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**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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vorgelegt von

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Abstract

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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this, researchers have pointed out for over half a century that results of published studies differ systematically from unpublished studies (Bakan, 1966; Bozarth & Roberts, 1972; Smart, 1964; Sterling, 1959).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.

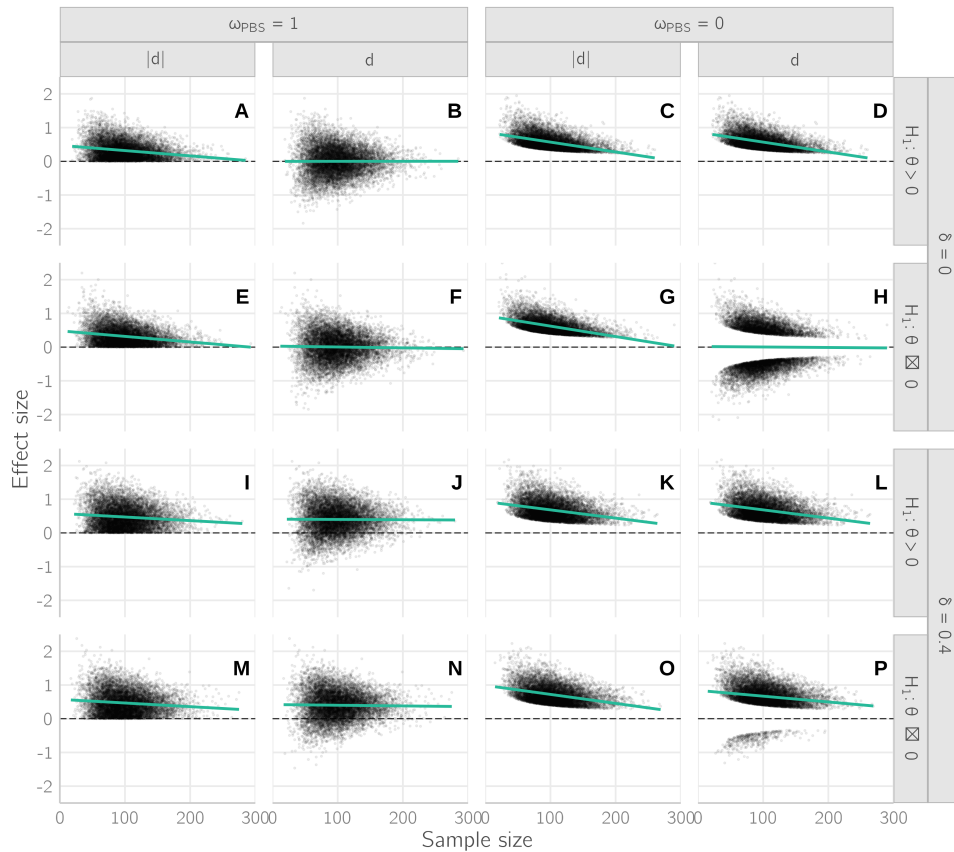
## Methodological Concerns

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 concerns. 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 studies<sup>1</sup> 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 correlation (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

<sup>1</sup>The 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.

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  $\rightarrow$  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 assumptions 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; Marszalek 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  $\mu_d$  and variance  $\sigma_d^2$ , 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  $\mu_d$  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  $\gamma_i$  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  $\alpha$ . If  $p_i \geq \alpha$ , a publication bias weight  $\omega_{\text{PBS}}$  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  $\omega_{\text{PBS}_i}$  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 Optimization via Simulated Annealing***

- 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  $\omega_{\text{PBS}}$
- 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  $\omega_{PBS}$  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; Smithson & Verkuilen, 2006). Beta regression is recognized for its adaptability in handling such deviations. We used a logit link for the mean parameter  $\mu$  and a identity link for the dispersion parameter that is hold constant so that beta regression model can be described by

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

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 replication 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), which was determined by an analytical sensitivity power analysis.

Equivalence bounds against which data is tested determined by the smallest effect size of interest -> in this case determined by analytical sensitivity power analysis -> based on effect sizes that we can reliably detect, considering the constraints imposed by the sample size resources available for this secondary analysis (Lakens, 2014)

### 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  $\omega_{\text{PBS}}$  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.

### SESOI

For all four hypotheses, we will establish the smallest effect size of interest (SESOI) based on effect sizes that we can reliably detect, considering the constraints imposed by the sample size resources available 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  $\Delta_U = 0.17$ ).



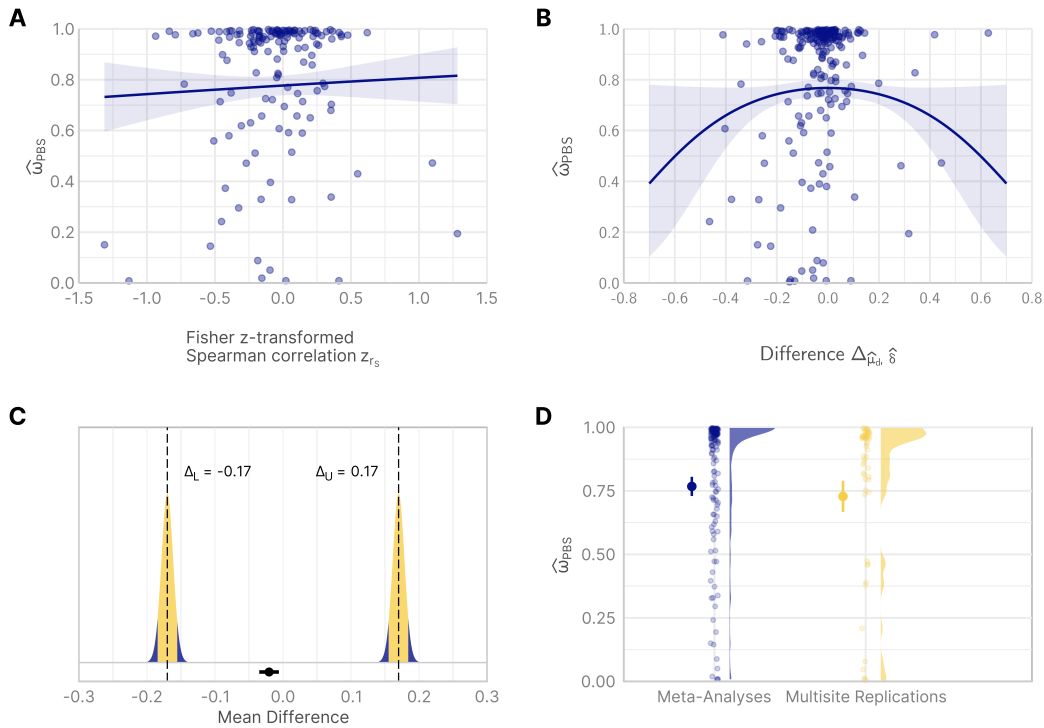
## Results

### Variable Dispersion of $\omega_{\text{PBS}}$

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  $\omega_{\text{PBS}}$  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.

**Figure 2**

*Visual Summary of Results from Four Hypotheses*



*Note.* **A.** Estimated publication bias parameter vs. Fisher z-transformed correlation coefficients. Fitted line: regression coefficients with 95

## Confirmatory Results of the Predictions from the Hypotheses

Regarding hypothesis  $\mathcal{H}_1$ , panel A of figure 2 depicts the relationship between estimated publication bias parameter  $\hat{\omega}_{\text{PBS}}$  and the Fisher  $z$ -transformed Spearman correlation coefficients  $z_{r_s}$  of the effect size sample size association in each meta-analysis. The observed slope was marginally positive, the sign of the coefficient was in the direction of the hypothesis, however statistically non-significant  $OR = 1.20$ , 95%  $CI$   $[0.77, \text{Inf}]$ ,  $SE = 0.27$ ,  $z = 0.69$ ,  $p = .245$ . This indicates, that lower values of  $z_{r_s}$  were not statistically significantly associated with lower publication bias parameter values  $\hat{\omega}_{\text{PBS}}$ . Additionally, to enhance the interpretability of the regression slope, we refitted the model with standardized values of  $z_{r_s}$  and computed the average marginal effects (Arel-Bundock, 2024). On average, for every standard deviation increase in the Fisher  $z$ -transformed correlation coefficient  $z_{r_s}$  ( $SD(z_{r_s}) = 0.31$ ), the model only predicted an increase of 1.11% in the publication bias parameter  $\hat{\omega}_{\text{PBS}}$ . In line with this, the general explanative power of the model as determined by the pseudo  $R^2$  (Ferrari & Cribari-Neto, 2004) was low,  $R^2 = 0.003$ .

Concerning  $\mathcal{H}_2$ , panel B of figure 1 depicts the relationship between estimated publication bias parameter as a function of the difference between the average effect size  $\hat{\delta}$  and the estimated mean parameter of the Gaussian effect size distribution  $\hat{\mu}_d$ . The corresponding estimated quadratic slope was negative as indicated by the predicted concave inverse u-shaped line and statistically significant at an  $\alpha$ -level of 5%,  $OR = 0.04$ , 95%  $CI$   $[0.00, 0.73]$ ,  $SE = 1.84$ ,  $z = -1.81$ ,  $p = .035$ . We again calculated the average marginal effect for improved interpretability. On average, for every standard deviation increase in  $\Delta_{\hat{\mu}_d, \hat{\delta}}$  ( $SD(\Delta_{\hat{\mu}_d, \hat{\delta}}) = 0.13$ ), the model only predicted an increase of -0.09% in the publication bias parameter  $\hat{\omega}_{\text{PBS}}$ . The overall explained variation of  $\omega_{\text{PBS}}$  by  $\Delta_{\hat{\mu}_d, \hat{\delta}}$  was low,  $R^2_{\text{pseudo}} = 0.026$ .

In relation to hypothesis  $\mathcal{H}_3$ , panel C of Figure 2 illustrate the mean difference  $\Delta_{\hat{\mu}_d, \hat{\delta}}$  between the estimated mean parameter of the Gaussian effect size distribution  $\hat{\mu}_d$  and the average effect size  $\hat{\delta}$ , along with its corresponding confidence interval. Additionally, the null  $t$ -distributions of the Two One-Sided Tests (TOST) against the equivalence bounds  $\Delta_{EQ} = (-0.17, 0.17)$  are illustrated. We only report the results of the  $t$ -test with the lower  $t$ -value in the main results as both tests must be significant to reject the null hypothesis (Lakens, 2017). Both one-sided paired  $t$ -tests were statistically significant,  $t(56) = 17.3$ ,  $SE = 0.01$ ,  $p < .001$ . This is also indicated by 90% confidence interval lying within the equivalence range in panel C of Figure 2. We additionally conducted an exploratory null hypothesis significance test to test the point hypothesis that the true mean difference of  $\Delta_{\hat{\mu}_d, \hat{\delta}}$  is exactly zero. The mean difference significantly deviated from zero  $M = -0.02$ , 90%  $CI$   $[-0.03, -0.01]$ ,  $t(56) = -2.36$ ,

$SE = 0.01$ ,  $p = 0.022$ . This indicates that, despite the significant null hypothesis significance test, the difference was too small to be considered meaningful according to the equivalence range  $\Delta_{EQ} = (-0.17, 0.17)$  of the equivalence test.

Finally, regarding hypothesis  $\mathcal{H}_4$ , panel D of Figure 2 illustrates the comparison between the estimated publication bias parameters for typical meta-analysis in comparison to multisite replication studies / registered reports. Already descriptively, contrary to our expectation that the estimated publication bias parameters for multisite replication studies (MR) would be greater (i.e., lower publication bias) than for regular meta-analysis (MA), the mean of the estimated publication bias values  $\omega_{\text{PBS}}$  of the regular meta-analysis subset is greater than the mean of the multisite replication subset ( $M_{\text{MA}} = 0.82$ ;  $M_{\text{MR}} = 0.79$ ). In line with this, slope of the beta regression was non-significant,  $OR = 0.81$ , 95% CI [0.61, Inf],  $SE = 0.18$ ,  $z = -1.17$ ,  $p = .879$ , as also indicated by the overlapping confidence interval of the predicted marginal means in panel D. Once more, we computed the average marginal effect to examine how the estimated publication bias parameter  $\hat{\omega}_{\text{PBS}}$  changes with the discrete shift from the reference level (multisite replication studies / registered reports) to typical meta-analysis, as predicted by the regression model, revealing a change of -3.93% in the opposing direction of the hypothesis.

## Diagnostic Evaluation of Parameter Estimation in SPEEC

As this study relies on empirical data to preliminarily assess the proposed SPEEC approach, the true values for the distributional parameters and the publication bias parameter are unknown. However, as discussed previously, publication bias is inherently absent by design in multisite replication studies and registered reports. Thus, the four distributional parameters  $(\mu_d, \sigma_d^2, \mu_n, \phi_n)$  within the SPEEC approach cannot be biased due to publication bias (especially the mean and variance of the effect size distribution). Leveraging this fact, we can use a subset of the data encompassing the multisite replication studies and registered reports for a diagnostic evaluation of the parameter estimation within the SPEEC approach. More specifically, we can derive Maximum Likelihood estimates for the distributional parameters to compare them with the corresponding values estimated by the SPEEC approach, anticipating approximate equivalence between the two approaches. This part of the analysis was not preregistered and conceived after the confirmatory analyses were conducted. Based on this comparative approach between ML and SPEEC, we formulated five diagnostic questions to assess parameter estimation:

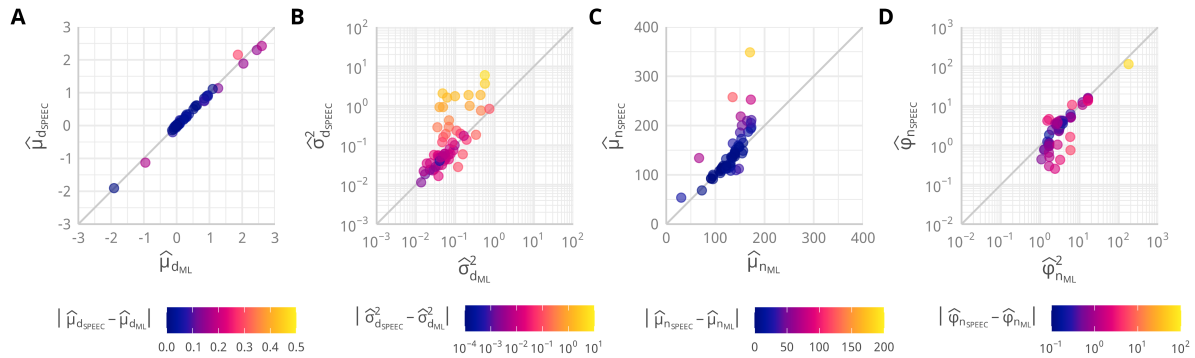
1. To what degree do the estimated distributional parameters differ between SPEEC and MLE?

2. How are the discrepancies in one parameter associated with those in the other distributional parameters across SPEEC and MLE? Specifically, does a consistency exist in the discrepancies between these parameters?
3. Does the discrepancy between SPEEC and MLE estimates of the distributional parameters correlate with the sample size of the multisite replication studies?
4. Is the discrepancy between SPEEC and MLE in the distributional parameters associated with sample size of multisite replication studies  $k$ ?
5. Is the discrepancy between SPEEC and MLE in the distributional parameters associated with the publication bias parameter  $\omega_{\text{PBS}}$ ?

The Maximum Likelihood estimates for the distributional parameters were obtained using the BFGS optimization algorithm. Additionally, the mean and median discrepancy between SPEEC and MLE were calculated to descriptively assess the average difference between the two estimation methods.

**Figure 3**

*Scatter Plot comparing the estimated Distributional Parameters via SPEEC and Maximum Likelihood*



*Note.* **A1.** Comparison of estimated mean parameter  $\mu_d$  from Gaussian effect size distribution. **A2.** Comparison of estimated variance parameter  $\sigma_d^2$  of Gaussian effect size distribution. Axes and colorbar are log (base 10) transformed. **B1.** Comparison of mean parameter  $\mu_n$  of Negative-Binomial sample size distribution **B2.** Comparison of dispersion parameter  $\phi_n^2$  of Negative-Binomial sample size distribution. Axes and colorbar are log (base 10) transformed.

Figure 3 provides a visual summary of the analysis comparing the distributional parameters estimated by the SPEEC method against those estimated by Maximum Likelihood Estimation (MLE). The diagonal line signifies perfect alignment between MLE and SPEEC estimates. Values below the diagonal indicate higher values for MLE compared to SPEEC, while values above the diagonal indicate the opposite.

Panel A reiterates the findings of the analysis of  $\mathcal{H}_3$ , suggesting a small discrepancy between the two methods in estimating the mean of the Gaussian effect size distribution,

$M(\Delta_{\mu_d}) = -0.03$ ,  $Mdn(\Delta_{\mu_d}) = -0.02$ . This discrepancy can be deemed practically negligible according to the equivalence test of  $\mathcal{H}_3$ . However, the other panels indicate contrasting outcomes.

Panel B reveals a systematic discrepancy in the estimation of the variance parameter of the Gaussian effect size distribution between SPEEC and MLE,  $M(\Delta_{\sigma_d^2}) = 0.59$ ,  $Mdn(\Delta_{\sigma_d^2}) = 0.11$ . Descriptively, this suggests that the variance was estimated to be greater in the SPEEC approach compared to MLE. Furthermore, this discrepancy increases in exponential looking trend and displays substantial heteroscedasticity with rising variance estimates from the MLE approach.

Similarly, Panel C also illustrates a systematic overestimation of the mean parameter of the Negative-Binomial sample size distribution by SPEEC in comparison to MLE ( $M(\Delta_{\mu_n}) = 138.04$ ,  $Mdn(\Delta_{\mu_n}) = 4.8$ ), which intensifies with higher mean parameter estimates from MLE.

Panel D shows that the SPEEC approach generally underestimates dispersion parameter of the sample size distribution in comparison to the ML estimate ( $M(\Delta_{\phi_n}) = -1$ ,  $Mdn(\Delta_{\phi_n}) = -0.18$ ) and also furthermore indicates a systematic relationship in the misestimation.

**Table 1**

*Pairwise Pearson Correlations between Difference of ML and SPEEC Distributional Parameters, Publication Bias Parameter and Meta-Analysis Size*

Variable	$\omega_{\text{PBS}}$	$ \Delta_{\mu_d} $	$ \Delta_{\sigma_d^2} $	$ \Delta_{\phi_n} $	$ \Delta_{\mu_n} $
$\omega_{\text{PBS}}$					
$ \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.* Computed  $p$ -values are corrected for multiple comparison using the correction by Benjamini & Hochberg (1995).  $|\Delta|$  is the absolute difference for each distributional parameter between SPEEC and MLE.

\* Significance \*\*\*  $p < .001$ ; \*\*  $p < .01$ ; \*  $p < .05$

Pairwise correlational analysis of the absolute absolute difference in the parameter estimates from the two estimation methods together with additional variables: publication bias parameter  $\hat{\omega}_{\text{PBS}}$  and the total number of primary replication studies  $k$  within each multisite replication project or registered report.

Table XX summarises the results of the correlational analysis.

Strong positive correlation between absolute difference between ML and SPEEC estimation of mean parameter and variance parameter of the Gaussian effect size distribution. This means that as the discrepancy between SPEEC and ML increases for mean parameter  $\mu_d$ , the discrepancy also increases for the variance parameter  $\sigma_d^2$  of the effect size distribution.

Moreover strong negative correlations between publication bias parameter  $\omega_{\text{PBS}}$  and divergence of between ML and SPEEC of the mean and variance parameter of the effect size distribution.

As the discrepancy between ML and SPEEC estimation increases for both mean  $|\Delta_{\mu_d}|$  and variance parameter  $|\Delta_{\sigma_d^2}|$ , the publication bias parameter  $\omega_{\text{PBS}}$  decreases, indicating more severe predicted publication bias.

Notably, no distributional parameter (divergence between ML and SPEEC) was significantly correlated with the total number of primary replications  $k$ .

No other significant correlations found.

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## 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 2**

*Beta Regression Results for  $\mathcal{H}_1$*

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

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

<sup>a</sup> OR

<sup>b</sup> Identity coefficient

**Table 3**

*Beta Regression Results for  $\mathcal{H}_2$*

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

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

<sup>a</sup> OR

<sup>b</sup> Identity

**Table 4***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 5***Beta Regression Results for  $\mathcal{H}_4$* 

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

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

<sup>a</sup>  $OR$

<sup>b</sup> Identity coefficient