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Bachelor of Science (B.Sc.) Psychologie

**Assessing Publication Bias in Meta-Analyses:
A Simulation-Based Estimation Approach Focusing on the Joint
Distribution of Effect Size and Sample Size**

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Abstract

Test

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Introduction

Science is commonly conceived as a cumulative enterprise (Cooper et al., 2019) with the overarching goal of attaining robust knowledge about the world (Kitcher, 1993). Within this landscape, researcher often study the same phenomenon, driven by the idea that generalizing and synthesizing findings from individual studies contributes to advancement of knowledge. However, this premise hinges on the underlying assumption, that the available scientific literature is representative for all conducted research (Song et al., 2010).

Contrary to this, researchers have pointed out for over half a century that results of published studies differ systematically from unpublished studies (Bakan, 1966; Bozarth & Roberts, 1972; Smart, 1964; Sterling, 1959). This discrepancy arises as the publication of a study often hinges on the direction or strength of its findings (Dickersin, 1990; Dickersin & Min, 1993) and is collectively known as *publication bias*. Especially in a publishing culture that prioritizes novelty and positive results (Nosek et al., 2012), many statistically nonsignificant studies end up in the “file-drawer” and never get published (Rosenthal, 1979).

The ramifications of publication bias are severe, culminating in inflated meta-analytical effect sizes (Franco et al., 2014; Stanley et al., 2021), heightened false-positive rate (Kicinski, 2014; Munafò & Flint, 2010), thereby increasing the risk of erroneous conclusions that may jeopardize the validity of research (Begg, 1994). These ramifications become especially relevant in the light of recent large-scale replication projects providing evidence for non-replicability of many psychological findings (Camerer et al., 2018; Ebersole et al., 2016, 2020; Klein et al., 2014, 2018; Open Science Collaboration, 2015). This underscores why publication bias identified as a major threat to replicable science (Munafò et al., 2017) and thus a considered as a significant contributor to the replication crisis (Renkewitz & Keiner, 2019). Given the myriad of issues associated with publication bias and its widespread impact, there has been considerable attention directed towards investigating methodologies to detect publication bias.

In this regard, there has been a great deal of research on publication bias detection techniques with numerous statistical methods developed over the past 50 years (Marks-Anglin & Chen, 2020). These statistical techniques can generally be classified into methodologies that operate with p -values and methodologies that are based on the relationship between effect size and sample size (Vevea et al., 2019). While both categories encompass highly sophisticated statistical techniques (CITATION?), a straightforward and frequently described method, that has been associated with publication bias, involves examining the correlation

between effect size and sample size. Additionally, this method encapsulates the central ideas of other approaches, such as Begg's rank correlation (Begg & Mazumdar, 1994), Egger's regression (Egger et al., 1997), and its proposed variants (for an overview see Song et al., 2010), all rooted in the relationship between effect size and sample size.

Reasoning of the n -ES correlation

The central tenets of the correlation of effect size and sample size as an indicator of publication bias originate from the concepts of the funnel plot and its asymmetry under the influence of publication bias that was introduced by Light & Pillemer (1984). When multiple studies investigate of common underlying effect, the empirical effect sizes (for example Cohen's d or Fisher- z transformed r) follow a normal distribution and fluctuate around the true effect size. Due to sampling error, the lower the sample sizes of individual studies, the less precision they exhibit to estimate the true effect size (i.e., larger standard error), leading to a larger variation around the true effect size. In the absence of publication bias this will result in a symmetric funnel shaped distribution (Light & Pillemer, 1984). However, when the publishing of studies is contingent on their statistical significance, the funnel plot will be asymmetric. As the statistical significance of p -values is jointly determined by the sample size (i.e., standard error of the test statistic) and effect size (i.e., test statistic), larger effect sizes attain statistical significance with smaller sample sizes, while smaller effect sizes necessitate larger sample sizes to be significant. Consequently, the negative correlation between effect size and sample size emerges because the threshold for the smallest effect sizes that is statistically significant decreases with increasing sample size (A. Linden et al., 2024). The correlation between effect size and sample size has been described and attributed to publication bias extensively in various research including psychology (Fritz et al., 2013; Kühberger et al., 2014; Levine et al., 2009), evolutionary biology and ecology (Jennions & Møller, 2002a; Jennions & Møller, 2002b; Møller & Jennions, 2001; Palmer, 1999), political science (Gerber et al., 2001) and educational research (R. E. Slavin et al., 2008; R. Slavin & Smith, 2009). Its prevalence across these disciplines highlights its role as a widely recognized and applied tool for the detection of publication bias.

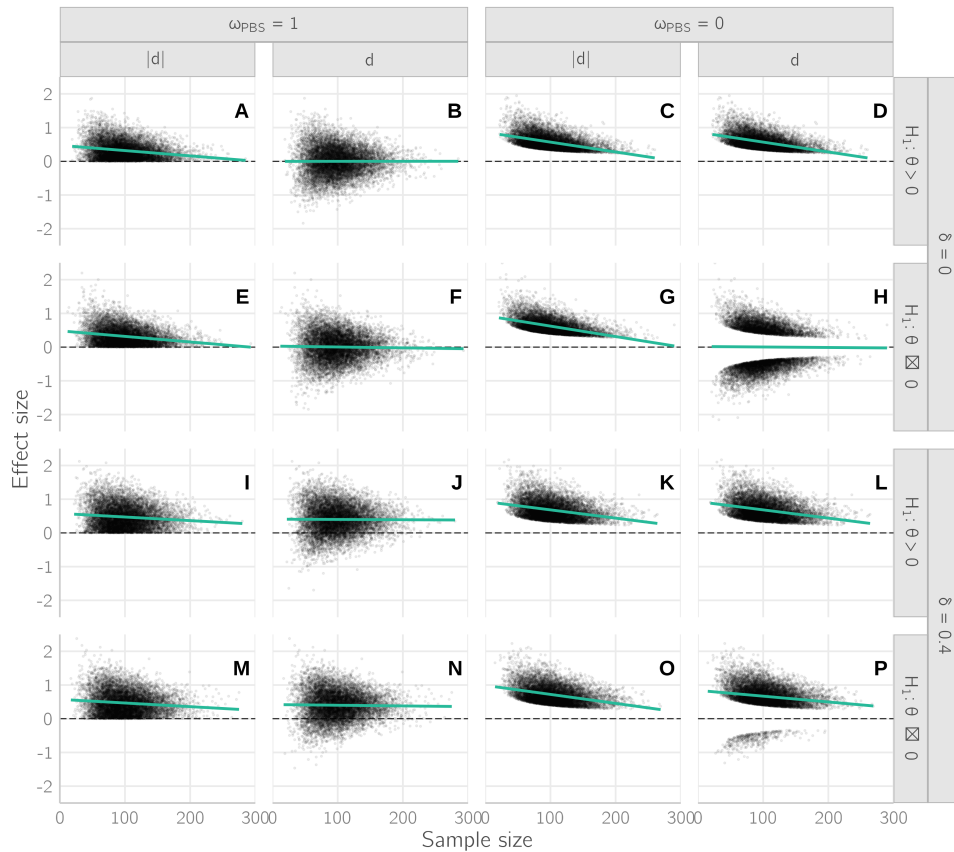
Methodological Concerns

Despite the significant attention and prevalent use of the effect size-sample size correlation in various research fields for detecting publication bias, coupled with its frequent acknowledgment as a valid indicator of such bias, there exist persisting methodological con-

cerns. As I will argue in the next section, these concerns have only been partially discussed and addressed in the existing literature and may compromise the validity of the interpretation of the correlation as an indicator of publication bias. To illustrate the inherent challenges of the effect size-sample size correlation as an indicator of publication bias, we simulated a set 10000 primary studies¹ on the same effect underlying effect and varied different parameters that contribute to its limitations (see figure 1). This includes the true effect size $\delta : \{0, 0.4\}$, the extent of publication bias $\omega_{PBS} : \{0, 1\}$ or how much less likely studies with non-significant p -values are compared to studies with significant results (in this extreme case either non-significant studies are not published at all, or there are no differences between non-significant and significant studies), the signedness of the effect size (d and $|d|$) and the type of hypothesis (directional $\mathcal{H}_1 : \theta > 0$ and non-directional $\mathcal{H}_1 : \theta \neq 0$)

Figure 1

n-ES Correlation in Simulated Data: Influence of Publication Bias, True Effect Size, and Signed vs. Unsigned Effects



Note:

Firstly, it is common practice to use unsigned effect sizes to estimate the n-es correla-

¹The selected number of primary studies aims to visually highlight its inherent limitations. The claims would still hold true even with a significantly lower number of primary studies (more representative of primary studies of meta-analysis e.g., 100), albeit with increased variability.

tion (Kühberger et al., 2014; Levine et al., 2009; e.g., R. Slavin & Smith, 2009; Weinerová et al., 2022). Whilst this is very common, it has only recently been acknowledged that the use of unsigned effect sizes can lead to a statistical artefact resulting in a small negative correlation, even in the absence of publication bias (A. Linden et al., 2024). As depicted in figure 1 (leftmost column compared to second leftmost column), the artificial correlation in absence of publication bias is most severe when the true effect is close to zero, as this condition leads to the most sign changes. Especially when considering that effect sizes in psychology are typically smaller than common benchmarks (Lovakov & Agadullina, 2021; Weinerová et al., 2022), and thus it is likely that the true effect sizes of psychological phenomena are often small, this exacerbates the problem of the statistical artifact.

If a negative correlation can emerge even in the absence of publication bias, this raises questions about the appropriate null hypotheses to test against, specifically, what correlation we would expect if publication bias is absent (A. Linden et al., 2024). There has been a long tradition in null hypothesis testing to use the nil null hypothesis (Cohen, 1994), which states that a population parameter is exactly zero. This is also very common in studies that have used the *n*-es correlation together with unsigned effect sizes (Kühberger et al., 2014; Levine et al., 2009; R. Slavin & Smith, 2009; Weinerová et al., 2022) and underscores a lack of thorough consideration for the potential falseness of this hypothesis in such cases. The determination of an appropriate null hypothesis for testing in these scenarios, however, remains uncertain.

Utilizing *signed* effect sizes may seem like a straightforward solution to the aforementioned problems, however, it introduces its own set of challenges. Especially, when researchers make non-directional hypothesis and where the true effect size is close to zero, the distribution of the signed effect sizes and sample size will be symmetrically hollowed out under the influence of publication bias. This symmetry (see Figure 1 H) will result in the correlation being zero, leading to a false negative - a failure to detect publication bias when it is present.

Apart from these more statistical challenges, there is also a more conceptual challenge.

-> *n*-es correlation somewhat misses the point of publication bias

- Fails to capture the point of publication bias -> depending on statistical significance -> effect size and sample size correlation only indirectly captures the censorship process of non-significant studies
- as figure shows, the non-linear relationship → critical test statistic value under which $p < \alpha$ nonlinear ->
- Harrer et al. (2021)
- **Questionable linearity assumption**

- Pearson correlation assumes that under publication bias \rightarrow linear relationship between effect size and sample size \rightarrow the higher the effect size the lower the required sample size for the effect to be significant and vice versa may give the (false) impression that this assumption holds
- But publication bias operates under statistical significance (which is most dominantly \rightarrow if p-value smaller than alpha threshold; CITATION) \rightarrow as figure shows, the non-linear relationship \rightarrow critical test statistic value under which $p < \alpha$ nonlinear
- Spearman correlation loosens the assumption of a linear effect in that the relationship has to be only strictly monotonic \rightarrow but still: this is not how publication bias operates

The present study

In summary the use of the effect size-sample size correlation as a method to assess publication bias suffers from various methodological challenges

Method

The **speec** Approach

Overview

- Simulation-based approach to estimate publication bias severity and correct potentially biased (inflated) effect sizes under present publication bias based on the joint distribution of effect size and sample size
 - Simulation of theoretical data -> joint distribution of effect size and sample size under marginal distributional assumptions -> Application of publication bias -> empirical kernel density estimation -> comparison of empirical and simulated data -> loss function
 - Implementation as an open source R package (alpha version) that is already available on GitHub: <https://github.com/jlschnatz/speec>
 - General steps
 - Simulate samples of joint distribution of effect size and sample size from marginal distributional assumptions
 - Application of publication bias
 - KDE for Simulated and Empirical Data
 - Compare Distributions using KL Divergence
 - Parameter Optimization via Simulated Annealing
- (1) Simulation of random samples from joint probability distribution of effect size and sample size
 - (2) Application of publication bias
 - (3) Kernel Density Estimation of drawn theoretical samples and the empirical empirical samples
 - (4) Computation of the divergence between the estimated probability of empirical against theoretical data
 - (5) Algorithmic optimization of bias and distributional parameters via simulated annealing

Simulation Framework

The marginal distribution for the total sample size n should be inherently modeled as a discrete distribution. Count data of this nature are commonly modeled using either a Poisson or Negative-Binomial distribution. In various psychological domains, sample size distributions often exhibit considerable variance and skewness (see for example Cafri et al., 2010; Marsza-

lek et al., 2011; Sassenberg & Ditrich, 2019; Shen et al., 2011; Szucs & Ioannidis, 2017). Considering this variability and skewness we opted for the Negative-Binomial distribution which can account for variance independently of the mean and thus handle overdispersed data effectively. We use the reparametrized mean-dispersion parametrization where the number of successes $r = \phi_n$ and the probability of success $p = \phi_n/(\mu_n + \phi_n)$ to model the study-specific total sample sizes n_i .

$$n_1, n_2, \dots, n_k \quad \text{where} \quad N \stackrel{\text{i.i.d.}}{\sim} \mathcal{NB}(\phi_n, \mu_n) \quad \text{for } i = 1, \dots, k \quad (1)$$

Concerning the marginal distribution of the effect size d , we assume a normal distribution with mean μ_d and variance σ_d^2 , where the effect size itself is assumed to originate from a common two-sample independent t -test design. To address the increasing precision in estimating the true effect size mean μ_d as sample size increases, contributing to the characteristic funnel shape of the effect size-sample size distribution, we compute the variance of the mean differences $\bar{x}_{i1} - \bar{x}_{i2}$, from which the effect sizes originate in this type of design. Subsequently, we derive a normalization factor γ_i by dividing each individual variance $\sigma_{\bar{x}_{i1} - \bar{x}_{i2}}^2$ with overall mean of those variances ensuring that $\bar{\gamma} = 1$.

$$\begin{aligned} \sigma_{\bar{x}_{i1} - \bar{x}_{i2}}^2 &= \sigma_d^2 / n_i \\ \gamma_i &= \frac{\sigma_{\bar{x}_{i1} - \bar{x}_{i2}}^2}{\sum_{i=1}^k \sigma_{\bar{x}_{i1} - \bar{x}_{i2}}^2 / k} \end{aligned} \quad (2)$$

With this normalization factor, the total variance of the individual variances of the individual variances is $\text{Var}(\gamma \cdot \sigma_d^2) = \sigma_d^2$. The study-specific effect sizes d_i are subsequently modeled as

$$d_1, d_2, \dots, d_k \quad \text{where} \quad D \sim \mathcal{N}(\mu_d, \gamma_i \cdot \sigma_d^2) \quad \text{for } i = 1, \dots, k \quad (3)$$

Definition and Application of Publication Bias

Following the simulation step of sampling k individual studies from the joint distribution of effect size and sample size given the distributional parameters, the subsequent step entails applying publication bias to these samples. As mentioned in the introduction, we operationalize publication bias in terms of the likelihood of a study being published conditional on the statistical significance of its results. Translated to this simulation setting we can calculate the two-tailed p -value of each individual study i from the random samples of effect size d_i and sample size n_i . We presume that individual studies i originate from a balanced

sample size design, where the group sample sizes n_{1i} and n_{2i} are defined as $n_i/2$ when the total sample size is even. If the total sample size is odd, the group sample sizes are determined as the ceilinged $\lceil n_i/2 \rceil$ and and floored $\lfloor n_i/2 \rfloor$ values, respectively. To calculate the p -value p_i of each simulated study, derived from t -value t_i

$$t_i = \left| \frac{d_i}{\sqrt{1/n_{1i} + 1/n_{2i}}} \right| \quad (4)$$

$$p_i = 2 \cdot P(t_i \mid df_i) \quad (5)$$

where $P(t_i, df_i)$ is the cumulative central t -distribution with degrees of freedom $df_i = n_{1i} + n_{2i} - 2$. Given each p -value p_i , publication bias is introduced by assigning each study i a weight

$$\omega_{\text{PBS}_i}(p_i) = \begin{cases} \omega_{\text{PBS}} & \text{for } p_i \geq \alpha \\ 1 & \text{otherwise} \end{cases} \quad (6)$$

given $\omega_{\text{PBS}} \in \mathbb{R} : 0 \leq \omega_{\text{PBS}} \leq 1$. This weight denotes the probability of a study i being selected conditional on the p -value and the type I error rate α . If $p_i \geq \alpha$, a publication bias weight ω_{PBS} is assigned, else the probability of a study being selected is 1, indicating no publication bias. We assume a fixed type I error rate for all simulated studies at the common threshold of $\alpha = .05$.

Following the computation of publication bias weight ω_{PBS_i} for each study i , the likelihood of a study being selected can be expressed as $\mathbb{P}(S_i = 1) = \omega_{\text{PBS}_i}$. Here, S_i serves as a binary indicator function, signifying whether study i is selected during the publication bias process.

$$S_i = \begin{cases} 0 & \text{study not selected} \\ 1 & \text{study selected} \end{cases} \quad (7)$$

Subsequently the two resulting subsets (d'_i, n'_i) and (d''_i, n''_i) from the initial random sample can be defined as

$$(d'_i, n'_i) = (d_i, n_i \mid S_i = 1) \quad \text{and} \quad (d''_i, n''_i) = (d_i, n_i \mid S_i = 0) \quad \text{for } i = 1, \dots, k. \quad (8)$$

Formulation as an Optimization Problem

In the subsequent phase, the statistical dissimilarity between the empirical meta-analytical data and the selected subset (d'_i, n'_i) of the simulated theoretical samples is evaluated by means of the Kullback-Leibler divergence (Kullback & Leibler, 1951). This assessment aims to quantify how closely the distributions of the theoretical samples align with the empirical data. To achieve this, the joint kernel density is estimated for both theoretical simulated and empirical data is estimated using a bivariate standard Gaussian kernel that is evaluated on a square grid, with a grid size of $n_{\text{grid}} = 2^7 + 1$ equidistant grid points in each dimension. The bounds of the square grid are determined based on the empirical meta-analytical data. To the define these bounds the maximum likelihood estimates for the parameters of the marginal distribution of effect size $(\hat{\mu}_d, \hat{\sigma}_d^2)$ and sample size $(\hat{\phi}_n, \hat{\mu}_n)$ are computed. Then, utilizing these estimates, the quantiles derived from inner 99th percentile ($p_1 = .005, p_2 = .995$) of the cumulative distribution are computed. Subsequently, the bounds are defined as the absolute minimum and maximum of these quantiles and then range of the empirical meta-analytical data , respectively.

$$[Q_n(p_1 | \hat{\phi}_n, \hat{\mu}_n), Q_d(p_1 | \hat{\phi}_n, \hat{\mu}_n)]$$

$$[Q_d(p_1 | \hat{\mu}_d, \hat{\sigma}_d^2), Q_d(p_1 | \hat{\mu}_d, \hat{\sigma}_d^2)]$$

Finally, KL-divergence is calculated from the kernel density estimates for both empirical and simulated data, providing a measure of their statistical distance.

$$D_{\text{KL}}(\hat{f}_e \parallel \hat{f}_t) = \sum_{u=1}^g \sum_{v=1}^g \hat{f}_e(u, v) \ln \left(\frac{\hat{f}_e(u, v)}{\hat{f}_t(u, v)} \right) \quad (9)$$

Algorithmic Parameter Optimization

- KL-divergence between empirical and simulated data, based on chosen parameter values for marginal distribution and publication bias severity serves as a loss function -> aim to find global minimum of loss function
- Which parameters are optimized -> distributional parameters $[\mu_d, \sigma_d^2, \phi_n, \mu_n]$ and publication bias parameter ω_{PBS}
- Simulated Annealing chosen as an optimization approach (Kirkpatrick et al., 1983)
- Metaheuristic enabling solving complex optimization problems (Husmann & Lange, 2022) -> Probabilistic optimization techniques to find global minimum of
- SA enables optimization of multimodal loss functions with a very high number of covariates than many other methods

- We use a version of SA as implemented in the *optimization* R Package (Husmann et al., 2017)
- Starting parameters for distribution parameters determined via maximum likelihood estimation, starting value for `w_pbs` set to 0.5
- Boundaries of parameter search space defined for all meta-analysis (all the same) -> defined so that search space goes beyond MLE estimates of meta-analysis (see table)

Secondary Data Description

- Secondary Data from A. H. Linden & Hönekopp (2021)

Statistical Analysis

All statistical analyses were performed using R (version 4.4.0, R. C. Team, 2023) in the RStudio Environment (version 2023.12.0.369, P. Team, 2023). Data and analysis scripts are made available members of Goethe University on the Local Infrastructure for Open Science (LIFOS) and the publicly on the Open Science Framework (OSF).

Regarding the hypotheses, where the publication bias parameter ω_{PBS} is the dependent variable ($\mathcal{H}_1, \mathcal{H}_2, \mathcal{H}_4$), beta regression as implemented in the *betareg* package (Zeileis et al., 2021) was used to analyse the data. This choice is motivated by the restriction of the parameter space for the publication bias to the standard unit interval, whereby non-normality, skewness and heteroscedasticity can anticipated (Cribari-Neto & Zeileis, 2010; ?). Beta regression is recognized for its adaptability in handling such deviations. We used a logit link for the mean parameter μ and a identity link for the dispersion parameter that is hold constant so that beta regression model can be described by

$$\begin{aligned} \omega_{\text{PBS}_i} &\sim \mathcal{B}(\mu_i, \phi) \\ \log\left(\frac{\mu_i}{1 - \mu_i}\right) &= x_i^\top \beta \end{aligned} \tag{10}$$

The independent variables for these three hypotheses are as follows: for \mathcal{H}_1 the independent variable was the Fisher z-transformed correlation coefficients for the correlation between effect size and sample size, where the transformation is defined as $z_r = 0.5 \ln\left(\frac{1+r}{1-r}\right)$. The independent variable for \mathcal{H}_2 is the difference $\Delta_{\hat{\mu}_d, \hat{\delta}}$ between the average effect size estimate of each meta-analysis $\hat{\delta}$ and the estimated mean parameter of the gaussian effect size distribution $\hat{\mu}_d$. Lastly, the independent variable is a binary indicator specifying the research synthesis type (normal meta-analysis or multisite replication studies), with multisite replica-

tion studies set as the reference level for regression. The beta-coefficients for these hypotheses were estimated using ML estimation with the *BFGS* optimizer.

To analyse \mathcal{H}_2 , we conducted an equivalence test using the Two One-Sided Tests (TOST) procedure to assess whether the presence of effects, large enough to be considered meaningful within specified equivalence bounds, can be rejected. To perform the tests, we utilized the *TOSTER* R package (Lakens & Caldwell, 2023), employing two-sample dependent Welch tests. The equivalence bounds against which the data is tested were defined by the smallest effect size of interest (SESOI).

We established the Smallest Effect Sizes of Interest (SESOI) for all four hypotheses through sensitivity power analyses, focusing on effect sizes reliably detectable within the constraints of available sample size resources for this secondary analysis (Lakens, 2014). . More specifically, we conducted three simulation-based (\mathcal{H}_1 , \mathcal{H}_3 , \mathcal{H}_4) and one analytical (\mathcal{H}_2) sensitivity power analysis to determine which effect sizes we have at least 80% power to detect, taking into account the constraints of the sample size and a fixed significance level $\alpha = .05$ (details see section [Power Analysis]). The resulting SESOI for each hypothesis is presented in Table 1. The SESOI for the equivalence hypothesis will define the equivalence bounds for the TOST procedure ($\Delta_L = -0.17$ and $\Delta_U = 0.17$).

Table 1

Smallest Effect Sizes of Interest for All Hypotheses

Hypothesis	SESOI	Unit
\mathcal{H}_1	1.28	OR
\mathcal{H}_2	0.59	OR
\mathcal{H}_3	0.17	raw
\mathcal{H}_4	1.28	OR

Note:

Power Analysis

The simulated-based sensitivity power analysis targeted a statistical power of 0.8 with a fixed significance level of $\alpha = .05$. Samples sizes varied across hypotheses: $n = 150$ for hypotheses 1 (only meta-analysis) and $n = 207$ for hypothesis 2 and 4 (both meta-analysis and multisite replication studies) and $n = 57$ (only multisite replication studies) for hypothesis 3. Predictor variables' distribution assumptions were specified as follows: Hypothesis 1 assumed a normal distribution ($\mu = -0.1; \sigma = 0.5$) for the Fisher z-transformed sample size effect

size correlation coefficients, with linear regression coefficient as the parameter of interest. Hypothesis 2 assumed equal means $\Delta = 0$ and a standard deviation $\sigma_{diff} = \sqrt{0.3^2 + 0.3^2}$ for the difference scores of $\hat{\mu}_d$ and $\hat{\delta}$, with the same assumptions for hypothesis 3, incorporating a quadratic regression coefficient as the parameter of interest. Beta-regression on ω_{PBS} in hypotheses 1, 3, and 4 involved simulations for different dispersion parameter $\phi = \{10, 20, 30\}$, as lower dispersion parameters result in reduced test power. We chose a conservative approach to define the SESOI for the parameters of interest ensuring a minimum power of 80% for the smallest simulated dispersion parameter $\phi = 10$. We set the SESOI for the parameters of interest more conservatively, ensuring a minimum power of 80% for the lowest dispersion parameter $\phi = 10$. The R code for the simulation-based sensitivity power analysis is available in the same directory as the preregistration.

Results

Variable Dispersion of ω_{PBS}

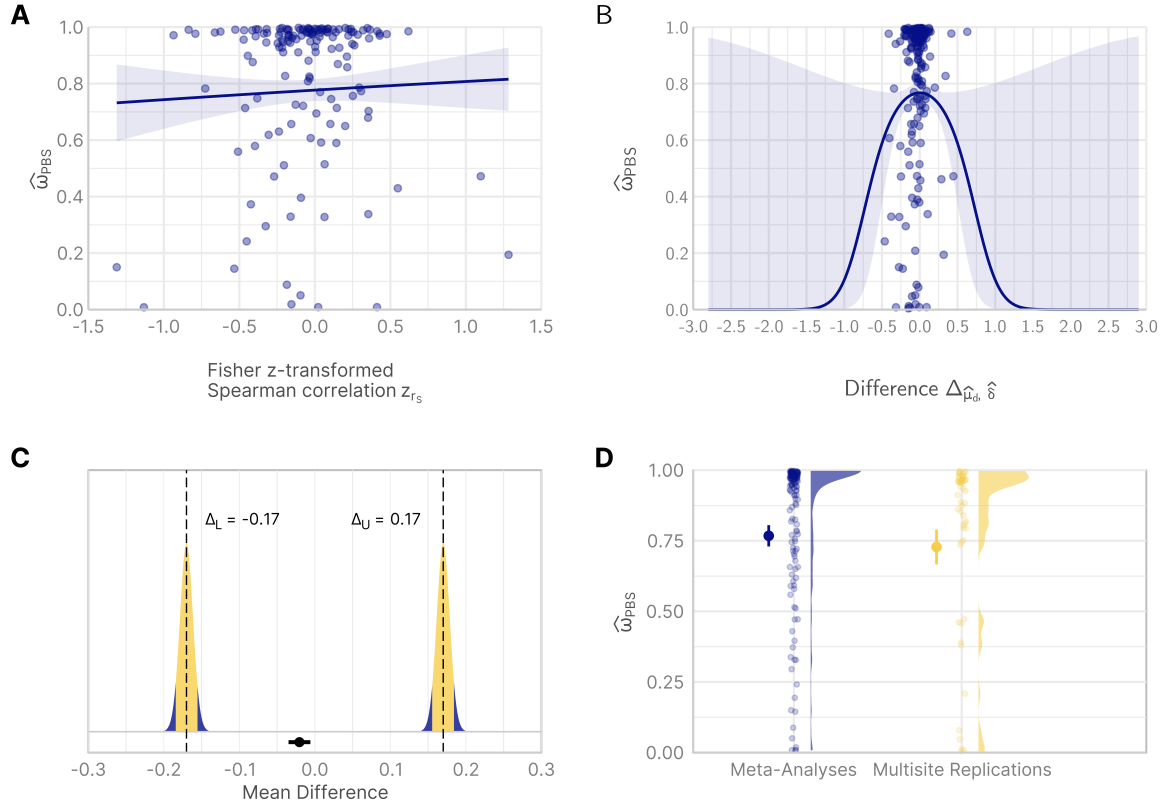
As an initial step, the assumptions made to determine the Smallest Effect Sizes of Interest (SESOI) for the four hypotheses were assessed. In the simulations-based sensitivity power analyses aimed at ascertaining the SESOIs (refer to the Appendix for details), three dispersion parameter conditions $\phi : \{10, 20, 30\}$ for the distribution of the publication bias parameter ω_{PBS} were simulated. Employing an intercept-only beta regression model with the complete dataset, the estimated dispersion parameter was $\hat{\phi} = 1.56$, 95% CI [1.27, 1.84], $SE = 0.15$, $z = 10.72$, $p < .001$. This finding contradicts our initial assumptions regarding the dispersion parameter's magnitude, rendering the interpretation of SESOIs for our hypotheses untenable. Consequently, it is appropriate to refrain from interpreting SESOIs in the subsequent analyses.

Confirmatory Results of the Predictions from the Hypotheses

Regarding hypotheses \mathcal{H}_1 , panel A of figure 2 depicts the relationship between the Fisher z-transformed Spearman correlation coefficients z_{r_s} of the association between effect size and sample size in each meta-analysis and the estimated publication bias parameter $\hat{\omega}_{\text{PBS}}$. The observed slope was marginally positive but statistically non-significant, $\log(OR) = 0.19$, $SE = 0.27$, $z = 0.69$, 95% CI [-0.34, 0.71], $p = 0.245$. This indicates, that lower correlation coefficients were not significantly linked to lower publication bias parameter values $\hat{\omega}_{\text{PBS}}$.

Concerning hypotheses II, panel B of figure 2 depicts the predicted values for the estimated publication bias parameter as a function of the difference between the average effect size estimate and the estimated mean parameter of Gaussian effect size distribution. The corresponding quadratic slope parameter was negative but not statistically significant, $\log(OR) = -3.34$, $SE = 1.84$, $z = -1.81$, 95% CI [-6.95, 0.27], $p = 0.070$.

In relation to hypothesis \mathcal{H}_3 , the depiction in panel C of Figure 2 presents the mean discrepancy between the estimated mean parameter of the Gaussian effect size distribution and the average effect size, along with its corresponding confidence interval. Additionally, the null t -distributions employed for executing the Two One-Sided Tests (TOST) procedure against the equivalence bounds $\Delta = (-0.17, 0.17)$ are illustrated. Both one-sided paired t -tests were statistically significant, lower t -value test $t(56) = 17.3$, $SE = 0.01$, $p < .001$. We additionally conducted a null hypothesis significance test to test the hypotheses that the true mean difference is exactly equal to zero. The mean difference significantly deviated from zero

Figure 2*Visual Summary of Results from Four Hypotheses*

Note. **A.** Estimated publication bias parameter as a function of Fisher z-transformed correlation coefficients. Fitted line represents the regression coefficients from the model and the 95% CI. **B.** Estimated publication bias parameter as a function of the difference between the estimated mean parameter and the average effect size. **C.** The mean difference between the estimated mean parameter and the average effect size and its 90% confidence interval compared against the null t-distribution for the lower and upper equivalence bounds. **D.** A comparison between the distribution of estimated publication bias parameter between normal meta-analyses and multisite replications. The pointrange indicates the marginal predicted values and the 95% confidence interval from the regression.

$M = -0.02$, 90% CI $[-0.03, -0.01]$ (effect size Hedge's $g_{rm} = -0.03$, 90% CI $[-0.05, -0.01]$), $t(56) = -2.36$, $SE = 0.01$, $p = 0.022$. This indicates that, despite the significant null hypothesis significance test, the difference was too small to be considered meaningful according to the equivalence range $\Delta = (-0.17, 0.17)$ of the equivalence test.

Finally, regarding hypothesis \mathcal{H}_4 , panel D shows a comparison between the estimated publication bias parameters for typical meta-analysis in comparison to multisite replication studies. Already descriptively, contrary to our expectation, the mean of the estimated publication bias values ω_{PBS} of the normal meta-analysis subset is greater than the mean of the multisite replication subset ($M_{MA} = 0.82$; $M_{MR} = 0.79$). In line with this, slope of the beta regression is non-significant, $\log(OR) = 0.45$, $SE = 0.18$, $z = -1.17$, 95% CI $[-0.56, 0.14]$, p

= 0.242, as also indicated by the overlappping confidence interval if the marginal means in panel D.

Diagnostic Checks

Table 2

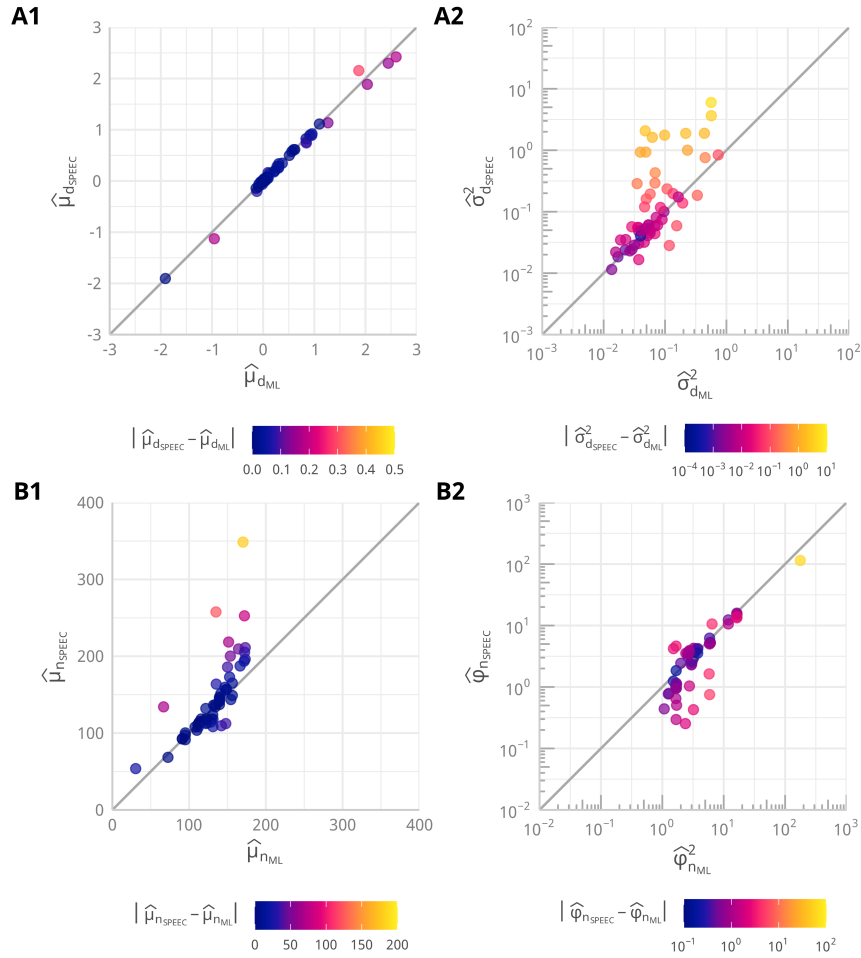
Pairwise Correlations: Differences Δ in Distributional Parameter Estimates between SPEEC and ML, Publication Bias Parameter ω_{PBS} and Meta-Analysis Size k

Variable	ω_{PBS}	$\Delta \mu_d$	$\Delta \sigma_d^2$	$\Delta \phi_n$	$\Delta \mu_n$
ω_{PBS}					
$\Delta \mu_d$	-0.44*** [-0.61, -0.23]				
$\Delta \sigma_d^2$	-0.37** [-0.56, -0.14]	0.67*** [0.54, 0.76]			
$\Delta \phi_n$	0.08 [-0.18, 0.33]	-0.06 [-0.31, 0.2]	-0.05 [-0.3, 0.21]		
$\Delta \mu_n$	0.04 [-0.22, 0.3]	0.14 [-0.12, 0.38]	0.05 [-0.22, 0.3]	-0.05 [-0.3, 0.21]	
k	-0.14 [-0.38, 0.12]	0.04 [-0.22, 0.29]	0.15 [-0.11, 0.39]	-0.18 [-0.41, 0.08]	0 [-0.25, 0.26]

Note: Test

Figure 3

Scatter Plot comparing the estimated Distributional Parameters via SPEEC vs. Maximum Likelihood



Note. **A1.** Comparison of estimated mean parameter μ_d from Gaussian effect size distribution. **A2.** Comparison of estimated variance parameter σ_d^2 of Gaussian effect size distribution. Axes and colorbar are log (base 10) transformed. **B1.** Comparison of mean parameter μ_n of Negative-Binomial sample size distribution **B2.** Comparison of dispersion parameter ϕ_n of Negative-Binomial sample size distribution. Axes and colorbar are log (base 10) transformed.

Discussion

Test

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Appendix

Appendix A: Power Analysis and SESOI

Appendix B: Open Science Statement

Appendix C: Regression Tables of Confirmatory Analyses

Table 3

Beta Regression Results for \mathcal{H}_1

Term	Estimate	CI (95%)	SE	z	p
Mean model component: μ					
Intercept	3.49 ^a	[2.79, 4.36]	0.11	10.93	< .001
z_{rs}	1.20 ^a	[0.71, 2.04]	0.27	0.69	.245
Precision model component: ϕ					
b_0	5.41 ^b	[3.73, 7.84]	0.19	8.89	< .001

Note: $LL = 129.28$, $MAE = 0.22$, $AIC = -252.57$, $BIC = -243.54$, $R^2 = 0.003$

^a OR ^b Identity coefficient

Table 4*Beta Regression Results for \mathcal{H}_2*

Term	Estimate	CI (95%)	SE	z	p
Mean model component: μ					
Intercept	3.30 ^a	[2.71, 4.03]	0.10	11.79	< .001
Quadratic	0.04 ^a	[9.58e-04,	1.84	-1.81	.035
$\Delta_{\hat{\mu}_d, \hat{\delta}}$		1.31]			
Precision model component: ϕ					
Intercept	4.85 ^b	[3.63, 6.47]	0.15	10.70	< .001

Note:

\$LL\$ = 164.25, \$MAE\$ = 0.23, \$AIC\$ = -322.51, \$BIC\$ = -312.51, \$R^2\$ = 0.026

^a \$OR\$^b Identity**Table 5***Two One-Sided Tests Result for \mathcal{H}_3*

Type	t	SE	df	p
NHST	-2.36	0.009	56	.022
TOST $\Delta < \Delta_L$	17.30	0.009	56	< .001
TOST $\Delta > \Delta_L$	-22.02	0.009	56	< .001

Note: NHST: Null Hypothesis Significance Test, TOST: Two One-Sided Test

Table 6*Beta Regression Results for \mathcal{H}_4*

Term	Estimate	CI (95%)	SE	z	p
Mean model component: μ					
Intercept	3.30 ^a	[2.67, 4.08]	0.11	11.07	< .001
Research	0.81 ^a	[0.57, 1.15]	0.18	-1.17	.879
Synthesis					
Type (MR)					
Precision model component: ϕ					
Intercept	4.79 ^b	[3.59, 6.38]	0.15	10.71	< .001

Note: MR: Multisite Replication; $LL = 163.31$, $MAE = 0.23$, $AIC = -320.62$, $BIC = -310.62$, $R^2 = 0.011$

^a OR ^b Identity coefficient