Does resting energy expenditure differ between women with and without polycystic ovary syndrome (PCOS): a systematic review and meta-analysis

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Background: Polycystic ovary syndrome (PCOS) is the most common endocrine condition affecting women of reproductive age. Women who are living with PCOS are also more likely to experience overwweight and obesity and this is assumed to be a result of a lower resting energy expenditure (REE). The assumption that REE is lower in women living with PCOS has also influenced lifestyle-based interventions in this group. Evidence supporting this reduced REE compared to women without PCOS, however, is not consistent. Objectives: To systematically search and meta-analyse the existing literature to determine if resting energy expenditure differs between women with and without PCOS. Design: A systematic search was conducted using PubMed, Medline and Web of Science databases of published research from January 1990 to January 2025. Studies that measured REE in women living with PCOS, both with and without control arms of women without PCOS, were included. Results: Seventeen independent studies were identified. Study populations ranged from X to X participants, with mean ages ranging from X to X years. Conclusions: TO ADD

Introduction

Polycystic ovary syndrome (PCOS) affects approximately 10% of women of reproductive age worldwide, making it the most common endocrine disorder affecting this population (Salari et al., 2024). Due to a number of factors including hyperandrogenism and alterations in insulin resistance, PCOS is believed to contribute to an increased risk of diabetes, metabolic syndrome and cardiovascular disease (Glintborg et al., 2018, 2022, 2024; Lim et al., 2019; Persson et al.,

2021), along with being a leading cause of anovulatory infertility in women (Joham et al., 2015). Furthermore, epidemiological data consistently demonstrates that women with PCOS are significantly more likely to suffer from overweight or obesity, compared to the general female population, with estimates ranging from 38% to 88% of PCOS patients falling into overweight or obese body mass index (BMI) categories (Barber, 2022; Barber & Franks, 2021).

The elevated incidence of overweight and obesity in PCOS is likely multifactorial with proposed mechanisms including blunted postprandial appetite hormone responses leading to reduced satiety and increased food cravings (Hirschberg et al., 2004; Moran et al., 2004; Stefanaki et al., 2024) and a reduced resting energy expenditure (REE) (Georgopoulos et al., 2009). Indeed, the study from Georgopoulos et al. (2009) examining REE using indirect calorimetry in women with PCOS reported that they had a significantly lower REE, ~400 kcal per day less, compared with women without PCOS. Notably, they also reported insulin resistance further influenced REE among women with PCOS, with those who were insulin resistant exhibiting an additional reduction of about ~500 kcal per day compared to women with PCOS who were not insulin resistant (Georgopoulos et al., 2009). Other studies similarly report lower BMR using indirect methods (such as prediction from bioelectrical impedance analysis or accelerometer physical activity data) in women with PCOS, also highlighting factors like insulin resistance or BMI category influence REE [Romualdi et al. (2019); churchillBasalMetabolicRate2015]. However, despite the widespread acceptance that women living with PCOS exhibit reduced REE based on studies such as these, other research has reported little to no difference in REE between women with and without PCOS (Graff et al., 2017; Larsson et al., 2016; Segal & Dunaif, 1990).

The consequences of widespread acceptance that REE is reduced in women living with PCOS should not be underestimated, particularly in light of the aforementioned incidence of overweight and obesity in this population. From a physiological perspective, if women with PCOS do exhibit a lower REE, this could imply a meaningful metabolic disadvantage that may influence dietary and nutritional guidance for weight management. However, if no such difference exists, the belief in a "slower metabolism" could instead serve as a deterrent to weight-loss efforts—efforts that are recognised as efficacious for improving PCOS symptoms (Alenezi et al., 2024; Holte et al., 1995) and are routinely recommended (Teede et al., 2023). It has been well documented that women with PCOS already experience higher rates of anxiety, depression, and lower quality of life (QOL) as a result of negative body image and weight-related concerns (Davitadze et al., 2023; Geller et al., 2025; Himelein & Thatcher, 2006; Hofmann et al., 2025). Consequently, they are often observed to engage in more frequent weight-loss attempts than women without PCOS (Pesonen et al., 2023). Clarifying the relationship between REE and PCOS may therefore help guide more accurate clinical recommendations and empower both practitioners and women with PCOS.

Therefore, to estimate the magnitude of difference in REE between women with and without PCOS, we completed a systematic review and meta-analysis of studies reporting REE in these populations.

Methods

This systematic review and meta-analysis was pre-registered on PROSPERO (CRD42024601434) initially on the 3^{rd} of December 2024 and performed in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) statement guidelines (Page et al., 2021). The primary aim of this review was to examine the descriptive question "Does resting energy expenditure (REE) differ between women with and without polycystic ovary syndrome (PCOS)?". We summarise and describe the studies in addition to quantitatively synthesising their results via meta-analysis.

Search Strategy

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

Eligibility Criteria

Studies were included in the systematic review if 1) participants were confirmed as women with PCOS between the ages of 18 to 65 years of age with or without insulin resistance; 2) otherwise healthy (e.g., non-diabetic, no cardiovascular disease); 3) had a measure of REE measured via multiple methods including direct/indirect calorimetry, doubly labelled water; and 4) trials were not retracted at the time of this analysis. Studies were excluded if they 1) used invalid or non-standard methods for measuring REE (e.g., predicted REE from body composition or accelerometer data); 2) non-peer-reviewed journal articles (including grey literature sources such as conference abstracts, theses and dissertations); and 3) were secondary analyses with the same primary outcome data as another included study.

The condition being studied was PCOS and we included observational cross-sectional design studies, in addition to studies of intervention where REE was reported for the population (and if present, the comparator i.e., women without PCOS) condition of interest. For clarity, studies

of any design were included if they reported the REE using the methods indicated for a sample of adult women with PCOS and who were otherwise healthy. This included both studies with and without samples of healthy control women without PCOS. As detailed in the statistical analysis section below, a Bayesian model with informative priors based on normative data for REE in health women without PCOS was included to provide control information indirectly where this was missing. The use of such priors is an efficient tool to incorporate historical information on a particular population in a conservative manner (Weber et al., 2021).

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

- Population
  - Inclusion criteria:
    - Women
    - 18-65 y
    - With or without insulin resistance (IR)
- Intervention(s) or exposure(s)
  - Otherwise healthy women with PCOS
- Comparator(s) or control(s)
  - Otherwise healthy control women without PCOS
- Outcome
- Inclusion criteria
  - Resting energy expenditure (REE) measured via multiple methods including direct/indirect calorimetry, doubly labelled water.
- Exclusion criteria:
  - Studies using invalid or non-standard methods for measuring REE (e.g., predicted REE from body composition or accelerometer data)

Data extraction (selection and coding)

Bibliometric data including authors, journal, and title of article were extracted. Descriptive statistics for age, body mass, fat mass, fat free mass, height, BMI, race, physical activity levels, country of investigation, information regarding glucose/insulin regulation and insulin resistance status (where available), diagnostic criteria for PCOS, and measurement method and device were extracted for each arm within each study in addition to sample size. Descriptive characteristics were then tabulated across studies for reporting.

For each arm, and observation time point if multiple observations reported (e.g., before and after an intervention), depending on what was reported by the authors we extracted the means,

medians, standard deviations, standard errors, lower and upper range values, and interquartile range for the unadjusted and/or body mass adjusted and/or fat free mass adjusted REE values. Where REE values adjusted for body mass and/or fat free mass were reported we used the reported body mass and/or fat free mass mean values for that arm to convert them to unadjusted REE values (i.e., multiplied them by body mass and/or fat free mass mean values). Where means and/or standard deviations were missing the latter were either calculated from standard errors and sample size, or all both were estimated from lower and upper range, interquartile range, median, and sample size depending on the information available using the methods of Wan et al. (2014). Further, where missing, height/body mass/BMI where estimated based on the means reported for these. The units of measurement for which REE was extracted and all REE values were converted to kcal/day. In one case (Pohlmeier et al., 2014) REE was reported relative to body mass and the unadjusted values were no longer available (confirmed by the authors) as such in this case we used the mean body mass to convert back to estimated REE unadjusted.

Studies with possible reporting errors

During data extraction it was noted that several studies from the same lab/research group (Georgopoulos et al., 2009; Koika et al., 2009; Kritikou et al., 2006; Saltamavros et al., 2007) contained a number of discrepancies that seemed to be possible reporting errors. This included, based on taking the authors results as written, standard errors that implied impossible or at least incredibly unlikely standard deviations, and discrepancies in sample size reporting throughout for most variables without explanation or where this was explained the sample sizes were discrepant with the text Further, data was not reported for the healthy control women without PCOS in three of the studies (Koika et al., 2009; Kritikou et al., 2006; Saltamavros et al., 2007), and REE was reported as an "adjusted" value whereby $\text{REE}_{adjusted} = \text{REE}_{group\ mean} + (\text{REE}_{adjusted} - \text{REE}_{predicted})$ and the $\text{REE}_{predicted}$ was obtained by substituting the individual lean body mass, fat mass, gender, and age in the linear regression equation generated by the data of all patients. In correspondence with the senior author we were unable to clarify the reporting discrepancies as the person responsible for the data/results was no longer contactable. The original data were also no longer available and so we could not calculated the unadjusted REE.

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

Statistical Analysis

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, transformations, analyses, plotting, and reporting are available in the corresponding GitHub repository https://github.com/jamessteeleii/pcos_ree_meta. We cite all software and packages used in the analysis pipeline using the `grateful` package (Rodriguez-Sanchez et al., 2023) which can be seen here: https://github.com/jamessteeleii/pcos_ree_meta/blob/main/grateful-report.pdf. The statistical analysis plan was linked in our pre-registration (PROSPERO: CRD42024601434) and available at the accompanying github repository. Any deviations from the pre-registration are noted below.

Given our research question our analysis was aimed at parameter estimation (Cumming, 2014) within a Bayesian meta-analytic framework (Kruschke & Liddell, 2018). For all analyses model parameter estimates and their precision, along with conclusions based upon them, are interpreted continuously and probabilistically, considering data quality, plausibility of effect, and previous literature, all within the context of each model. The `renv` package (Ushey et al., 2023) was used for package version reproducibility and a function based analysis pipeline using the `targets` package (Landau et al., 2023) was employed (the analysis pipeline can be viewed by downloading the R Project and running the function `targets::tar_visnetwork()`). Effect sizes and their variances were all calculated using the `metafor` packages `escalc()` function (Viechtbauer, 2023). The main package `brms` (Bürkner et al., 2023) was used in fitting all the Bayesian meta-analysis models. Prior and posterior draws were taken using `marginaleffects` (Arel-Bundock et al., 2023) and `tidybayes` (Kay & Mastny, 2023) packages. All visualisations are created using `ggplot2` (Wickham et al., 2023), `tidybayes`, and the `patchwork` (Pedersen, 2023) packages.

Main Pre-registered Models

We adopted an arm-based multiple condition comparison (i.e., network) type model given that the studies included had arms of women with PCOS both with, and without, a non-PCOS control arm (Hong et al., 2016), and also in some cases multiple observations of REE in the different arms included in the study (for example, where an intervention was conducted and pre- and post-intervention REE was reported). In typical contrast-based meta-analyses data is limited to the effect sizes for paired contrasts between arms and thus studies that include both arms (i.e., relative effects between non-PCOS control vs PCOS arms); however, in arm-based analyses the data are the absolute effects within each arm and information is borrowed across studies to enable both within condition absolute, and between condition relative contrasts to be estimated. We made use of historical information regarding REE in healthy control women without PCOS by setting informative priors based on meta-analysis of large scale studies reporting normative data for REE in this population. This was included to provide control information indirectly where this was missing from particular studies. The use of

historical priors like this is an efficient tool to incorporate historical information on a particular population in a conservative manner in meta-analyses (Weber et al., 2021). From this model we focus on reporting the global grand mean estimate for the fixed between condition relative contrast for non-PCOS control vs PCOS arms as our primary estimand of interest (i.e., β_1 in both mean and standard deviation models). We examined both raw mean REE (i.e., the absolute mean REE in kcals per day for each arm) in addition to the between person standard deviation in REE (i.e., the absolute standard deviation in REE in kcals per day for each arm). Both models were multilevel in that they included nested random intercepts for both study, and arm within study, and in addition and in deviation from our pre-registration we also included lab as a random intercept as in some cases we had multiple studies from the same lab or research group included. Lastly, the inclusion of a random intercept for each effect size was also accidentally omitted from our pre-registration and so this is also included in the model.

Mean REE Model

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

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

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

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

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

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

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

where $\hat{\theta}_{ijkl}$ is the lth mean REE estimate from the kth arm, for the jth study, conducted by the ith lab and σ^2_{ijkl} is the corresponding sampling error for that estimate. The random intercepts for the ith lab, jth study, kth arm, and lth mean REE estimate are $\alpha_{0,lab[i]}$, $\alpha_{0,study[j]}$, $\alpha_{0,arm[k]}$, and $\alpha_{0,effect[l]}$ respectively each with standard deviation of $\sigma^2_{0,lab[i]}$, $\sigma^2_{0,study[j]}$, $\sigma^2_{0,arm[k]}$, and $\sigma^2_{0,effect[l]}$. The parameter β_0 represents the fixed effect estimate of REE for control conditions and β_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 $\alpha_{1,lab[i]}$ and $\alpha_{1,study[j]}$ respectively, and these effects were also modelled as correlated with the corresponding random intercepts with covariance Σ_{lab} and Σ_{study}, and corr_{lab} and $\text{corr}_{\text{study}}$ correlation matrices.

The priors for this model were as follows:

$$\beta_0 \sim \text{Student-}t(3,\ 1441.81,\ 84.56)$$
$$\beta_1 \sim \text{Student-}t(3,\ 0,\ 200)$$

$$\sigma^2_{0,\text{study}} \sim \text{Half-student-}t(3,\ 149.89,\ 82.91)$$
$$\sigma^2_{0,\text{lab}},\ \sigma^2_{1,\text{lab}},\ \sigma^2_{1,\text{study}},\ \sigma^2_{0,\text{arm}}, \sigma^2_{0,\text{effect}} \sim \text{Half-student-}t(3,\ 0,\ 112.4)$$

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

where the prior for β_0, which corresponded to the model intercept and mean REE in the control condition, was set based on meta-analysis of the mean REEs for women from two large studies of healthy people (Pavlidou et al., 2023; Velasquez, 2011) though set with a conservative degrees of freedom for the Student-t distribution.The random intercept $\sigma^2_{0,study}$ was set similarly to this. The prior for the fixed effect β_1, reflecting the difference between control and PCOS conditions was set based on a wide range of possible values considering the minimum and and maximum values of the ranges reported in the two studies noted (i.e., 2492 - 908 = 1584). We then set a prior that permits values approximately across this range of values with the majority of it's mass centred around zero. The remaining random effects were set based on the default weakly regularising priors for `brms` and scaled to the expected response values using a half-student-t distribution with 3 degrees of freedom and $\mu = 0$, and both correlation matrices corr_{lab} and corr_{study} were set with an LKJcorr(1) distribution.

Standard Deviation of REE Model

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

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

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

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

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

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

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

$$\alpha_{0,\mathrm{arm}[k]} \sim \mathcal{N}(0, \sigma^2_{0,\mathrm{arm}}), \text{ for arm k} = 1, \ldots, \mathrm{K}$$

$$\alpha_{0,\mathrm{effect}[l]} \sim \mathcal{N}(0, \sigma^2_{0,\mathrm{effect}}), \text{ for effect l} = 1, \ldots, \mathrm{L}$$

where $\log(\hat{\theta}_{ijkl})$ is the lth natural logarithm of the standard deviation of REE estimate from the kth arm, for the jth study, conducted by the ith lab and σ^2_{ijkl} is the corresponding sampling error for that estimate. The random intercepts for the ith lab, jth study, kth arm, and lth standard deviation of REE estimate are $\alpha_{0,lab[i]}$, $\alpha_{0,study[j]}$, $\alpha_{0,arm[k]}$, and $\alpha_{0,effect[l]}$ respectively each with standard deviation of $\sigma^2_{0,lab[i]}$, $\sigma^2_{0,study[j]}$, $\sigma^2_{0,arm[k]}$, and $\sigma^2_{0,effect[l]}$. The parameter β_0 represents the fixed effect estimate of standard deviation of REE for control conditions and β_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 $\alpha_{1,lab[i]}$ and $\alpha_{1,study[j]}$ respectively, and these effects were also modelled as correlated with the corresponding random intercepts with covariance Σ_{lab} and Σ_{study}, and $\mathrm{corr}_{\mathrm{lab}}$ and $\mathrm{corr}_{\mathrm{study}}$ correlation matrices. Finally, β_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 lth natural logarithm of the mean REE estimate from the kth arm, for the jth study, conducted by the ith lab and $\sigma^2_{\log(y_{i,\mathrm{mean},[\mathrm{ijkl}]})}$ is the corresponding sampling error for that estimate.

The priors for this model were as follows:

$$
\begin{aligned}
\beta_0 &\sim \text{Student-}t(3,\ 5.54,\ 0.80) \\
\beta_1 &\sim \text{Student-}t(3,\ 0,\ 5.3) \\
\beta_2 &\sim \text{Student-}t(3,\ 0,\ 2.5) \\
\\
\text{mean}(\tilde{m}) &\sim \text{Half-student-}t(3,\ 7.28,\ 0.62) \\
\text{sd}(\tilde{m}) &\sim \text{Half-student-}t(3,\ 0,\ 5) \\
\\
\sigma^2_{0,\text{study}} &\sim \text{Half-student-}t(3,\ 1.08,\ 1.06) \\
\sigma^2_{0,\text{lab}},\ \sigma^2_{1,\text{lab}},\ \sigma^2_{1,\text{study}},\ \sigma^2_{0,\text{arm}}, \sigma^2_{0,\text{effect}} &\sim \text{Half-student-}t(3,\ 0,\ 2.5) \\
\\
\text{corr}_{\text{lab}} &\sim \text{LKJ}(1) \\
\text{corr}_{\text{study}} &\sim \text{LKJ}(1)
\end{aligned}
$$

where the prior for β_0, which corresponded to the model intercept and standard deviation of REE in the control condition, was again set based on meta-analysis of the standard deviation of REEs for women from two large studies of healthy people (Pavlidou et al., 2023; Velasquez, 2011) though set with a conservative degrees of freedom for the Student-t distribution.The random intercept $\sigma^2_{0,study}$ was set similarly to this. The prior for the fixed effect β_1, reflecting the difference between control and PCOS conditions was set based on a wide range of possible values. Given that in many cases of variables in the field there is an approximate relationship of ~1 for the natural logarithm of the standard deviation conditioned upon the natural logarithm of the mean (Steele et al., 2023) we set this prior to reflect the range of differences on the on the log scale (i.e., log(1584)). We then set a prior that permits values approximately across this range of values with the majority of it's mass centred around zero. The prior for the fixed effect β_2, reflecting the relationship between the natural logarithm of the mean REE with the natural logarithm of the standard deviation of REE, was set it to be weakly informative centred on zero with a wide scale to indicate uncertainty in this outcome specifically despite the typical relationship close to ~1. Priors for the measurement error of the natural logarithm of the mean REE estimate $\tilde{m}$ were again based upon meta-analysis of the aforementioned studies, though measurement error has to be positive, as does the corresponding standard deviation of this error, so we set these to conservative wide half-student-t distributions. The remaining random effects were set based on the default weakly regularising priors for `brms` and scaled to the expected response values using a half-student-t distribution with 3 degrees of freedom and $\mu = 0$, and both correlation matrices corr_{lab} and corr_{study} were set with an LKJcorr(1) distribution.

Post-processing of models

For both models we examined trace plots along with $\hat{R}$ values to examine whether chains have converged, and posterior predictive checks for each model to understand the model implied distributions. From each model we took draws from the posterior distributions for the conditional absolute estimates for each condition (i.e., controls and PCOS) by study incorporating random effects, the global grand mean absolute estimates for each condition ignoring random effects, and the global grand mean between condition relative contrast for controls vs PCOS conditions ignoring random effects. The between condition relative contrast for controls vs PCOS conditions corresponded to $\beta 1$ in each model and was our primary estimand of interest; for the mean REE model this corresponded to the absolute difference in mean REE, and for the standard deviation of REE model this corresponded to the natural logarithm of the ratio of standard deviations of REE which was exponentiated (note, all log standard deviation of REE model estimates were exponentiated back to the original scale to aid interpretibility). We present the full probability density functions for posterior visually, and also to calculate mean and 95% quantile intervals (QI: i.e., 'credible' or 'compatibility' intervals) for each estimate providing the most probable value of the parameter in addition to the range from 2.5% to 97.5% percentiles given our priors and data.

Sensitivity analyses

Pairwise contrast based models

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

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

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

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

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

where $\hat{\theta}_{ijkl}$ is the pairwise effect size, either the mean difference in REE or the log coefficient of variation ratio (calculated as PCOS vs control), for the jth study, conducted by the ith lab and σ^2_{ijkl} is the corresponding sampling error for that effect size estimate. The random intercepts for the ith lab, jth study are $\alpha_{0,lab[i]}$ and $\alpha_{0,study[j]}$ respectively each with standard deviation of $\sigma^2_{0,lab[i]}$, $\sigma^2_{0,study[j]}$. The parameter β_0 represents the fixed effect estimate of the pairwise effect size i.e., the pooled estimate of the contrast between conditions. The priors for β_0 in these models were set as default weakly regularising which is set on an intercept that results when internally centering all population-level predictors around zero to improve sampling efficiency and scaled to the expected response values using a student-t distribution;

for the mean difference in REE this was student-$t(3,\ 5.5,\ 50.2)$ and for the log coefficient of variance ratio this was student-$t(3,\ -0.1,\ 2.5)$. The random effects were set similarly scaled to the expected response values but using a half-student-t distribution centred on zero. From these models we calculated the mean and 95% quantile intervals (i.e., 'credible' or 'compatibility' intervals) for the β_0 (comparable to the β_1 from the corresponding mean and standard deviation of REE arm-based models) for each effect size providing the most probable value of the parameter in addition to the range from 2.5% to 97.5% percentiles given our priors and data.

Additional sensitivity analyses

As noted in the section above, *"Studies with possible reporting errors"*, we opted to conduct analysis with and without the inclusion of four studies with possible reporting errors we could not resolve (Georgopoulos et al., 2009; Koika et al., 2009; Kritikou et al., 2006; Saltamavros et al., 2007). Thus the main models described above were run with and without these studies, the main results are presented without them and the sensitivity results with them are reported separately below.

As a further sensitivity analysis, given the inclusion of some studies with interventions in women with PCOS reporting REE at multiple timepoints such as mid- and post-intervention (Bruner et al., 2006; Moran et al., 2006; Pohlmeier et al., 2014) but lacking of control women without PCOS, we opted to also conduct sensitivity analysis excluding these and only examining their baseline results in addition to the cross-sectional studies. As such, the main models described above were run without the follow-up (i.e., mid- or post-intervention timepoints) from these studies and only using the baseline results in addition to other cross-sectional studies.

Results

Systematic Review

Our systematic review identified 13 studies from 12 lab/research groups including 24 arms (Control arms = 9, PCOS arms = 15) and a total of 918 participants (Controls: minimum n = 9, median n = 29, maximum n = 54; PCOS: minimum n = 5, median n = 28, maximum n = 266). Descriptive characteristics of the arms and participants in these studies are reported in Table 1. Note, these numbers exclude the four studies previously noted with possible reporting issues that were unresolved (they are however summarised in Table 1).

Table 1: Descriptive characteristics of arms and participants for included studies

Authors	Article title	Condition	Diagnostic criteria	Sample size	Age†	Body mass†*	Height†*	BMI†*	Fat mass†	Fat free mass†	Race/Ethnicity	Physical activity	Country of study	Metabolic health	Measurement Method
Segal and Dunaif (1990)	Resting metabolic rate and postprandial thermogenesis in polycystic ovarian syndrome.	Control		11	28 (3.32)	62.5 (4.97)				48.8 (3.32)			USA	OGTT (75g oral glucose load), Fasting glucose (mM) = 4.5 (SE = 0.2), Fasting insulin (pM) = 72 (SE = 7), Glucose area (mM 2 hours) = 9.9 (SE = 0.2), Insulin area (pM 2 hours) = 653 (SE = 7)	Indirect Calorimetry: Sensormedics Horizon Metabolic Measurement Cart (Sensormedics Corporation, Anaheim, CA, USA)
		Control		9	29 (6)	86.9 (17.4)				50.4 (7.5)			USA	OGTT (75g oral glucose load), Fasting glucose (mM) = 4.8 (SE = 0.1), Fasting insulin (pM) = 122 (SE = 14), Glucose area (mM 2 hours) = 9.9 (SE = 0.2), Insulin area (pM 2 hours) = 1300 (SE = 294)	Indirect Calorimetry: Sensormedics Horizon Metabolic Measurement Cart (Sensormedics Corporation, Anaheim, CA, USA)
		PCOS	PCOS was diagnosed by elevation of one or more plasma and androgen levels in the presence of chronic oligomenorrhea or ammenorrhea.	10	25 (6.32)	84.1 (8.54)				48.7 (3.79)			USA	OGTT (75g oral glucose load), Fasting glucose (mM) = 4.9 (SE = 0.1), Fasting insulin (pM) = 223 (SE = 43), Glucose area (mM 2 hours) = 12.4 (SE = 0.9), Insulin area (pM 2 hours) = 2872 (SE =381)	Indirect Calorimetry: Sensormedics Horizon Metabolic Measurement Cart (Sensormedics Corporation, Anaheim, CA, USA)
Robinson et al., (1992)	Postprandial thermogenesis is reduced in polycystic ovary syndrome and is associated with increased insulin resistance.	PCOS	PCOS was defined by the clinical features: amenorrhoea or oligomenorrhoea menstrual cycle longer than 35 days) and/or hirsutism (score greater than 8) with polycystic ovaries on ultrasound scanning.	14	29 (6.44)	78.05 (18.95)	169.86*	27.05 (8.64)		51.12 (4.69)	Matched between groups but not reported		UK	Short insulin tolerance test (0.5 U/kg body weight), Glucose slope (median, 3-15 mins) = 148.5 (SD = 20.5), Peak insulin (mU/L, 4 mins) = 265 (SD = 18)	Indirect Calorimetry: Deltatrac metabolic monitor (Datex Instrumentarium, Helsinki, Finland)
		Control		14	30.25 (4.98)	76.62 (19.03)	165.87*	27.85 (6.65)		50.12 (7.99)	Matched between groups but not reported		UK	Short insulin tolerance test (0.5 U/kg body weight), Glucose slope (median, 3-15 mins) = 183.5 (SD = 26), Peak insulin (mU/L, 4 mins) = 273 (SD = 9)	Indirect Calorimetry: Deltatrac metabolic monitor (Datex Instrumentarium, Helsinki, Finland)
Bruner et al., (2006)	Effects of exercise and nutritional counseling in women with polycystic ovary syndrome	PCOS	Rotterdam criteria	7	32.3 (2.65)	100.5 (17.73)	166.62*	36.2 (5.29)				Sedentary at baseline (not specified how defined)	Canada	Fasting insulin (pmol/L) = 116.7 (SE = 42.2)	Indirect Calorimetry: Sensorimedics VMAX 29 series metabolic cart (Sensorimedics, Yorba Linda, CA, USA)
		PCOS	Rotterdam criteria	5	28.4 (6.04)	94.8 (13.86)	159.85*	37.1 (7.6)				Sedentary at baseline (not specified how defined)	Canada	Fasting insulin (pmol/L) = 233.8 (SE = 77.4)	Indirect Calorimetry: Sensorimedics VMAX 29 series metabolic cart (Sensorimedics, Yorba Linda, CA, USA)
Moran et al., (2006)	Short-term meal replacements followed by dietary macronutrient restriction enhance weight loss in polycystic ovary syndrome	PCOS	Rotterdam criteria	34	32.62 (5.17)	96 (19.24)	165.85*	34.9 (6.79)	34.9 (8.75)	61.5 (12.24)	White/Caucasian	24-h physical activity record for all 7 d/wk in weeks 0, 8, 20, and 32 (but not actually reported)	Australia	Fasting glucose (mmol/L) = 5.2 (SE = 0.1), Fasting insulin (mU/L, baseline only) = 12.86 (SD = 6.95), HOMA (baseline only) = 2.5 (SD = 1.77)	Indirect Calorimetry: Deltatract metabolic monitor (Datex Division Instrumentarium Corp., Helsinki, Finland) using a ventilated canopy
Cosar et al., (2008)	Resting metabolic rate and exercise capacity in women with polycystic ovary syndrome.	PCOS	Rotterdam criteria	31	25.9 (5.3)			26.97 (5.12)					Turkey	Fasting glucose/insulin (ratio) = 6.01 (SD = 3.72), Fasting glucose (mg/dL) = 95.64 (SD = 11.03), Fasting insulin (mIU/mL) = 16.13 (SD = 9.86)	Indirect Calorimetry: Quark b2 (Cosmed, Rome, Italy) with a computerized metabolic card
		Control		29	27.1 (4.8)			26.03 (5.66)					Turkey	Fasting glucose/insulin (ratio) = 13.56 (SD = 6.13), Fasting glucose (mg/dL) = 92.49 (SD = 10.66), Fasting insulin (mIU/mL) = 7.25 (SD = 3.01)	Indirect Calorimetry: Quark b2 (Cosmed, Rome, Italy) with a computerized metabolic card
Graff et al., (2013)	Dietary glycemic index is associated with less favorable anthropometric and metabolic profiles in polycystic ovary syndrome women with different phenotypes	PCOS	Rotterdam criteria	61	22.7 (6.2)			28.9 (5.6)			White/Caucasian - 87.6%	Physical activity (steps/day, median) = 5519 (IQR = 3658-7002)	Brazil	OGTT (75g oral glucose load), Fasting glucose (md/dL) = 86.8 (SD = 91), Fasting insulin (uU/mL, median) = 16.7 (IQR = 9.8-21.2), HOMA-IR (median) = 3.5 (IQR = 2.1-4.7)	Indirect Calorimetry: Fitmate (Cosmed, Rome, Italy)
		Control		44	25 (5.6)			27.1 (5.7)			White/Caucasian - 87.6%	Physical activity (steps/day, median) = 5811 (IQR = 4339-7267)	Brazil	OGTT (75g oral glucose load), Fasting glucose (md/dL) = 87 (SD = 7.5), Fasting insulin (uU/mL, median) = 9.9 (IQR = 6.8-12.5), HOMA-IR (median) = 2.1 (IQR = 1.4-2.8)	Indirect Calorimetry: Fitmate (Cosmed, Rome, Italy)
Pohlmeier er al., (2014)	Effect of a low-starch/low-dairy diet on fat oxidation in overweight and obese women with polycystic ovary syndrome.	PCOS	Rotterdam criteria	10	29.6 (4.6)	105.4 (14.5)	165.46*	38.5 (4.2)	52.4 (14.8)	52.3 (10.7)	White/Caucasian = 6, Hispanic = 3, Native American = 1	Physical activity level (ratio of TDEE/RMR) = 1.65	USA	OGTT (75g oral glucose load), Fasting glucose (md/dL) = 91.6 (SD = 9.4), Fasting insulin (ug/mL) = 35.3 (SD = 7), Glucose at 2 hours (mg/dL) = 131.7 (SD = 45.2), Insulin at 2 hours (ug/mL) = 271.6 (SD =285), HbA1c (%) = 5.5 (SD = 0.4)	Indirect Calorimetry: ParvoMedics TrueOne 2400 Canopy System (ParvoMedics, Sandy, Utah, USA).
Doh et al., (2016)	The Relationship between Adiposity and Insulin Sensitivity in African Women Living with the Polycystic Ovarian Syndrome: A Clamp Study.	PCOS	Rotterdam criteria	6	26 (5.19)			34.1 (3.56)	41.2 (12.45)	56.3 (4.97)	African	Engaged in sporting activities < 2 days/week - 50%	Cameroon	Hyperinsulinemic euglycemic clamp technique, M-value (mg/kg/min, median) = 6.6 (IQR = 5.5-7.3)	Indirect Calorimetry: Korr ReeVue indirect calorimetry (KorrMedical Technologies, Inc., Salt Lake City, UT 84120, USA)
		PCOS	Rotterdam criteria	8	27 (3.71)			26.4 (2.97)	23.3 (6.89)	47.4 (5.56)	African	Engaged in sporting activities < 2 days/week - 87.5%	Cameroon	Hyperinsulinemic euglycemic clamp technique, M-value -(mg/kg/min, median) = 9.1 (IQR 7.7-10)	Indirect Calorimetry: Korr ReeVue indirect calorimetry (KorrMedical Technologies, Inc., Salt Lake City, UT 84120, USA)
		Control		10	23 (0.74)			22.5 (3.63)	17.1 (7.56)	45.9 (6.67)	African	Engaged in sporting activities < 2 days/week - 80%	Cameroon	Hyperinsulinemic euglycemic clamp technique, M-value (mg/kg/min, median) = 11.9 (IQR = 9.4-14.5)	Indirect Calorimetry: Korr ReeVue indirect calorimetry (KorrMedical Technologies, Inc., Salt Lake City, UT 84120, USA)
Larsson et al., (2016)	Dietary intake, resting energy expenditure, and eating behavior in women with and without polycystic ovary syndrome.	PCOS	Modified Rotterdam criteria	72	30.2 (4.4)	79.6 (20.3)	167.12*	28.5 (7.2)					Sweden		Indirect Calorimetry: Deltatrack II Metabolic Monitor ventilated hood system (Datex, Helsinki, Finland).
		Control		30	27.8 (3.6)	70.9 (17.1)	169.77*	24.6 (5)					Sweden		Indirect Calorimetry: Deltatrack II Metabolic Monitor ventilated hood system (Datex, Helsinki, Finland).
Graff et al., (2017)	Saturated Fat Intake Is Related to Heart Rate Variability in Women with Polycystic Ovary Syndrome.	PCOS	Rotterdam criteria	84	23.5 (6.3)			29.4 (6.4)			White/Caucasian - 92.9%	Physical activity (steps/day, median) = 5821 (IQR = 3821-7664)	Brazil	OGTT (75g oral glucose load), Fasting glucose (md/dL) = 87.4 (SD = 8.4), Glucose at 2 hours (mg/dL) = 103.6 (SD = 31.5), HOMA-IR (median) = 3.4 (IQR = 1.8-4.7)	Indirect Calorimetry: Fitmate (Cosmed, Rome, Italy)
		Control		54	26.2 (6.5)			27.2 (5.8)			White/Caucasian - 88.9%	Physical activity (steps/day, median) = 6002 (IQR = 4375-7427)	Brazil	OGTT (75g oral glucose load), Fasting glucose (md/dL) = 86.8 (SD = 7.9), Glucose at 2 hours (mg/dL) = 97 (SD = 20.9), HOMA-IR (median) = 2.1 (IQR = 1.5-2.8)	Indirect Calorimetry: Fitmate (Cosmed, Rome, Italy)
Rodriques et al., (2017)	Low validity of predictive equations for calculating resting energy expenditure in overweight and obese women with polycystic ovary syndrome.	PCOS	Rotterdam criteria	30	30.8 (5.4)	85.3 (13.1)	161.76*	32.6 (3.7)				"Physical activity level was assessed using criteria established by the Institute of Medicine" 66.7% classified sedentary, 33.3% classified as low activity level	Brazil		Indirect Calorimetry: Meta-CheckTM metabolic rate analysis system (model 7100; Korr Medical Technologies, Salt Lake City, UT, USA)
Broskey et al., (2017)	Assessing Energy Requirements in Women With Polycystic Ovary Syndrome: A Comparison Against Doubly Labeled Water.	PCOS	1990 National Institutes of Health criteria	28	28.6 (5)	104.1 (19.3)	161.52*	39.9 (8.3)	51.6 (15.4)	52.5 (7.5)	White/Caucasian - 50%, African American - 50%	Physical activity level (ratio of TDEE/RMR) = 1.6 (SD = 0.2)	USA	Fasting glucose (md/dL) = 89.9 (SD = 6.9), Fasting insulin (uU/mL) = 18.8 (SD = 10.6), HOMA-IR = 4.3 (SD = 2.7)	Doubly Labelled Water: Oral dose (1.0 g/kg bodyweight) of amixture that contained 1 part deuterium (2H 99.9% enriched) and 19 parts Oxygen-18 (18O10% enriched), followed by 100 mL of tap water used to rinse the dose container.
Tosi et al., (2024)	Resting energy expenditure in women with polycystic ovary syndrome	PCOS	Rotterdam criteria	266	23.3 (5.2)			28.3 (7.4)	27.1 (14.4)	49 (7.7)			Italy	Hyperinsulinemic euglycemic clamp technique, M-value (mg/kg_FFM x min) = 9.8 (SD = 3.7), 72.5% classified as IR based on cut off value of 11.76, Fasting glucose (mg/dL) = 85.3 (SD = 9.5), Fasting insulin (mU/L) = 16.2 (SD = 12.6)	Indirect Calorimetry: Quark RMR instrument (Cosmed, Cernusco sul Naviglio, Italy) equipped with a ventilated hood
		Control		51	25.2 (3.6)			20.5 (2)	13.6 (5.4)	42.8 (4.6)			Italy	Fasting glucose (mg/dL) = 83.4 (SD = 5.9), Fasting insulin (mU/L) = 7 (SD = 5)	Indirect Calorimetry: Quark RMR instrument (Cosmed, Cernusco sul Naviglio, Italy) equipped with a ventilated hood

PCOS = polycystic ovary syndrome; BMI = body mass index; OGTT = oral glucose tolerance test; HOMA-IR = homeostatic model assessment of insulin resistance

†Values are Mean (SD) unless otherwise specified; note, some have been calculated/estimated from corresponding standard error, range, iqr, median, and sample size (see data and code)

†*Indicates that this mean was estimated from the corresponding means for body mass/height/BMI

Mean REE Model Results

The main model for mean REE resulted in a posterior distribution for the contrast between control and PCOS conditions with a mean point estimate of 30 kcal/day with a 95% quantile interval ranging from -47 kcal/day to 113 kcal/day suggesting there is a 95% probability that the true difference lies between these values given our priors and the data from included studies. The corresponding conditional estimates for the control condition and PCOS condition respectively were 1442 kcal/day [95%QI:1334 kcal/day to 1553 kcal/day] and 1472 kcal/day [95%QI:1359 kcal/day to 1587 kcal/day]. These results including the full visualisation of the posterior distribution, in addition to the conditional estimates by study, can be seen in Figure 1.

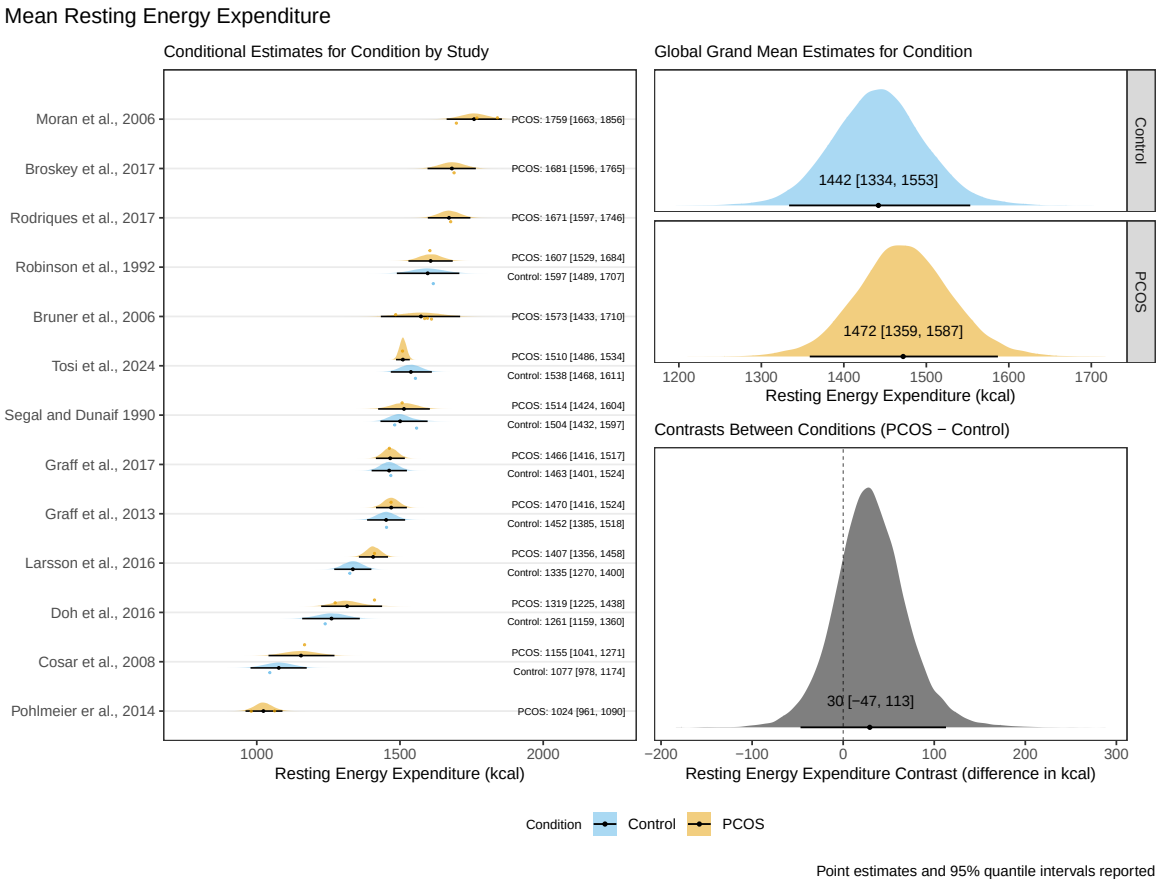

Figure 1: Posterior distribution, mean point estimates, and 95% quantile intervals for conditional estimates by study, global grand mean estimates by condition, and the contrast between conditions for mean resting energy expenditure of control women without PCOS and women with PCOS.

Standard Deviation of REE Model Results

The main model for the between participant standard deviation of REE resulted in a posterior distribution for the contrast ratio between control and PCOS conditions with a mean point estimate of 0.98 with a 95% quantile interval ranging from 0.71 to 1.33 suggesting there is a 95% probability that the true ratio of standard deviations lies between these values given our priors and the data from included studies. The corresponding conditional estimates for the standard deviations of the control condition and PCOS condition respectively were 238 kcal/day [95%QI:178 kcal/day to 312 kcal/day] and 229 kcal/day [95%QI:169 kcal/day to 303 kcal/day]. These results including the full visualisation of the posterior distribution, in addition to the conditional estimates by study, can be seen in Figure 2.

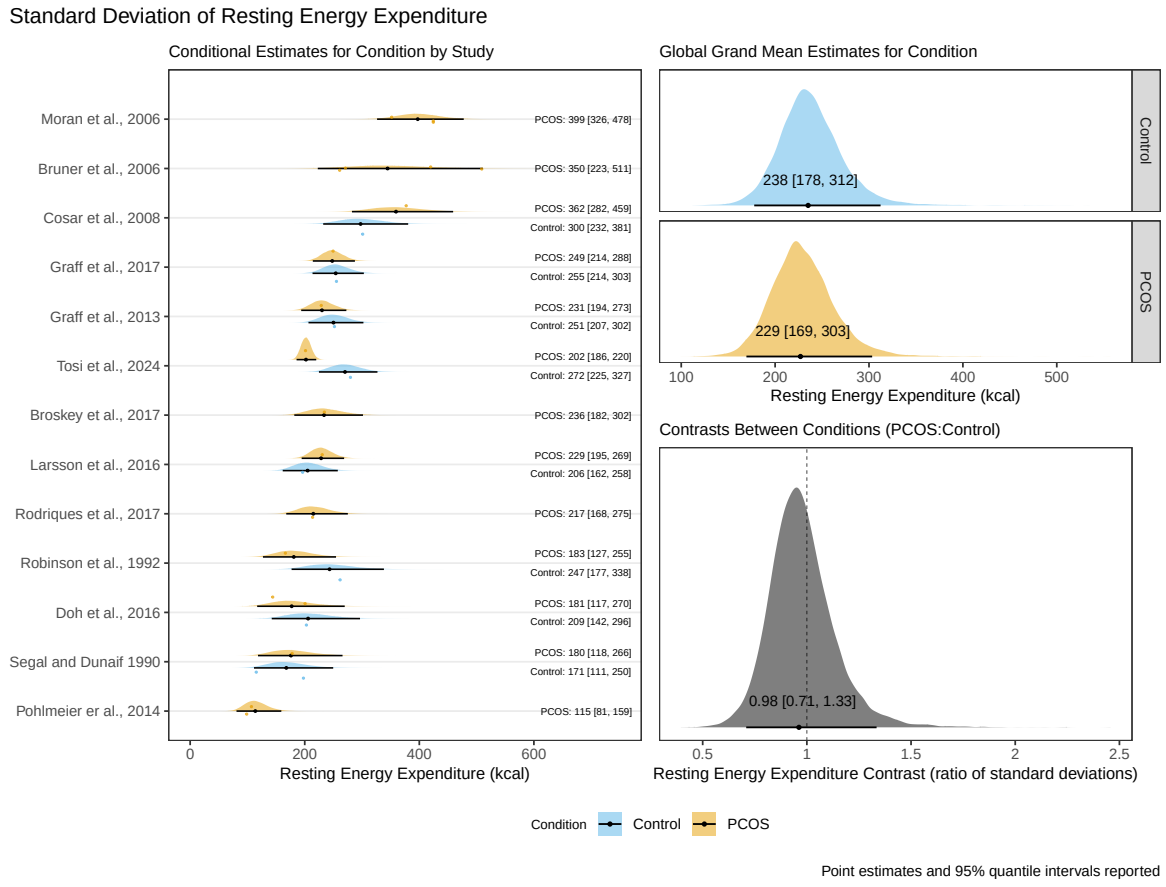

Figure 2: Posterior distribution, variance point estimates, and 95% quantile intervals for conditional estimates by study, global grand variance estimates by condition, and the contrast ratio between conditions for the standard deviation of resting energy expenditure of control women without PCOS and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

Sensitivity Analyses

Pairwise Models

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

Models including studies with possible reporting issues

For mean REE the model including the studies noted with possible reporting issues that were not resolved (Georgopoulos et al., 2009; Koika et al., 2009; Kritikou et al., 2006; Saltamavros et al., 2007) still resulted in qualitatively similar inferences suggesting little difference between control and PCOS conditions with mean point estimate of 15 kcal/day with a 95% quantile interval ranging from -69 kcal/day to 95 kcal/day. This was similar for the standard deviation of REE with the model including these studies resulting in a contrast ratio between control and PCOS conditions with a mean point estimate of 1.21 with a 95% quantile interval ranging from 0.68 to 2.13.

Basline REE Measurement Models

For mean REE the model which included only baseline REE measurements from studies involving interventions (Bruner et al., 2006; Moran et al., 2006; Pohlmeier et al., 2014), in addition to other cross-sectional studies, also resulted in qualitatively similar inferences suggesting little difference between control and PCOS conditions with mean point estimate of 32 kcal/day with a 95% quantile interval ranging from -46 kcal/day to 118 kcal/day. This was the case also for the standard deviation of REE with this model resulting in a contrast ratio between control and PCOS conditions with a mean point estimate of 0.97 with a 95% quantile interval ranging from 0.71 to 1.36.

Discussion

TO WRITE

References

Alenezi, S. A., Elkmeshi, N., Alanazi, A., Alanazi, S. T., Khan, R., & Amer, S. (2024). 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*, *13*(16), 4934. https://doi.org/10.3390/jcm13164934

Arel-Bundock, V., cre, cph, Diniz, M. A., Greifer, N., & Bacher, E. (2023). *Marginaleffects: Predictions, Comparisons, Slopes, Marginal Means, and Hypothesis Tests*.

Barber, T. M. (2022). Why are women with polycystic ovary syndrome obese? *British Medical Bulletin*, *143*(1), 4–15. https://doi.org/10.1093/bmb/ldac007

Barber, T. M., & Franks, S. (2021). Obesity and polycystic ovary syndrome. *Clinical Endocrinology*, *95*(4), 531–541. https://doi.org/10.1111/cen.14421

Bruner, B., Chad, K., & Chizen, D. (2006). Effects of exercise and nutritional counseling in women with polycystic ovary syndrome. *Applied Physiology, Nutrition, and Metabolism = Physiologie Appliquee, Nutrition Et Metabolisme*, *31*(4), 384–391. https://doi.org/10.1139/h06-007

Bürkner, P.-C., Gabry, J., Weber, S., Johnson, A., Modrak, M., Badr, H. S., Weber, F., Ben-Shachar, M. S., Rabel, H., Mills, S. C., & Wild, S. (2023). *Brms: Bayesian Regression Models using 'Stan'*.

Cumming, G. (2014). The New Statistics: Why and How. *Psychological Science*, *25*(1), 7–29. https://doi.org/10.1177/0956797613504966

Davitadze, M., Malhotra, K., Khalil, H., Hebbar, M., Tay, C. T., Mousa, A., PCOS SEva team, Teede, H., Brennan, L., Stener-Victorin, E., & Kempegowda, P. (2023). Body image concerns in women with polycystic ovary syndrome: A systematic review and meta-analysis. *European Journal of Endocrinology*, *189*(2), R1–R9. https://doi.org/10.1093/ejendo/lvad110

Geller, S., Levy, S., & Avitsur, R. (2025). Body image, illness perception, and psychological distress in women coping with polycystic ovary syndrome (PCOS). *Health Psychology Open*, *12*, 20551029251327441. https://doi.org/10.1177/20551029251327441

Georgopoulos, N. A., Saltamavros, A. D., Vervita, V., Karkoulias, K., Adonakis, G., Decavalas, G., Kourounis, G., Markou, K. B., & Kyriazopoulou, V. (2009). Basal metabolic rate is decreased in women with polycystic ovary syndrome and biochemical hyperandrogenemia and is associated with insulin resistance. *Fertility and Sterility*, *92*(1), 250–255. https://doi.org/10.1016/j.fertnstert.2008.04.067

Glintborg, D., Kolster, N. D., Ravn, P., & Andersen, M. S. (2022). Prospective Risk of Type 2 Diabetes in Normal Weight Women with Polycystic Ovary Syndrome. *Biomedicines*, *10*(6), 1455. https://doi.org/10.3390/biomedicines10061455

Glintborg, D., Ollila, M.-M., Møller, J.-J. K., Pesonen, P., Persson, S., Elenis, E., Rubin, K. H., Gissler, M., Andersen, M. S., Sundström-Poromaa, I., & Piltonen, T. (2024). 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), *39*(8), 1823–1834. https://doi.org/10.1093/humrep/deae124

Glintborg, D., Rubin, K. H., Nybo, M., Abrahamsen, B., & Andersen, M. (2018). Cardiovascular disease in a nationwide population of Danish women with polycystic ovary syndrome. *Cardiovascular Diabetology*, *17*(1), 37. https://doi.org/10.1186/s12933-018-0680-5

Graff, S. K., Mario, F. M., Magalhães, J. A., Moraes, R. S., & Spritzer, P. M. (2017). Saturated Fat Intake Is Related to Heart Rate Variability in Women with Polycystic Ovary Syndrome. *Annals of Nutrition & Metabolism*, *71*(3-4), 224–233. https://doi.org/10.1159/000484325

Himelein, M. J., & Thatcher, S. S. (2006). Depression and body image among women with polycystic ovary syndrome. *Journal of Health Psychology*, *11*(4), 613–625. https://doi.org/10.1177/1359105306065021

Hirschberg, A. L., Naessén, S., Stridsberg, M., Byström, B., & Holtet, J. (2004). 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*, *19*(2), 79–87. https://doi.org/10.1080/09513590400002300

Hofmann, K., Decrinis, C., Bitterlich, N., Tropschuh, K., Stute, P., & Bachmann, A. (2025). Body image and mental health in women with polycystic ovary syndrome-a cross-sectional study. *Archives of Gynecology and Obstetrics*, *312*(1), 177–190. https://doi.org/10.1007/s00404-024-07913-4

Holte, J., Bergh, T., Berne, C., Wide, L., & Lithell, H. (1995). 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*, *80*(9), 2586–2593. https://doi.org/10.1210/jcem.80.9.7673399

Hong, H., Chu, H., Zhang, J., & Carlin, B. P. (2016). A Bayesian missing data framework for generalized multiple outcome mixed treatment comparisons. *Research Synthesis Methods*, *7*(1), 6–22. https://doi.org/10.1002/jrsm.1153

Joham, A. E., Teede, H. J., Ranasinha, S., Zoungas, S., & Boyle, J. (2015). 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)*, *24*(4), 299–307. https://doi.org/10.1089/jwh.2014.5000

Kay, M., & Mastny, T. (2023). *Tidybayes: Tidy Data and 'Geoms' for Bayesian Models*.

Koika, V., Marioli, D. J., Saltamavros, A. D., Vervita, V., Koufogiannis, K. D., Adonakis, G., Decavalas, G., & Georgopoulos, N. A. (2009). 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*, *161*(2), 317–322. https://doi.org/10.1530/EJE-08-1014

Kritikou, S., Saltamavros, A. D., Adonakis, G., Koika, V., Koufogiannis, K., Spyropoulos, K., Kourounis, G., Kyriazopoulou, V., Georgopoulos, N. A., & Flordellis, C. (2006). The α2B and β3 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*(2).

Kruschke, J. K., & Liddell, T. M. (2018). The Bayesian New Statistics: Hypothesis testing, estimation, meta-analysis, and power analysis from a Bayesian perspective [Journal Article].

Psychonomic Bulletin & Review, *25*, 178–206.

Landau, W. M., Warkentin, M. T., Edmondson, M., Oliver, S., Mahr, T., & Company, E. L. and. (2023). *Targets: Dynamic Function-Oriented 'Make'-Like Declarative Pipelines*.

Larsson, I., Hulthén, L., Landén, M., Pålsson, E., Janson, P., & Stener-Victorin, E. (2016). Dietary intake, resting energy expenditure, and eating behavior in women with and without polycystic ovary syndrome. *Clinical Nutrition (Edinburgh, Scotland)*, *35*(1), 213–218. https://doi.org/10.1016/j.clnu.2015.02.006

Lim, S. S., Kakoly, N. S., Tan, J. W. J., Fitzgerald, G., Bahri Khomami, M., Joham, A. E., Cooray, S. D., Misso, M. L., Norman, R. J., Harrison, C. L., Ranasinha, S., Teede, H. J., & Moran, L. J. (2019). 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*, *20*(2), 339–352. https://doi.org/10.1111/obr.12762

Moran, L. J., Noakes, M., Clifton, P. M., Wittert, G. A., Tomlinson, L., Galletly, C., Luscombe, N. D., & Norman, R. J. (2004). 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*, *89*(7), 3337–3344. https://doi.org/10.1210/jc.2003-031583

Moran, L. J., Noakes, M., Clifton, P. M., Wittert, G. A., Williams, G., & Norman, R. J. (2006). Short-term meal replacements followed by dietary macronutrient restriction enhance weight loss in polycystic ovary syndrome. *The American Journal of Clinical Nutrition*, *84*(1), 77–87. https://doi.org/10.1093/ajcn/84.1.77

Page, M. J., McKenzie, J. E., Bossuyt, P. M., Boutron, I., Hoffmann, T. C., Mulrow, C. D., Shamseer, L., Tetzlaff, J. M., Akl, E. A., Brennan, S. E., Chou, R., Glanville, J., Grimshaw, J. M., Hróbjartsson, A., Lalu, M. M., Li, T., Loder, E. W., Mayo-Wilson, E., McDonald, S., … Moher, D. (2021). The PRISMA 2020 statement: An updated guideline for reporting systematic reviews. *BMJ*, *372*, n71. https://doi.org/10.1136/bmj.n71

Pavlidou, E., Papadopoulou, S. K., Seroglou, K., & Giaginis, C. (2023). Revised Harris–Benedict Equation: New Human Resting Metabolic Rate Equation. *Metabolites*, *13*(2), 189. https://doi.org/10.3390/metabo13020189

Pedersen, T. L. (2023). *Patchwork: The Composer of Plots*.

Persson, S., Elenis, E., Turkmen, S., Kramer, M. S., Yong, E.-L., & Poromaa, I. S. (2021). Higher risk of type 2 diabetes in women with hyperandrogenic polycystic ovary syndrome. *Fertility and Sterility*, *116*(3), 862–871. https://doi.org/10.1016/j.fertnstert.2021.04.018

Pesonen, E., Nurkkala, M., Niemelä, M., Morin-Papunen, L., Tapanainen, J. S., Jämsä, T., Korpelainen, R., Ollila, M.-M., & Piltonen, T. T. (2023). 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.)*, *31*(4), 1108–1120. https://doi.org/10.1002/oby.23681

Pohlmeier, A. M., Phy, J. L., Watkins, P., Boylan, M., Spallholz, J., Harris, K. S., & Cooper, J. A. (2014). 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 Appliquee, Nutrition Et Metabolisme*, *39*(11), 1237–1244. https://doi.org/10.1139/apnm-2014-0073

Rodriguez-Sanchez, F., cre, cph, Jackson, C. P., Hutchins, S. D., & Clawson, J. M. (2023). *Grateful: Facilitate Citation of R Packages*.

Romualdi, D., Versace, V., Tagliaferri, V., De Cicco, S., Immediata, V., Apa, R., Guido, M., & Lanzone, A. (2019). 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*, *42*(9), 1089–1097. https://doi.org/10.1007/s40618-019-01029-2

Salari, N., Nankali, A., Ghanbari, A., Jafarpour, S., Ghasemi, H., Dokaneheifard, S., & Mohammadi, M. (2024). Global prevalence of polycystic ovary syndrome in women worldwide: A comprehensive systematic review and meta-analysis. *Archives of Gynecology and Obstetrics*, *310*(3), 1303–1314. https://doi.org/10.1007/s00404-024-07607-x

Saltamavros, A. D., Adonakis, G., Kritikou, S., Koika, V., Koufogiannis, K., Spyropoulos, K., Kourounis, G., Flordellis, C., Kyriazopoulou, V., & Georgopoulos, N. A. (2007). Alpha 2 beta adrenoreceptor 301-303 deletion polymorphism in polycystic ovary syndrome. *Clinical Autonomic Research: Official Journal of the Clinical Autonomic Research Society*, *17*(2), 112–114. https://doi.org/10.1007/s10286-007-0403-6

Segal, K. R., & Dunaif, A. (1990). Resting metabolic rate and postprandial thermogenesis in polycystic ovarian syndrome. *International Journal of Obesity*, *14*(7), 559–567.

Steele, J., Fisher, Smith, Schoenfeld, Yang, & and Nakagawa, S. (2023). Meta-analysis of variation in sport and exercise science: Examples of application within resistance training research. *Journal of Sports Sciences*, *41*(17), 1617–1634. https://doi.org/10.1080/02640414.2023.2286748

Stefanaki, K., Karagiannakis, D. S., Peppa, M., Vryonidou, A., Kalantaridou, S., Goulis, D. G., Psaltopoulou, T., & Paschou, S. A. (2024). Food Cravings and Obesity in Women with Polycystic Ovary Syndrome: Pathophysiological and Therapeutic Considerations. *Nutrients*, *16*(7), 1049. https://doi.org/10.3390/nu16071049

Teede, H. J., Tay, C. T., Laven, J. J. E., Dokras, A., Moran, L. J., Piltonen, T. T., Costello, M. F., Boivin, J., Redman, L. M., Boyle, J. A., Norman, R. J., Mousa, A., & Joham, A. E. (2023). Recommendations From the 2023 International Evidence-based Guideline for the Assessment and Management of Polycystic Ovary Syndrome. *The Journal of Clinical Endocrinology and Metabolism*, *108*(10), 2447–2469. https://doi.org/10.1210/clinem/dgad463

Ushey, K., cre, Wickham, H., Software, P., & PBC. (2023). *Renv: Project Environments*.

Velasquez, J. R. (2011). The Use of Ammonia Inhalants Among Athletes. *Strength & Conditioning Journal*, *33*(2), 33. https://doi.org/10.1519/SSC.0b013e3181fd5c9b

Viechtbauer, W. (2023). *Metafor: Meta-Analysis Package for R*.

Wan, X., Wang, W., Liu, J., & Tong, T. (2014). Estimating the sample mean and standard deviation from the sample size, median, range and/or interquartile range. *BMC Medical Research Methodology*, *14*(1), 135. https://doi.org/10.1186/1471-2288-14-135

Weber, S., Li, Y., Iii, J. W. S., Kakizume, T., & Schmidli, H. (2021). Applying Meta-Analytic-Predictive Priors with the R Bayesian Evidence Synthesis Tools. *Journal of Statistical Software*, *100*, 1–32. https://doi.org/10.18637/jss.v100.i19

Wickham, H., Chang, W., Henry, L., Pedersen, T. L., Takahashi, K., Wilke, C., Woo, K., Yutani, H., Dunnington, D., Posit, & PBC. (2023). *Ggplot2: Create Elegant Data Visual-*

isations Using the Grammar of Graphics.