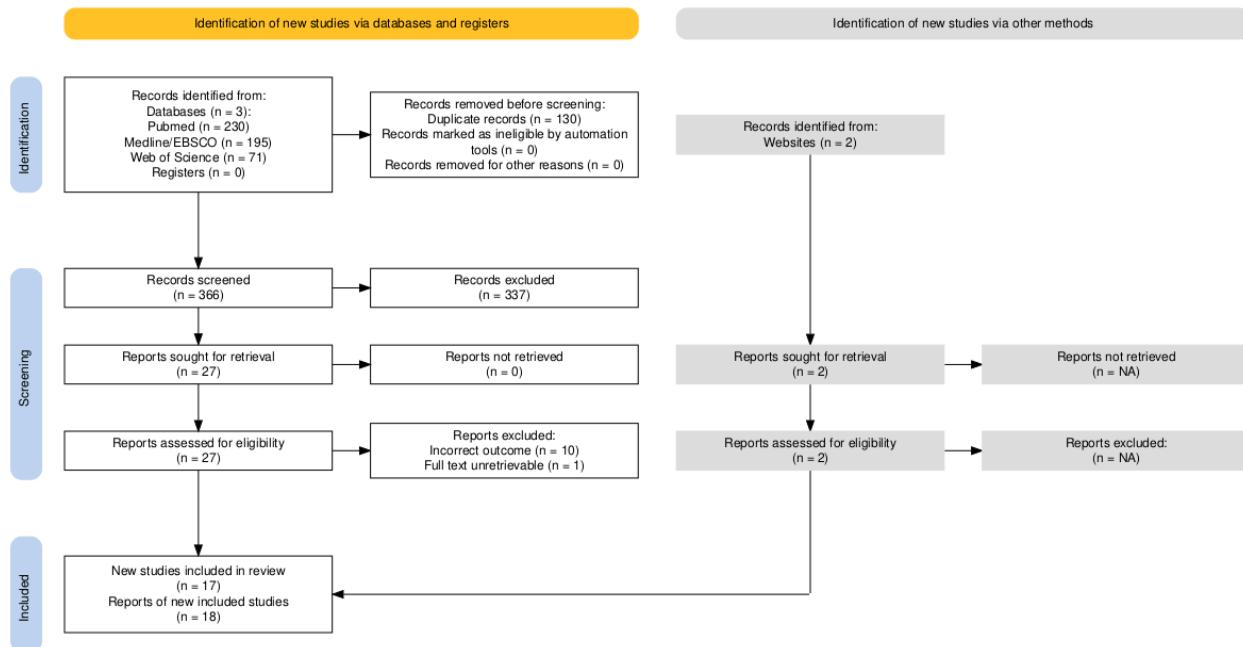


Figure 1: PRISMA 2020 flow diagram template for systematic reviews. Note that a “report” could be a journal article, preprint, conference abstract, study register entry, clinical study report, dissertation, unpublished manuscript, government report or any other document providing relevant information.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

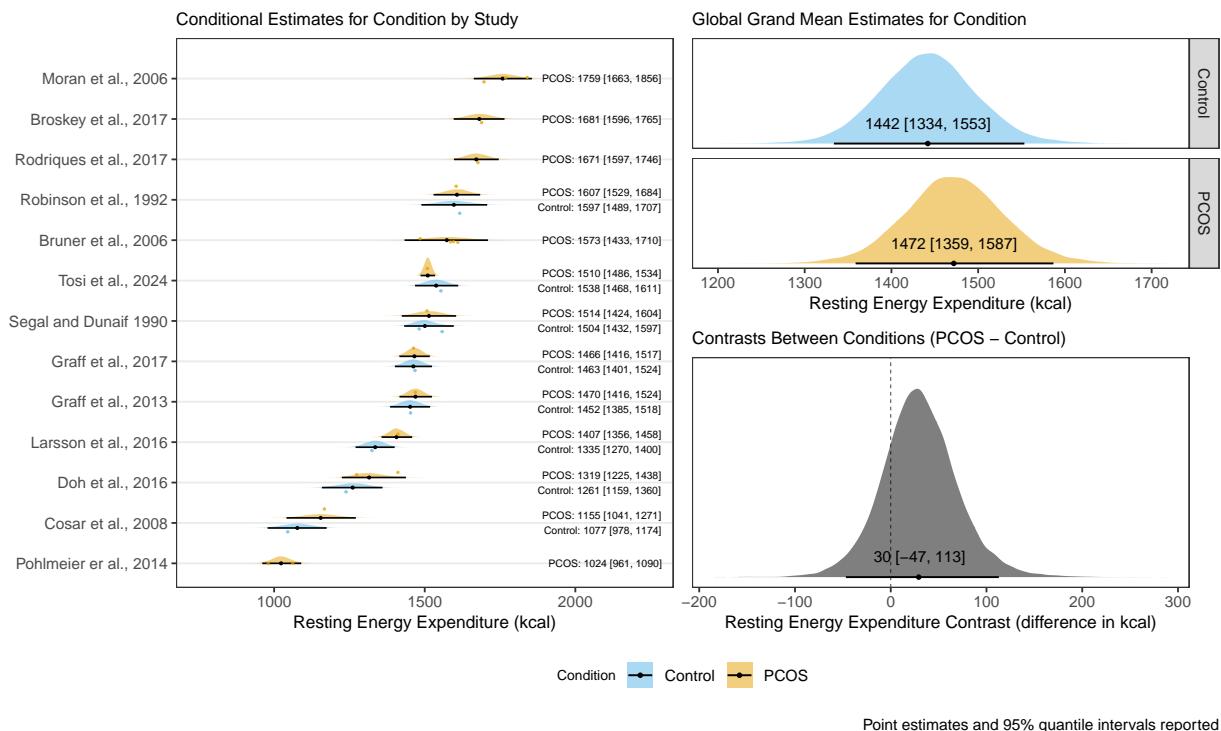


Figure 2: Posterior distribution, mean point estimates, and 95% quantile intervals for conditional estimates by study, global grand mean estimates by condition, and the contrast between conditions for mean resting energy expenditure of control women without PCOS and women with PCOS.

### Standard Deviation of Resting Energy Expenditure

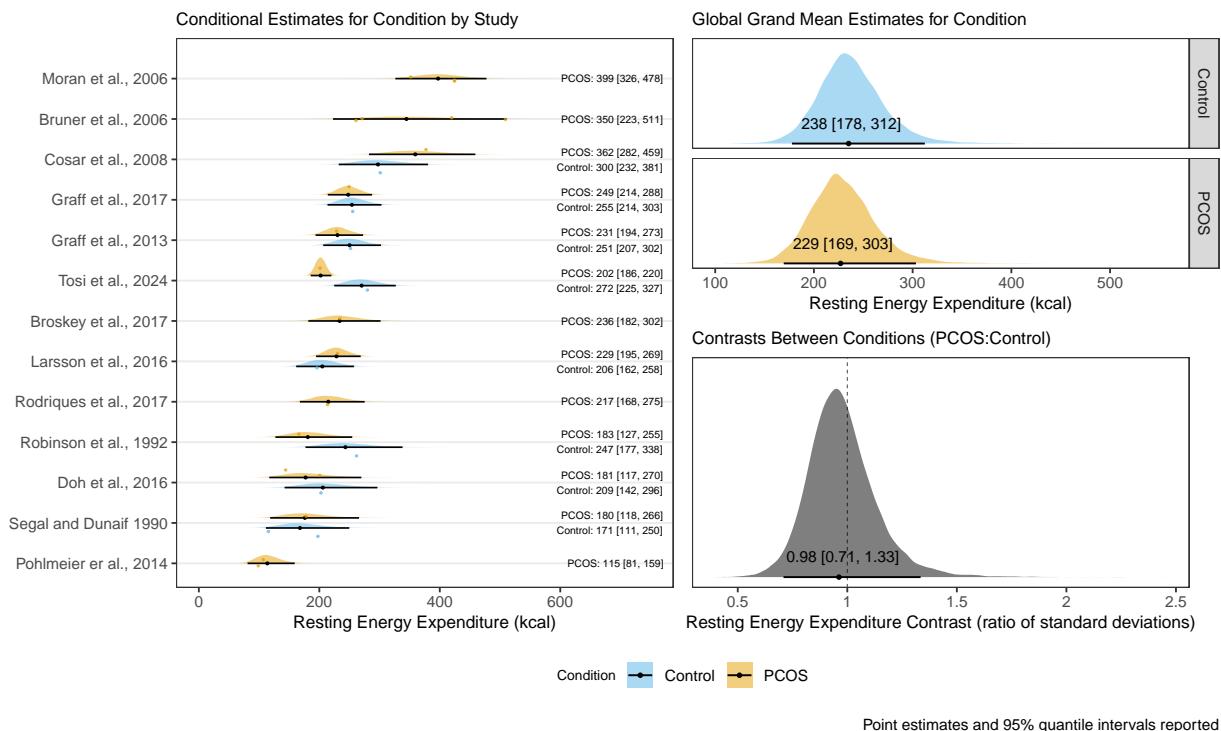


Figure 3: Posterior distribution, variance point estimates, and 95% quantile intervals for conditional estimates by study, global grand variance estimates by condition, and the contrast ratio between conditions for the standard deviation of resting energy expenditure of control women without PCOS and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