
COMBINING SUPPORT FOR HYPOTHESES OVER HETEROGENEOUS STUDIES WITH BAYESIAN EVIDENCE SYNTHESIS: A SIMULATION STUDY

A PREPRINT

Thom Benjamin Volker 

Department of Methodology and Statistics
Utrecht University
Utrecht, 3584 CH
t.b.volker@uu.nl

Irene Klugkist 

Department of Methodology and Statistics
Utrecht University
Utrecht, 3584 CH
i.klugkist@uu.nl

2023-12-22

ABSTRACT

Scientific claims gain credibility by replicability, especially if replication under different circumstances and varying designs yields equivalent results. Aggregating results over multiple studies is, however, not straightforward, and when the heterogeneity between studies increases, conventional methods such as (Bayesian) meta-analysis and Bayesian sequential updating become infeasible. *Bayesian Evidence Synthesis*, built upon the foundations of the Bayes factor, allows to aggregate support for conceptually similar hypotheses over studies, regardless of methodological differences. We assess the performance of Bayesian Evidence Synthesis over multiple effect and sample sizes, with a broad set of (inequality-constrained) hypotheses using Monte Carlo simulations, focusing explicitly on the complexity of the hypotheses under consideration. The simulations show that this method can evaluate complex (informative) hypotheses regardless of methodological differences between studies, and performs adequately if the set of studies considered has sufficient statistical power. Additionally, we pinpoint challenging conditions that can lead to unsatisfactory results, and provide suggestions on handling these situations. Ultimately, we show that Bayesian Evidence Synthesis is a promising tool that can be used when traditional research synthesis methods are not applicable due to insurmountable between-study heterogeneity.

1 Introduction

In recent years, a meta-analytic way of thinking has been advocated in the scientific community. This approach is grounded in the belief that a single study is merely contributing to a larger body of evidence (e.g., [Asendorpf et al., 2016](#); [Cumming, 2014](#); [Goodman et al., 2016](#)). Such evidence gains credibility only by replicability of the findings with new data ([Schmidt, 2009](#)), because the ability to replicate research findings ensures that the findings represent *true* phenomena rather than artefacts. The replication crisis in the social sciences placed the importance of replication back on the research agenda and kickstarted multiple replication initiatives. Accordingly, multiple efforts aimed at fostering replication were undertaken, such as journals or journal sections devoted to replication studies (e.g., Royal Society Open Science, Registered Replication Reports, Journal of Personality and Social Psychology) and grant opportunities for replication studies (e.g., [NWO, 2020](#)). Moreover, multiple scholars legitimately emphasized the importance of replication studies for the future of science (e.g., [Baker, 2016](#); [Brandt et al., 2014](#); [Munafò et al., 2017](#)).

This renewed interest in replication was mostly directed toward studies that are highly similar, using a methodology and research design that mimics the original study as closely as possible (e.g., [Camerer et al., 2016, 2018](#); [Klein et al., 2014](#); [Nosek et al., 2021](#); [Open Science Collaboration, 2015](#)). These studies, commonly referred to as *direct*, *exact* or *close* replications, are primarily concerned with the statistical reliability of the results. As such, direct replications are tailored towards assessing whether or not the results of the initial study are due to chance. Agreement between the findings of a direct replication and the findings of the initial study increases the confidence in the accuracy of the original findings. However, direct replicability is a necessary but insufficient condition for making scientific claims. If

the results of the studies depend on methodological flaws, inferences from all studies will lead to suboptimal or invalid conclusions (Lawlor et al., 2017; Munafò and Smith, 2018).

Conceptual replications protect against placing too much confidence in findings that depend on methodological shortcomings. A conceptual replication primarily assesses the validity and generalizability of a study, by testing whether similar results can be obtained under different circumstances, or using different methods and operationalizations (Nosek et al., 2012). The rationale is that a phenomenon that can be observed in a variety of research settings is more likely to represent a *true* effect than a finding that replicates only under particular circumstances (Crandall and Sherman, 2016). Additionally, different methodologies used in different studies may have different strengths and weaknesses, that may all affect the conclusions drawn from the data. Combining evidence from multiple approaches mitigates the effect of these strengths and weaknesses, and thereby enhances the validity and the robustness of the final conclusion (Lawlor et al., 2017; Lipton, 2003; Mathison, 1988; Munafò and Smith, 2018; Nosek et al., 2012).

In the conventional framework of direct replications, combining evidence over studies is relatively straightforward, because established methods as (Bayesian) meta-analysis (Lipsey and Wilson, 2001; Sutton and Abrams, 2001) or Bayesian sequential updating (Schönbrodt et al., 2017) can be applied to aggregate the results. These methods pool the parameter estimates or effect sizes obtained in the individual studies (Cooper et al., 2009). Even if the studies are not identical, but still considerably similar in terms of study design and analysis methods, meta-analysis can be used, and moderators can be added to explain variability in the estimated effects. However, if the studies differ considerably with regard to research design, operationalizations of key variables or statistical models used, the parameter estimates or effect sizes will not be comparable. For example, the set of studies may include both experimental and correlational studies, varying covariates, or use different operationalizations of the same construct (e.g., different measurement instruments or different tasks). Under such circumstances, aggregating the varying effect sizes may not be meaningful, which renders the use of these conventional approaches infeasible.

To overcome this problem, Kuiper et al. (2013) proposed a new method called *Bayesian Evidence Synthesis* (*BES*). The key of the approach is to aggregate the *evidence* for a scientific theory or overarching hypothesis, rather than effect sizes, by aggregating Bayes factors obtained in individual studies. In every single study, a statistical hypothesis can be formulated that reflects the overall theory, but that accounts for characteristics of the data and research methodology unique to that study. The evidence for each study-specific hypothesis can be expressed using a Bayes factor (*BF*), rendering the relative support for the hypothesis of interest over some alternative hypothesis (Kass and Raftery, 1995). If the study-specific hypotheses reflect the same underlying theory, the effect sizes might be too heterogeneous to aggregate, but the support for the theory, quantified using Bayes factors, can be meaningfully combined. After all, each individual study provides a certain amount of evidence for or against this overarching theory. The evidence in each study can be aggregated into a single measure that reflects the support for the theory over all studies combined.

The popularity of Bayesian statistics in general (e.g., Lynch and Bartlett, 2019) and Bayesian hypothesis evaluation specifically (Van de Schoot et al., 2017) rapidly increased over recent years, especially in the context of individual studies. In contrast to the classical null hypothesis testing paradigm, Bayesian hypothesis evaluation enables researchers to compare potentially non-nested hypotheses with (multiple) equality- and/or order-constraints, by balancing the fit of the hypotheses to the data with the complexity of the candidate hypotheses (Klugkist et al., 2005; Hoijtink et al., 2019). Moreover, the Bayesian framework allows to quantify the amount of evidence from the data for or against the hypothesis of interest, rather than merely assessing whether the classical null hypothesis can be rejected (Wagenmakers et al., 2018). Yet, although the theoretical foundations of *BES* have been laid out (Kuiper et al., 2013), and the method has been applied successfully in substantive research (e.g., Kevenaar et al., 2021; Zondervan-Zwijnenburg et al., 2020a,b; Volker, 2022), methodological research on Bayesian evaluation of hypotheses has hardly been extended to the case in which evidence is combined over multiple studies. It is not obvious how research on Bayesian hypothesis evaluation within studies translates to the situation in which evidence is combined over multiple studies. As a consequence, the required conditions for adequate performance of *BES* are unknown.

In this paper, we assess the performance of *BES* in various scenarios relevant to applied researchers. In multiple simulations, we apply *BES* to aggregate the evidence for conceptually similar hypotheses that are evaluated using different statistical models, while varying the operationalizations of key variables in these hypotheses. We restrict the simulations to the evaluation of regression coefficients in three members of the generalized linear model family: ordinary least squares (OLS), logistic and probit regression, because of their widespread appearance in empirical research. Note, however, that the applicability of *BES* reaches beyond these methods: as long as conceptually similar hypotheses can be evaluated using Bayes factors, *BES* can be used to aggregate the results.

This flexibility may pose challenges to the application of *BES*. Different operationalizations of key variables may affect the complexities of the hypotheses evaluated in the individual studies. As Bayes factors directly depend on the complexities of the hypotheses, the risk is that the resulting amount of evidence largely depends on the specification of the hypotheses, rather than on their veracity. Our simulations assess how common procedures in data handling

affect the complexities of the hypotheses and the resulting performance of *BES*. Additionally, we investigate in which situations *BES* does not perform adequately, and provide suggestions on how such situations can be handled. Yet, before discussing the simulations, a detailed description of *BES* tailored to the specification of the simulation study is provided. The paper concludes with an extensive discussion of the implications of the simulations.

2 Aggregating Evidence with *BES*

Using *BES* to quantify the evidence for a scientific theory requires three building blocks. First, *BES* requires multiple studies on the same phenomenon, that all assess a conceptually similar hypothesis (Kuiper et al., 2013). Hence, in each study, a hypothesis must be formulated that reflects this scientific theory, but that also accounts for the specifics of the study, such as the empirical design, the analysis model and operationalizations of key variables. Second, these hypotheses are evaluated in each study separately. In the Bayesian framework, this can be done by calculating the Bayes factor for an hypothesis over an alternative hypothesis, using the posterior distribution of the model parameters. Third, the support for the theory in each individual study is aggregated using an updating procedure that renders the support for the theory over all studies combined. Each step is outlined in more detail in this section.

2.1 Informative hypotheses

When translating a conceptual hypothesis (i.e., the scientific theory) to a statistical hypothesis, researchers conventionally relied on the null hypothesis testing framework. In the context of regression models, this generally implies that researchers evaluate the null hypothesis, H_0 , stating that the regression coefficients β_k (where k is an index) equal zero, against an unconstrained alternative hypothesis, H_u , indicating that they can have any value. Several authors emphasized shortcomings of this approach, remarking that the ability to reject a null hypothesis is hardly illuminating, because the null is seldom (exactly) true (Cohen, 1994; Lykken, 1991) and rarely corresponds to the expectations researchers have (Cohen, 1990; Van de Schoot et al., 2011; Royall, 1997; Trafimow et al., 2018). Multiple researchers argued that scientific expectations can be better captured in an informative hypothesis (Hoijtink, 2012; Van de Schoot et al., 2011).

Informative hypotheses allow to formalize the expectations researchers have on the parameters of the statistical model (Hoijtink, 2012). Rather than expecting that the regression coefficients under evaluation are equal to zero, researchers may, for instance, expect that all are positive. In a regression model with three predictors, this expectation yields

$$H_i : \{\beta_1, \beta_2, \beta_3\} > 0.$$

However, informative hypotheses are not restricted to inequality-constrained hypotheses. An informative hypothesis might contain equality and inequality constraints between parameters and between combinations of parameters, as well as expectations regarding effect sizes or range constraints (Hoijtink et al., 2019). Combining these features yields great flexibility to researchers to formalize their theories, potentially resulting in fairly complex hypotheses. For example, the expectation that β_1 and β_2 are of the same size, while both are positive but smaller than β_3 , can be formalized as

$$H_{i'} : 0 < \{\beta_1 = \beta_2\} < \beta_3.$$

The flexibility with regard to specifying hypotheses is especially advantageous in the context of *BES*, where studies may differ in the operationalizations of key variables, and thus in the hypotheses that are evaluated.

2.2 Hypothesis evaluation using the Bayes factor

Within the Bayesian framework, the support for (informative) hypotheses can be expressed in terms of a Bayes factor (Kass and Raftery, 1995). Bayes factors require that a likelihood function (i.e., an analysis model for the data) is combined with a prior distribution, resulting in the posterior distribution of the parameters. The prior and posterior distribution can subsequently be used to calculate the Bayes factor.

2.2.1 Likelihood of the parameters

After formulating the hypotheses of interest, researchers need to specify a statistical model (i.e., a likelihood function) that allows to evaluate these hypotheses when analyzing the data (Lynch, 2007). A likelihood function quantifies the support in the data for each potential parameter value. This paper is restricted to OLS, logistic and probit regression, which are all part of the generalized linear model (GLM) family (note that there are many other models that fall under the GLM family). GLMs extend the linear regression model to deal with response variables that are assumed to follow a distribution from the exponential family. This is done by using a link function $g(\mu) = X\beta$, where X is an $n \times p$ matrix containing n observations' scores on p predictor variables including an indicator for the intercept, β is a $p \times 1$ vector containing the regression coefficients including intercept, and $\mu = E(y|X)$, where y is an $n \times 1$ vector with response values. The purpose of the link function is to model each observation's expected response $E(y|X)$ in terms of a linear combination of the predictor variables. Accordingly, the likelihood of the parameters given the data under

the generalized linear model is formalized as

$$L(\boldsymbol{\beta}, \phi | \mathbf{y}, \mathbf{X}) \equiv f(\mathbf{y} | \mathbf{X}, \boldsymbol{\beta}, \phi) = \prod_{i=1}^n f(y_i | \mathbf{x}_i, \boldsymbol{\beta}, \phi),$$

where i indexes each observation and ϕ denotes the variance or dispersion parameter.

One of the best known models in the class of GLMs is the linear regression model, which uses the identity link function, such that $g(\boldsymbol{\mu}) = \boldsymbol{\mu} = \mathbf{X}\boldsymbol{\beta}$. The likelihood function of the linear regression model is defined by

$$f(\mathbf{y} | \mathbf{X}, \boldsymbol{\beta}, \phi) = \prod_{i=1}^n \frac{1}{\sqrt{2\pi\sigma^2}} \exp\left\{-\frac{(y_i - \mathbf{x}_i\boldsymbol{\beta})^2}{2\sigma^2}\right\},$$

where $\phi = \sigma^2$. When the outcome \mathbf{y} is dichotomous rather than continuous, the binomial distribution can be used. The corresponding likelihood is defined by

$$f(\mathbf{y} | \mathbf{X}, \boldsymbol{\beta}) = \prod_{i=1}^n p_i^{y_i} (1 - p_i)^{1-y_i},$$

where y_i is either 0 or 1, and p_i is specified according to the logit link $g(\boldsymbol{\mu}) = \log\left(\frac{\boldsymbol{\mu}}{1-\boldsymbol{\mu}}\right) = \mathbf{X}\boldsymbol{\beta}$ for logistic regression, or probit link $g(\boldsymbol{\mu}) = \Phi^{-1}(\boldsymbol{\mu}) = \mathbf{X}\boldsymbol{\beta}$, where Φ^{-1} denotes the inverse cumulative distribution function of the standard normal distribution, for probit regression models. The dispersion parameter is omitted because it equals $\phi = 1$ for logistic and probit models.

2.2.2 Prior distribution

After specifying the analysis model, the prior distribution for the model parameters must be defined. Because the prior for an informative hypothesis can be obtained by truncating the unconstrained (or encompassing) prior (e.g., Klugkist et al., 2005), we first discuss the latter. The prior distribution reflects which parameter values are most likely before observing the data, and hence may be more or less informative depending on the amount of prior knowledge researchers possess. Although researchers have substantial freedom in specifying the prior distribution, inadequate choices have adverse consequences for hypothesis evaluation and comparison, rendering it a complicated task (O'Hagan, 1995). A practical solution to the specification of the prior for hypothesis evaluation, is to use a fractional unconstrained prior (O'Hagan, 1995) that protects against a subjective specification (Gu et al., 2018). This approach entails that a non-informative improper prior $p_u(\boldsymbol{\beta}, \phi)$ is updated with a small fraction b of the information in the data (i.e., the likelihood), resulting in a proper default prior distribution (e.g., Gu et al., 2018; Mulder et al., 2010), such that

$$p_u(\boldsymbol{\beta}, \phi | \mathbf{y}, \mathbf{X}, b) = \frac{p_u(\boldsymbol{\beta}, \phi) f(\mathbf{y} | \mathbf{X}, \boldsymbol{\beta}, \phi)_b}{\int \int p_u(\boldsymbol{\beta}, \phi) f(\mathbf{y} | \mathbf{X}, \boldsymbol{\beta}, \phi)_b \partial \boldsymbol{\beta} \partial \phi}.$$

Mulder and Olsson-Collentine (2019) proved that, under the linear model, updating a noninformative improper Jeffrey's prior with a fraction $b = \frac{p+1}{n}$ of the likelihood renders a Cauchy distribution (i.e., a Student \mathcal{T} prior distribution with 1 degree of freedom) for the regression coefficients,

$$p_u(\boldsymbol{\beta} | \mathbf{y}, \mathbf{X}, b) = \mathcal{T}(\hat{\boldsymbol{\beta}}, \hat{\Sigma}_{\boldsymbol{\beta}}/b, 1),$$

with location parameter $\hat{\boldsymbol{\beta}}$ (the maximum likelihood estimates of the regression coefficients) and scale parameter $\hat{\Sigma}_{\boldsymbol{\beta}}/b$ (the unbiased estimate of the covariance matrix of the regression coefficients over b). A discussion of the prior for the dispersion parameter is omitted, because nuisance parameters are integrated out when calculating the Bayes factor (Gu et al., 2018).

When the model under evaluation is not (multivariate) normal, but another GLM, obtaining an analytically tractable posterior may be difficult, regardless of the choice of prior (Gelman et al., 2004). Nevertheless, large-sample theory dictates that a fractional prior and posterior for the regression coefficients can be approximated by a multivariate normal distribution (Gelman et al., 2004). In this case, Gu et al. (2018) showed how a fractional prior can be obtained by updating a non-informative prior with a fraction $b = \frac{J}{n}$ of the likelihood. This approach renders an approximately normal prior distribution for the regression coefficients

$$p_u(\boldsymbol{\beta} | \mathbf{y}, \mathbf{X}, b) \approx \mathcal{N}(\hat{\boldsymbol{\beta}}, \hat{\Sigma}_{\boldsymbol{\beta}}/b).$$

with mean vector $\hat{\boldsymbol{\beta}}$ and covariance matrix $\hat{\Sigma}_{\boldsymbol{\beta}}/b$. Typically, J equals the number of independent constraints in all hypotheses of interest within a study, but different specifications are possible (for an elaborate discussion on appropriate values for J , see Gu et al., 2018; Hoijtink, 2021).

After specifying a prior for the unconstrained hypothesis, the encompassing prior approach can be used to obtain a prior for an informative hypothesis by truncating the unconstrained prior (e.g., [Klugkist et al., 2005](#); [Mulder et al., 2010](#); [Mulder, 2014](#)). The prior for an informative hypothesis is proportional to the region of the unconstrained prior that is in agreement with the constraints imposed by the hypothesis under consideration. That is, $p_i(\beta|\mathbf{y}, \mathbf{X}, b) \propto p_u(\beta|\mathbf{y}, \mathbf{X}, b) \mathbf{1}_{\mathcal{B}_i}$, where $\mathbf{1}_{\mathcal{B}_i}$ is an indicator for the admissible parameter space \mathcal{B}_i under hypothesis H_i ([Gu et al., 2018](#)).

2.2.3 Posterior distribution

Combining the likelihood of the parameters with the prior distribution yields the posterior distribution. The posterior quantifies the support for each parameter value after observing the data. Under the fractional prior approach, the unconstrained fractional prior is updated with the remaining fraction $1 - b$ of the likelihood. This approach yields the posterior distribution under an unconstrained hypothesis

$$P_u(\beta, \phi|\mathbf{y}, \mathbf{X}) = \frac{f(\mathbf{y}|\mathbf{X}, \beta, \phi)_{1-b} p_u(\beta, \phi|\mathbf{y}, \mathbf{X}, b)}{\int \int f(\mathbf{y}|\mathbf{X}, \beta, \phi)_{1-b} p_u(\beta, \phi|\mathbf{y}, \mathbf{X}, b) \partial\beta \partial\phi},$$

where $f(\mathbf{y}|\mathbf{X}, \beta, \phi)_{1-b}$ yields the fraction $1 - b$ of the likelihood. [Mulder and Olsson-Collentine \(2019\)](#) proved that updating a $\mathcal{T}(\hat{\beta}, \hat{\Sigma}_\beta/b, 1)$ prior distribution with the remaining fraction of the likelihood of the normal linear model yields a multivariate Student \mathcal{T} posterior distribution

$$P_u(\beta|\mathbf{y}, \mathbf{X}) = \mathcal{T}(\hat{\beta}, \hat{\Sigma}_\beta, n - k),$$

with location parameter $\hat{\beta}$, scale parameter $\hat{\Sigma}_\beta$ and $df = n - k$ degrees of freedom.¹

For GLMs other than the normal linear regression model, as well as hierarchical and structural equation models, [Gu et al. \(2018\)](#) discussed how the posterior distribution of the regression coefficients can be approximated by a multivariate normal distribution, which yields

$$P_u(\beta|\mathbf{y}, \mathbf{X}) \approx \mathcal{N}(\hat{\beta}, \hat{\Sigma}_\beta).$$

Because a truncated prior distribution under an informative hypothesis yields a density that is equal to zero at the truncated part of the distribution, and the likelihood function is unaffected by the hypothesis, the posterior distribution under an informative hypothesis also yields a truncated version of the unconstrained posterior. That is, the posterior distribution under hypothesis H_i yields $P_i(\beta|\mathbf{y}, \mathbf{X}) \propto P_i(\beta|\mathbf{y}, \mathbf{X}) \mathbf{1}_{\mathcal{B}_i}$.

2.2.4 Bayes factors

The Bayes factor comparing hypotheses H_i and $H_{i'}$ is defined as the ratio of the marginal likelihoods of these hypotheses, such that

$$BF_{ii'} = \frac{m(\mathbf{y}|\mathbf{X}, H_i)}{m(\mathbf{y}|\mathbf{X}, H_{i'})}.$$

This measure can be directly interpreted as the evidence in the data for hypothesis H_i versus the evidence in the data for hypothesis $H_{i'}$. As such, if $BF_{ii'} = 7$, hypothesis H_i obtains 7 times more support than hypothesis $H_{i'}$. The marginal likelihood of hypothesis H_i is defined as

$$\begin{aligned} m(\mathbf{y}|\mathbf{X}, H_i) &= \int \int_{\beta \in \mathcal{B}_i} P_i(\beta, \phi|\mathbf{y}, \mathbf{X}) \partial\beta \partial\phi \\ &= \int \int_{\beta \in \mathcal{B}_i} f(\mathbf{y}|\mathbf{X}, \beta, \phi) p_i(\beta, \phi|\mathbf{y}, \mathbf{X}) \partial\beta \partial\phi, \end{aligned}$$

which, when using the fractional prior and posterior, yields

$$m(\mathbf{y}|\mathbf{X}, H_i) = \int \int_{\beta \in \mathcal{B}_i} f(\mathbf{y}|\mathbf{X}, \beta, \phi)_{1-b} p_i(\beta, \phi|\mathbf{y}, \mathbf{X}, b) \partial\beta \partial\phi.$$

Whereas, in general, obtaining the marginal distribution is a complicated endeavor, the encompassing prior approach greatly simplifies this task ([Klugkist et al., 2005](#)). In fact, [Gu et al. \(2018\)](#) showed that when comparing an informative

¹ Again, a discussion of the posterior distribution of the nuisance parameters is omitted, because these are integrated out when calculating the Bayes factors.

hypothesis H_i with an unconstrained hypothesis H_u , the ratio of the two marginal likelihoods boils down to

$$\begin{aligned} BF_{iu} &= \frac{\int \int_{\beta \in B_i} f(\mathbf{y} | \mathbf{X}, \beta, \phi)_{1-b} p_i(\beta, \phi | \mathbf{y}, \mathbf{X}, b) \partial \beta \partial \phi}{\int \int f(\mathbf{y} | \mathbf{X}, \beta, \phi)_{1-b} p_u(\beta, \phi | \mathbf{y}, \mathbf{X}, b) \partial \beta \partial \phi} \\ &= \int \int_{\beta \in B_i} \frac{f(\mathbf{y} | \mathbf{X}, \beta, \phi)_{1-b}}{\int \int f(\mathbf{y} | \mathbf{X}, \beta, \phi)_{1-b} p_u(\beta, \phi | \mathbf{y}, \mathbf{X}, b) \partial \beta \partial \phi} \frac{p_u(\beta, \phi | \mathbf{y}, \mathbf{X}, b) \mathbf{1}_{B_i}}{\int \int_{\beta \in B_i} p_u(\beta, \phi | \mathbf{y}, \mathbf{X}, b) \partial \beta \partial \phi} \partial \beta \partial \phi \\ &= \frac{\int \int_{\beta \in B_i} P_u(\beta, \phi | \mathbf{y}, \mathbf{X}) \partial \beta \partial \phi}{\int \int_{\beta \in B_i} p_u(\beta, \phi | \mathbf{y}, \mathbf{X}, b) \partial \beta \partial \phi}. \end{aligned}$$

That is, the constrained prior can be factored into an unconstrained prior, an indicator for the admissible parameter space under hypothesis H_i and the normalizing marginal constrained prior distribution. This yields a marginal prior and marginal posterior distribution, integrated over the parameter space in line with the hypothesis.

In the current definition of the Bayes factor, however, the prior distribution is centered at the maximum likelihood estimates of the regression coefficients, as implied by the fraction of information from the likelihood. This is problematic when testing inequality-constrained hypotheses (e.g., $H_i : \beta > 0$ versus $H_{i'} : \beta \leq 0$), because one of the two hypotheses obtains a larger prior plausibility unless the estimated coefficient lies exactly on the boundary of the hypotheses (e.g., $\hat{\beta} = 0$). When using a symmetric prior, balancing the unconstrained prior on the boundary of the hypotheses ensures that both hypotheses receive the same *a priori* support. Additionally, when testing equality-constrained hypotheses, [Jeffreys \(1961\)](#) remarked that centering the prior around the hypothesized value is substantively desirable, because the *a priori* expectation is that the estimate should be close to the hypothesized value, otherwise there is little merit in testing precisely this hypothesis. Based on these considerations, several scholars advocated to center the prior distribution around the focal point of interest when calculating the Bayes factor ([Gu et al., 2018](#); [Mulder, 2014](#); [Zellner and Siow, 1980](#); [Mulder and Gu, 2021](#)). This procedure renders the *adjusted* fractional Bayes factor, which is defined by

$$BF_{iu} = \frac{\int \int_{\beta \in B_i} P_u(\beta, \phi | \mathbf{y}, \mathbf{X}) \partial \beta \partial \phi}{\int \int_{\beta \in B_i} p_u^*(\beta, \phi | \mathbf{y}, \mathbf{X}, b) \partial \beta \partial \phi},$$

where the prior for the coefficients is centered around the hypothesized values. After integrating out the nuisance parameters, this yields

$$BF_{iu} = \frac{\int_{\beta \in B_i} P_u(\beta | \mathbf{y}, \mathbf{X}) \partial \beta}{\int_{\beta \in B_i} p_u^*(\beta | \mathbf{y}, \mathbf{X}, b) \partial \beta} = \frac{f_i}{c_i}.$$

Consequently, BF_{iu} is defined as the proportion of the posterior distribution that is in line with hypothesis H_i , denoted fit (f_i), over the proportion of the adjusted unconstrained prior distribution that is in line with hypothesis H_i , denoted complexity (c_i). As such, the Bayes factor serves as Occam's razor, by balancing the fit of the data to the hypothesis (i.e., the marginal posterior) with the complexity of the hypothesis.

This formulation of the Bayes factor is relatively easy to compute for both equality and inequality constrained informative hypotheses. When the hypothesis of interest only contains equality constraints, one can make use of the Savage-Dickey density ratio ([Dickey, 1971](#)), which yields

$$BF_{i_0 u} = \frac{f_{i_0}}{c_{i_0}} = \frac{P_u(\beta = \mathbf{B}_{i_0} | \mathbf{y}, \mathbf{X})}{p_u^*(\beta = \mathbf{B}_{i_0} | \mathbf{y}, \mathbf{X}, b)},$$

which is the ratio of the densities of the posterior $P_u(\beta = \mathbf{B}_{i_0} | \mathbf{y}, \mathbf{X})$ and adjusted prior $p_u^*(\beta = \mathbf{B}_{i_0} | \mathbf{y}, \mathbf{X}, b)$, evaluated at the location of the hypothesized values. When the hypothesis of interest contains merely inequality constraints, the Bayes factor is expressed as the ratio between the volume of the posterior that is in line with the hypothesis and the volume of the prior that is in line with the hypothesis, such that

$$BF_{i_1 u} = \frac{f_{i_1}}{c_{i_1}} = \frac{\int_{\beta \in B_{i_1}} P_u(\beta | \mathbf{y}, \mathbf{X}) \partial \beta}{\int_{\beta \in B_{i_1}} p_u^*(\beta | \mathbf{y}, \mathbf{X}, b) \partial \beta}.$$

In the most complex scenario where the hypothesis of interest contains both equality and inequality constraints, the Bayes factor equals

$$BF_{i_{01} u} = \frac{f_{i_{01}}}{c_{i_{01}}} = \frac{P_u(\beta_{i_0} = \mathbf{B}_{i_0} | \mathbf{y}, \mathbf{X})}{p_u^*(\beta_{i_0}^* = \mathbf{B}_{i_0} | \mathbf{y}, \mathbf{X}, b)} \times \frac{\int_{\beta_{i_1} \in B_{i_1}} P_u(\beta_{i_1} | \beta_{i_0} = \mathbf{B}_{i_0}, \mathbf{y}, \mathbf{X}) \partial \beta}{\int_{\beta_{i_1} \in B_{i_1}} p_u^*(\beta_{i_1} | \beta_{i_0} = \mathbf{B}_{i_0}, \mathbf{y}, \mathbf{X}, b) \partial \beta},$$

where β_{i_0} and β_{i_1} are the equality and inequality constrained parameters under the hypothesis, respectively (Gu et al., 2018). Additionally, the Bayes factor between two informative hypotheses is easy to calculate due to transitivity of the Bayes factor. That is, both informative hypotheses can be expressed with reference to the unconstrained hypothesis, such that comparing two informative hypotheses H_i and $H_{i'}$ yields

$$BF_{ii'} = \frac{BF_{iu}}{BF_{i'u}} = \frac{f_i/c_i}{f_{i'}/c_{i'}}.$$

A special case of an alternative informative hypothesis is a complement hypothesis. The complement of hypothesis H_i implies “not H_i ” and reflects all possible parameter values outside the constraints imposed by the hypothesis under consideration. Evaluating a single informative hypothesis against its complement yields

$$BF_{ic} = \frac{BF_{iu}}{BF_{cu}} = \frac{f_i/c_i}{(1-f_i)/(1-c_i)}.$$

Because a hypothesis and its complement cover mutually exclusive regions of the entire parameter space, evaluating against the complement is more powerful than evaluating against an unconstrained hypothesis (Klugkist and Volker, 2023).

When comparing a set of hypotheses, it is generally insightful to translate the Bayes factors into posterior model probabilities (*PMPs*; Kass and Raftery, 1995). The posterior model probabilities quantify the support for each hypothesis on a common scale on the interval (0, 1), which facilitates the interpretation, and allows to take prior knowledge into account through prior model probabilities. The posterior model probabilities for hypothesis H_i , with $i = 1, 2, \dots, m$ (with m the number of hypotheses under consideration), are given by

$$PMP(H_i) = \frac{\pi_i BF_{iu}}{\sum_{i'=1}^m \pi_{i'} BF_{i'u}},$$

where π_i indicates the prior model probability of hypothesis H_i , and render the relative plausibility of a finite set of hypotheses after observing the data.

2.3 Bayesian Evidence Synthesis

The procedure of quantifying the support for a hypothesis using Bayes factors can be extended to express the support for a hypothesis or scientific theory over multiple studies (Kuiper et al., 2013). When all considered studies assess an overarching theory, the Bayes factor in each study renders a certain degree of support for or against this theory. Using *Bayesian Evidence Synthesis* (*BES*), the aggregated support for the theory is obtained by updating the model probabilities with each new study. In this sense, *BES* proceeds sequentially. After conducting the first study, the support for the hypothesis of interest can be expressed as a posterior model probability. This first posterior model probability can be used as prior model probability for the second study. Combined with the support for the hypothesis in the second study, this renders a posterior model probability that reflects the total amount of evidence for or against the hypothesis in the first and second study combined. Hence, the posterior model probabilities after study t can be used as prior model probabilities in study $t+1$ (Kuiper et al., 2013). Independent of the order of the studies, this process can be repeated for a total of T studies, which yields

$$PMP(H_i)^{(T)} = \frac{\pi_i^{(0)} \prod_{t=1}^T BF_{iu}^{(t)}}{\sum_{i'=1}^m \pi_{i'}^{(0)} \prod_{t=1}^T BF_{i'u}^{(t)}},$$

where $\pi_i^{(0)}$ indicates the prior model probability for hypothesis H_i before any study has been conducted. It can be reasonable to consider all hypotheses equally likely before observing any data, which renders equal prior model probabilities for all hypotheses (i.e., $\pi_i^{(0)} = 1/m$). With equal prior model probabilities, the product of Bayes factors contains the same information as the aggregated posterior model probabilities. Ultimately, the final posterior model probabilities reflect the relative support for the theory over all studies.

3 Simulations

Although *BES* allows to combine support for a hypothesis that is tested differently over studies, this approach also poses challenges. Bayes factors do not only depend on the fit of the data to the hypothesis, but also on the complexity of the hypothesis. Conceptually similar hypotheses with distinct operationalizations in different studies can have different complexities. Consider a hypothesis that implies a positive relationship between a construct of interest, measured by multiple indicators, and an outcome. Whereas some researchers may combine multiple indicators into a single scale score, others may analyze the effects of the separate indicators. Similarly, researchers may categorize

continuous variables. Such variation in measurement and data handling results in hypotheses with different complexities, affecting the resulting Bayes factors. The upcoming section presents the first set of eight simulations. We briefly assess how statistical power affects the performance of *BES* in terms of the aggregated support for the true hypothesis, and then zoom in on how different choices in data handling, resulting in conceptually similar hypotheses with different complexities, affect *BES*. We first outline the general set-up, after which we discuss the specifics per pair of simulations. The methodological details and the results of the respective simulation(s) are integrated to enhance the readability. Hereafter, a second set of simulations zooms in on those scenarios in which *BES* performs inadequately. The simulations are conducted in R ([R Core Team, 2021](#), Version 4.1.0; a full simulation archive containing all R-scripts and results is available on GitHub, see Data Availability Statement).

3.1 Simulations part 1 - assessing the effect of complexity

In part one of the simulations, eight simulation conditions are assessed. The focus of these simulations is mainly on how the complexity of the candidate hypotheses affects the performance of *BES*, while simultaneously varying the sample and effect sizes. In all simulations, we consistently apply *BES* on a collection of three studies that all assess the same hypothesis. The hypotheses are varied in a pairwise manner between simulation conditions, such that a comparison is made between conceptually similar hypotheses that have different complexities, due to different choices in data handling. Such data handling is performed *after* generating the data (we discuss the operationalizations of hypotheses per simulation condition in subsequent sections). To demonstrate that *BES* is applicable despite between-study heterogeneity, data representing the three studies is generated and analysed with three statistical models: ordinary least squares (OLS), logistic and probit regression. Within each of the eight simulations, we consider a common set of sample sizes ($n \in \{25, 50, 100, 200, 400, 800\}$) and effect sizes ($R^2 \in \{0.02, 0.09, 0.25\}$), corresponding to small, medium and large effects as defined by [Cohen, 1988](#), that are equivalent for the three studies. Whereas the conventional R^2 is used for studies generated with OLS regression, McKelvey and Zavoina's $R^2_{M&K}$ ([1975](#)) is used for studies generated with logistic and probit models, due to its close empirical resemblance of the conventional R^2 ([Hagle and Mitchell, 1992; DeMaris, 2002](#)). In each study, the Bayes factor for the hypothesis of interest is calculated using the R-package BFPack ([Mulder et al., 2021](#)), after which *BES* is applied with equal initial prior model probabilities for the hypotheses under consideration. The performance of *BES* is assessed over 1000 iterations for each combination of effect and sample size. Performance is measured in terms of the aggregated Bayes factors of the hypothesis of interest against an unconstrained hypothesis (on a log-scale to mitigate scaling issues), and in terms of posterior model probabilities against both an unconstrained hypothesis and a complement hypothesis.²

In all simulation conditions, data is generated based on the same relationship between predictors and outcome, regardless of the model used to generate the data. Each study consists of an outcome \mathbf{y} , that can be continuous or dichotomous, and $p = 6$ predictor variables in the $n \times p$ matrix \mathbf{X} , with columns $\mathbf{x}_1, \mathbf{x}_2, \dots, \mathbf{x}_6$. The weights of the relationship between \mathbf{X} and \mathbf{y} is captured in the column vector $\mathbf{a} = [0 \ 1 \ 1 \ 1 \ 2 \ 3]'$, such that the population regression coefficients are specified as $\beta_1 = 0$, $\beta_2 = \beta_3 = \beta_4$, $\beta_5 = 2\beta_{2,3,4}$ and $\beta_6 = 3\beta_{2,3,4}$. All predictor variables are normally distributed with zero mean vector $\boldsymbol{\mu}$ and covariance matrix $\boldsymbol{\Sigma}$, with common covariance $\rho_{k,k'} = 0.3$, such that

$$\boldsymbol{\mu} = \begin{bmatrix} 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{bmatrix}, \quad \boldsymbol{\Sigma} = \begin{bmatrix} 1 & & & & & \\ 0.3 & 1 & & & & \\ 0.3 & 0.3 & 1 & & & \\ 0.3 & 0.3 & 0.3 & 1 & & \\ 0.3 & 0.3 & 0.3 & 0.3 & 1 & \\ 0.3 & 0.3 & 0.3 & 0.3 & 0.3 & 1 \end{bmatrix}.$$

Consequently, the regression coefficients are defined by

$$\boldsymbol{\beta} = \mathbf{a} \sqrt{\frac{\text{Var}(\hat{\mathbf{y}})}{\mathbf{1}'(\mathbf{a}\mathbf{a}' \odot \boldsymbol{\Sigma})\mathbf{1}}},$$

where $\text{Var}(\hat{\mathbf{y}}) = \text{Var}(\mathbf{X}\boldsymbol{\beta})$ is defined as a function of the effect size,³ $\mathbf{1}$ is a p -dimensional one vector, $\mathbf{1}'$ is its transpose and \odot indicates the Hadamard (element-wise) product. The population-level regression coefficients are displayed in Table 1 for all models and effect sizes (rounded at three decimals, which may slightly distort their displayed relative sizes). Continuous outcomes are drawn from a normal distribution

$$\mathbf{y}_{\text{OLS}} \sim \mathcal{N}(\mathbf{X}\boldsymbol{\beta}, (1 - R^2)\mathbf{I}),$$

²Note that Bayes factors against a complement hypothesis become infinitely large when the fit of the hypothesis tends to 1.

³ $\text{Var}(\hat{\mathbf{y}}_{\text{OLS}}) = R^2$ under OLS when \mathbf{y} has a variance of $\sigma_y^2 = 1$, while $\text{Var}(\hat{\mathbf{y}}_{\text{logistic}}) = \frac{R^2 \pi^2}{1 - R^2}$ and $\text{Var}(\hat{\mathbf{y}}_{\text{probit}}) = \frac{R^2}{1 - R^2}$ for logistic and probit regression, respectively.

Table 1: Population-level regression coefficients for ordinary least squares (OLS), logistic and probit regression, given effect sizes of $R^2 \in \{0.02, 0.09, 0.25\}$.

R^2	OLS				Logistic				Probit			
	β_1	$\beta_{2,3,4}$	β_5	β_6	β_1	$\beta_{2,3,4}$	β_5	β_6	β_1	$\beta_{2,3,4}$	β_5	β_6
0.02	0.000	0.026	0.051	0.077	0.000	0.047	0.094	0.141	0.000	0.026	0.052	0.078
0.09	0.000	0.054	0.109	0.163	0.000	0.103	0.207	0.310	0.000	0.057	0.114	0.171
0.25	0.000	0.091	0.181	0.272	0.000	0.190	0.380	0.570	0.000	0.105	0.209	0.314

with mean vector $\mathbf{X}\beta$, common residual variance $\sigma_\epsilon^2 = 1 - R^2$ and \mathbf{I} an n -dimensional identity matrix. Binary outcomes are drawn from a Bernoulli distribution

$$\mathbf{y}_{\text{logistic}} \sim \mathcal{B}\left(\frac{\exp\{\mathbf{X}\beta\}}{1 + \exp\{\mathbf{X}\beta\}}\right), \text{ and } \mathbf{y}_{\text{probit}} \sim \mathcal{B}(\Phi\{\mathbf{X}\beta\}),$$

with Φ indicating the cumulative normal distribution.⁴

3.1.1 Simulation 1 and 2

In simulation 1 and 2, we assess the consequences of including one underpowered study in the set of three for the performance of *BES*, while simultaneously assessing the aforementioned variations in sample and effect size. Simulation 1 completely adheres to the outlined set up. In simulation 2, we randomly select one of the three studies to have a sample size of $n = 25$. The sample sizes of the two other studies are unaffected, and incrementally increase from 25 to 800. Each generated study is analyzed with a regression model containing all six predictors and an intercept (which equals 0 in the population). We evaluate the hypothesis $H_{1,2} : \beta_4 < \beta_5 < \beta_6$,⁵ and thus do not consider hypotheses with different complexities, yet.

In simulation 1 and 2, the aggregated Bayes factors for the true hypothesis $H_{1,2} : \beta_4 < \beta_5 < \beta_6$ against the unconstrained hypothesis H_u increase with sample size and effect size (Figure 1). In simulation 1 (the red boxes), for a small effect size ($R^2 = 0.02$), $H_{1,2}$ is more often supported than H_u only when the sample size is at least $n \geq 400$. For $n = 400$ and $R^2 = 0.02$, the median aggregated Bayes factor (the middle horizontal black line in each box) is slightly above zero on the logarithmic scale, indicating that there is more support for the true hypothesis $H_{1,2}$ than for H_u in about 50% of the iterations. When the effect sizes increase, $H_{1,2}$ is preferred over H_u in most iterations when $n \geq 100$ (when $R^2 = 0.09$) or when $n \geq 50$ (when $R^2 = 0.25$). For 800 observations per study and a large effect, the aggregated Bayes factor approaches an upper bound, that is due to evaluating against an unconstrained alternative hypothesis. That is, when the hypothesis fits the data perfectly (i.e., $f_i = 1$), the Bayes factor within a study cannot exceed $1/c_i$, and the aggregated Bayes factor cannot exceed $\prod_t^T 1/c_i^{(t)}$. In simulation 2 (the blue boxes), this upper bound is not reached in any condition, as the support for $H_{1,2}$ is generally smaller than in simulation 1. Likewise, larger sample sizes (for the two unaffected studies) and effect sizes are required before $H_{1,2}$ obtains more support than H_u in the majority of the iterations. These findings are to be expected, because studies with the smallest sample size often provide support against $H_{1,2}$. Replacing a study with a larger sample size by a study with a sample size of $n = 25$ thus leads to a decrease in the aggregated support.

Additionally, the aggregated support for the true hypothesis $H_{1,2}$ versus both the unconstrained and complement hypothesis is quantified with posterior model probabilities (*PMPs*; Figure 2). These results also show that the support for the true hypothesis increases with the sample and effect size. For the smallest effect size, the average aggregated support does not exceed 0.70, indicating that $H_{1,2}$ is hardly favored over H_u . For medium and large effects, the *PMPs* tend to 1 when the sample size is large enough. Comparing simulation 1 and 2 shows that considering a single underpowered study in the set of studies substantially reduces the average aggregated *PMPs*. When the effect size equals $R^2 = 0.09$, the average aggregated support is larger for three studies with a sample size of $n = 400$ (in simulation 1; the red lines) than for two studies with a sample size of $n = 800$ and one study with $n = 25$ (in simulation 2; the blue lines). Hence, although the total number of observations is higher in the latter setting, the average support for the true hypothesis is not, regardless of the alternative hypothesis. Lastly, Figure 2 shows that evaluating against the complement hypothesis consistently renders more support for the true hypothesis, although the difference is relatively small.

⁴Complete separation in data with binary outcomes occurred sporadically for the smallest sample sizes, and was handled by generating a new data set.

⁵The hypotheses are numbered such that they correspond to the simulation in which they are evaluated.

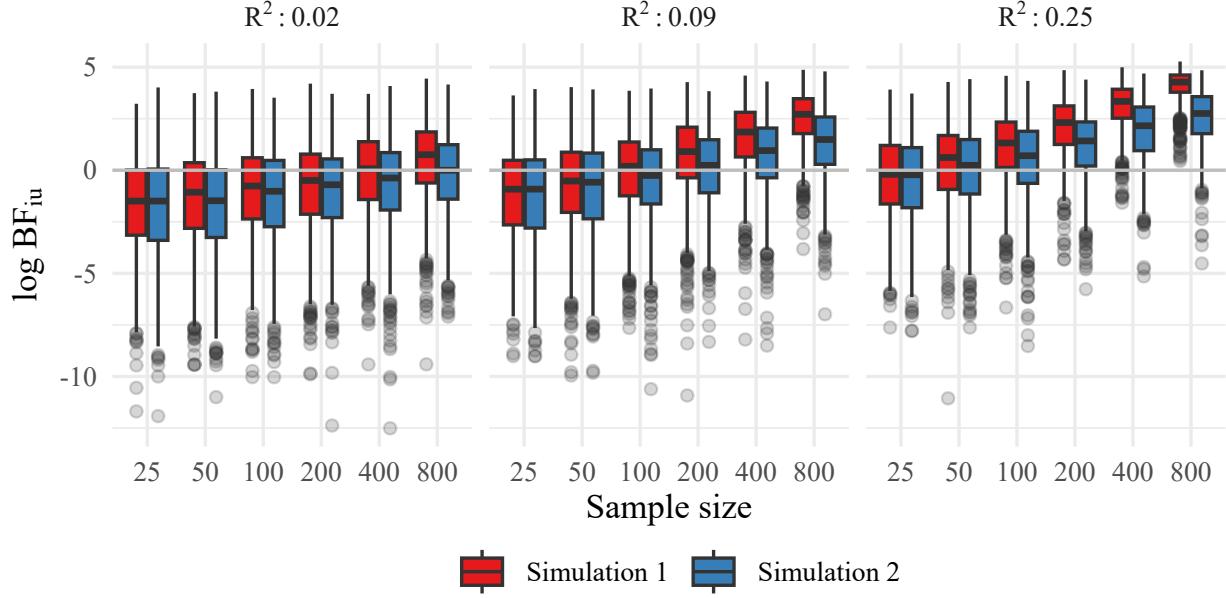


Figure 1: Aggregated Bayes factors for hypothesis $H_{1,2} : \beta_4 < \beta_5 < \beta_6$ versus H_u over three studies (with linear, logistic and probit models). In simulation 2, one of the three studies is randomly selected to have a small sample size ($n = 25$).

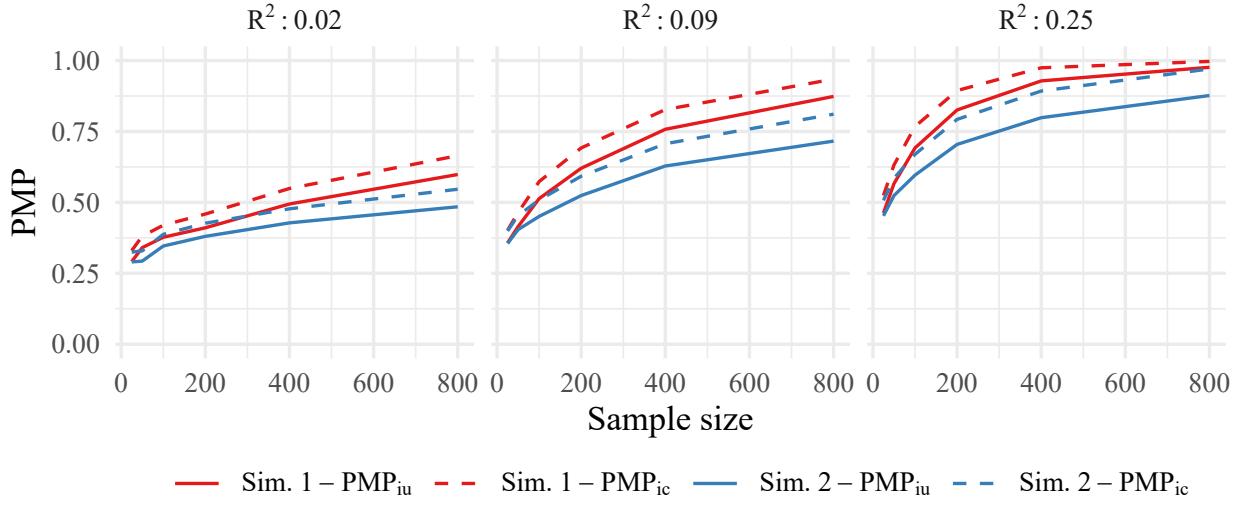


Figure 2: Aggregated $PMPs$ for hypothesis $H_{1,2} : \beta_4 < \beta_5 < \beta_6$ versus H_u or H_c over three studies (with linear, logistic and probit models). In simulation 2, one of the three studies is randomly selected to have a small sample size ($n = 25$).

3.1.2 Simulation 3 and 4

Simulations 3 and 4 assess how the complexities of hypotheses affect BES , by varying the operationalizations of a construct of interest. Both simulations consider the expectation that x_6 and y are positively related (after controlling for x_1 to x_5). In simulation 3, x_6 is considered as is, and the hypothesis $H_3 : \beta_6 > 0$ is evaluated. In simulation 4, x_6 is categorized into equally sized tertiles in each study, corresponding to *low*, *medium* and *high* scoring groups (which is, despite advice against it, a common procedure in many areas of research; e.g., [Bennette and Vickers, 2012](#); [DeCoster et al., 2011](#)). Capturing the expected positive relationship between x_6 and y into an informative hypothesis yields $H_4 : \beta_{\text{low}} < \beta_{\text{medium}} < \beta_{\text{high}}$, where each coefficient reflects the mean of that group controlled for the five

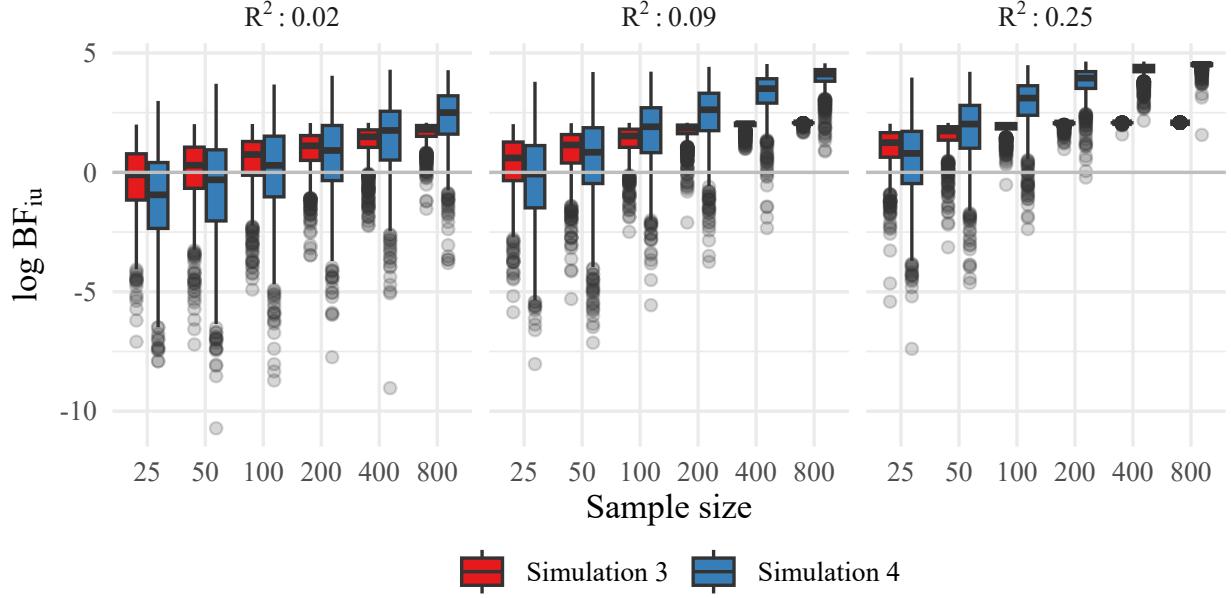


Figure 3: Aggregated Bayes factors for hypothesis $H_3 : \beta_6 > 0$ versus H_u in simulation 3 and $H_4 : \beta_{\text{low}} < \beta_{\text{medium}} < \beta_{\text{high}}$ versus H_u in simulation 4 over three studies (with linear, logistic and probit models).

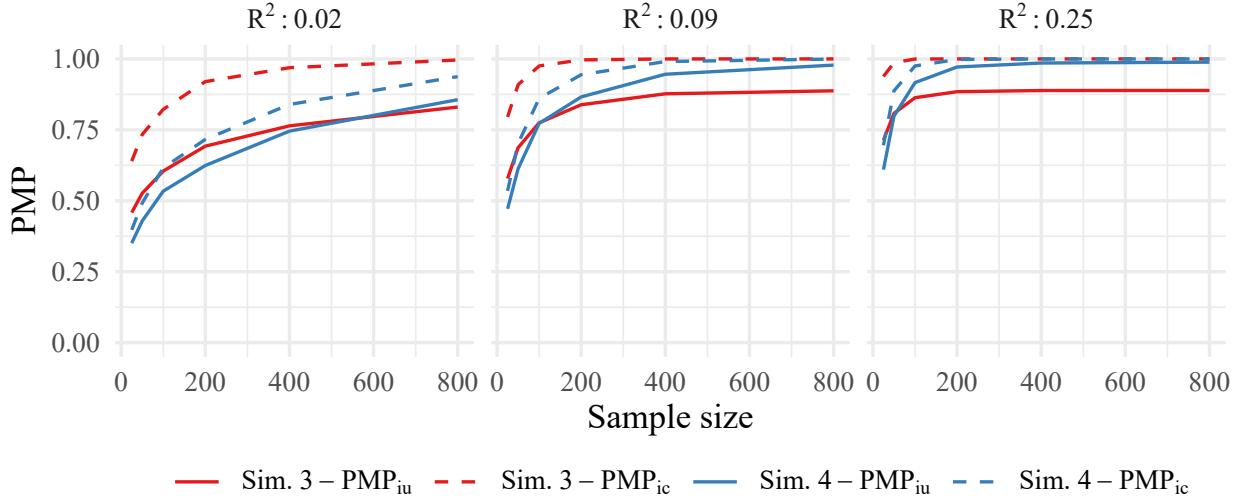


Figure 4: Aggregated PMPs for hypothesis $H_3 : \beta_6 > 0$ versus H_u and H_c in simulation 3, and for $H_4 : \beta_{\text{low}} < \beta_{\text{medium}} < \beta_{\text{high}}$ versus H_u or H_c in simulation 4 over three studies (with linear, logistic and probit models).

other variables. Consequently, the complexity of H_4 is substantially smaller than the complexity of H_3 , because more constraints are placed on the parameters. Note, however, that categorizing a continuous variable also reduces the information in this variable, resulting in less statistical power.

Figure 3 and Figure 4 show that the support for the true hypotheses $H_3 : \beta_6 > 0$ and $H_4 : \beta_{\text{low}} < \beta_{\text{medium}} < \beta_{\text{high}}$ increases with sample and effect size. This is a recurrent pattern when the hypothesis of interest is in line with the specifications of the parameters. In simulation 3 (the red boxes), H_3 obtains more support than H_u in more than 50% of the iterations when the sample size is at least $n \geq 50$ for small effect sizes. When medium and large effect sizes are considered, all sample sizes yield more support for H_3 than H_u in the majority of the iterations. For all effect sizes, the aggregated Bayes factor tends to its maximum if the sample size is large enough (given a complexity of $c_i = 1/2$ per study in this simulation, the maximum aggregated Bayes factor is approximately equal to 2.08 on a logarithmic

scale). In simulation 4 (the blue boxes), there is initially more support for H_u than for the hypothesis of interest for the smallest effect size as compared to simulation 3. Additionally, the true hypothesis H_4 obtains less support than H_3 for the smallest sample sizes considered (i.e., for $n \leq 100$ when $R^2 = 0.02$, $n \leq 50$ when $R^2 = 0.09$ and for $n = 25$ when $R^2 = 0.25$). Both findings result from H_4 having a smaller complexity than H_3 , which requires more statistical power to find support. Yet, categorizing x_6 results in less statistical power. Whereas the aggregated support for H_3 quickly reaches its maximum, the support for H_4 continues to increase to higher levels. For $n = 800$ when $R^2 = 0.09$, and for $n \geq 400$ when $R^2 = 0.25$, the support for H_4 also reaches its maximum, but this maximum is substantially higher than the maximum in simulation 3, because the complexity of the hypothesis is smaller in simulation 4.

The posterior model probabilities also clearly pinpoint the upper bound of the support for H_3 when evaluated against H_u (the red solid line in Figure 4), especially for medium and large effects. When H_3 obtains full support from the data, the posterior model probabilities cannot exceed 8/9. The support for H_3 evaluated against the complement hypothesis is unrestricted (the dashed red line), and therefore quickly tends to 1 for all effect sizes. When evaluating against H_u , the aggregated support for H_4 (the solid blue line in Figure 4) is smaller than for H_3 for small samples but increases to almost 1 when the support for H_3 already reached its maximum, due to the smaller complexity of H_4 . Additionally, there is only a small difference between evaluating H_4 against the unconstrained and the complement hypothesis in terms of *PMPs*: the support for H_4 is unconvincing initially, but increases with the sample and effect size for both alternatives.

3.1.3 Simulation 5 and 6

Simulations 5 and 6 also examine how the complexities of the candidate hypotheses affect *BES* by varying the operationalizations of key constructs. Assume that the variables x_2 , x_3 and x_4 are indicators of the same theoretical construct. In simulation 5, the three indicators are collapsed into a single scale variable x_{scale} by taking the average of these variables for each observation in each of the studies, which is common scientific practice (Bauer and Curran, 2016). Accordingly, the hypothesis under evaluation is $H_5 : \beta_{\text{scale}} > 0$, analysed in a model including an intercept and x_1 , x_5 and x_6 as control variables. In simulation 6, the variables are left as is and included separately in the analyses, such that hypothesis $H_6 : \{\beta_2, \beta_3, \beta_4\} > 0$ is evaluated (with the same control variables). Due to these specifications, H_5 has a larger complexity than H_6 , due to H_6 addressing multiple parameters. Simultaneously, taking the average of three indicators reduces the standard error or β_{scale} , resulting in more power to detect an effect.

In simulation 5 (the red boxes in Figure 5), H_5 obtains more support than H_u in more than 50% of the iterations over all sample and effect sizes, except for $n = 25$ and $R^2 = 0.02$. Additionally, the aggregated support for $H_5 : \beta_{\text{scale}} > 0$ reaches its maximum for medium (at $n \geq 400$) and large (at $n \geq 100$) effect sizes. In simulation 6 (the blue boxes in Figure 5), where the predictors are considered separately, the pattern is rather different. When the effect size is small, the unconstrained hypothesis obtains most support, unless the sample size is at least $n = 400$. For medium and large effect sizes, H_6 obtains more support than H_u when $n \geq 100$ and $n \geq 50$, respectively, and the support in terms of Bayes factors tends to much higher levels than in simulation 5.

Figure 6 for simulation 5 and 6 shows a similar pattern as Figure 4 for simulation 3 and 4. The support for H_5 tends to its maximum when compared with H_u (the solid red line), especially for a large effect, while comparing against the complement leads to *PMPs* close to 1 (the dashed red line). In simulation 6, the complexity of the hypothesis of interest is smaller, and the difference between evaluating against H_u (the solid blue line) or H_c (the dashed blue line) decreases on the scale of the *PMPs*. For small sample and effect sizes, this renders small *PMPs*, to such an extent that H_u is preferred over H_6 for the smallest sample and effect sizes. Yet, the smaller complexity also yields that the maximum *PMP* when evaluating against H_u is larger in simulation 6 than in simulation 5. If the sample and effect size are sufficiently large, the *PMPs* for H_6 are close to 1, regardless of whether H_6 is evaluated against H_u or H_c . These results show that more statistical power is required in order to evaluate more specific hypotheses.

3.1.4 Simulation 7 and 8

All previous simulations were concerned about whether *BES* provides adequate results when a correct informative hypothesis is evaluated against the unconstrained or complement hypothesis. In simulation 7 and 8, we assess the performance of *BES* when the hypothesis of interest is incorrect. We therefore expect *BES* to render *less* support for the hypotheses of interest when the sample and effect size increase. In simulation 7, we consider $H_7 : \{\beta_2, \beta_3, \beta_4\} < 0$, implying a negative relationship between the indicators x_2 , x_3 and x_4 and the outcome y , which is for each parameter in the opposite direction of the true value. Hence, the unconstrained and complement alternative hypotheses are correct, although rather unspecific, in these simulations. In simulation 8, we evaluate the *partially incorrect* hypothesis $H_8 : \{\beta_1, \beta_2, \beta_3\} > 0$, which is correct for β_2 and β_3 , but incorrect for β_1 , because $\beta_1 = 0$. This specification renders the unconstrained hypothesis correct, while the complement hypothesis is also partially incorrect, because the true parameter value of β_1 is exactly on the boundary of H_8 and H_c . In both simulations, the analysis model contains an intercept and the remaining variables as control variables.

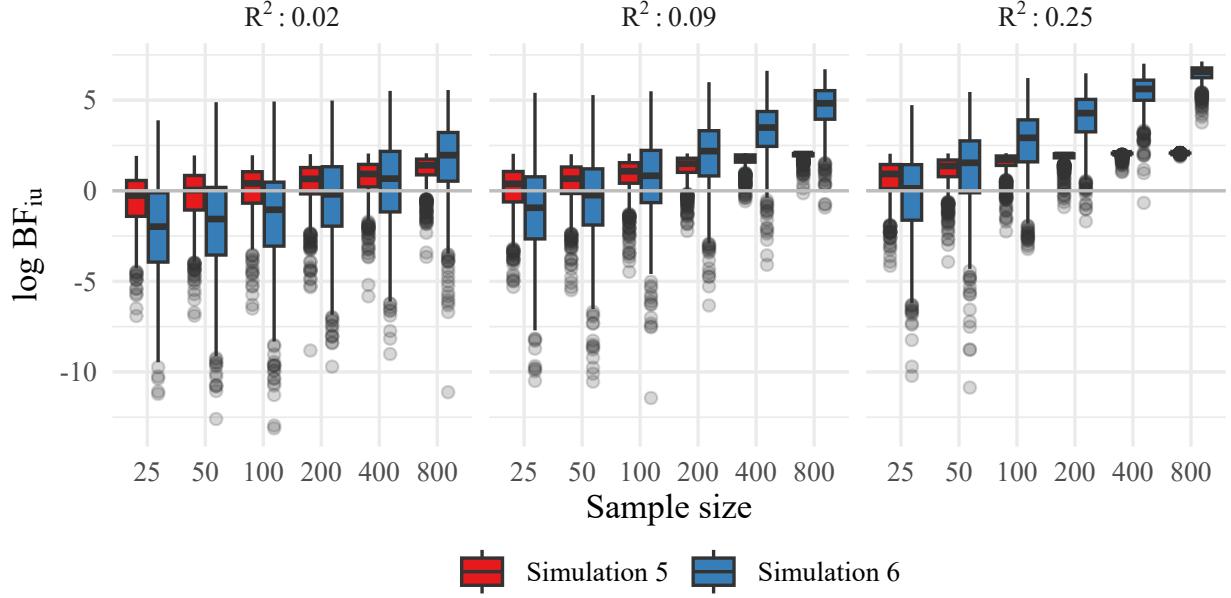


Figure 5: Aggregated Bayes factors for hypothesis $H_5 : \beta_{\text{scale}} > 0$ versus H_u in simulation 5 and $H_6 : \{\beta_2, \beta_3, \beta_4\} > 0$ versus H_u in simulation 6 over three studies (with linear, logistic and probit models).

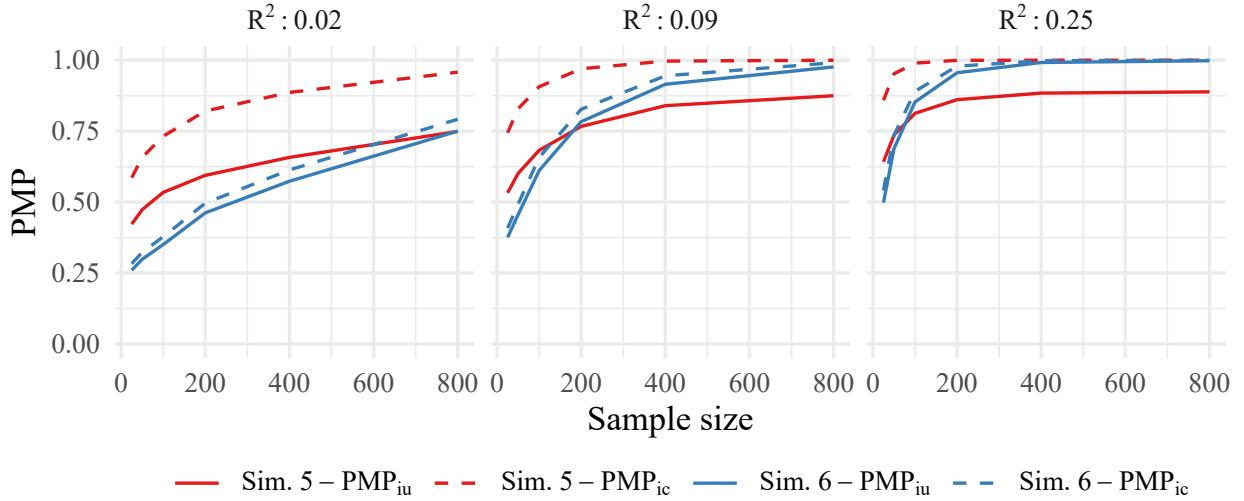


Figure 6: Aggregated PMPs for hypothesis $H_5 : \beta_{\text{scale}} > 0$ versus H_u and H_c in simulation 5 and $H_6 : \{\beta_2, \beta_3, \beta_4\} > 0$ versus H_u or H_c in simulation 6 over three studies (with linear, logistic and probit models).

The red boxes in Figure 7 show that in simulation 7, the support for the incorrect hypothesis of interest quickly decreases, rendering more support for the unconstrained hypothesis. In fact, already from the smallest sample sizes onward, there is less support for H_7 than for the correct hypothesis H_u , which further decreases when the sample and effect size increase. In simulation 8, the support for the partially incorrect hypothesis H_8 increases, rather than decreases, with the sample size and effect size (the blue boxes in Figure 7). Whereas for small sample and effect sizes the correct unconstrained hypothesis is preferred over H_8 , the partially incorrect H_8 eventually obtains more support. Although this behavior is undesirable, it is not surprising. The posterior distribution of β_1 is, on average, for 50% in line with the constraints of H_8 , because the true parameter value is on the boundary of the hypothesis, while for larger sample and effect sizes, the posterior of β_2 and β_3 is almost completely in line with this hypothesis. Hence, even though the hypothesis is partially incorrect, the fit generally exceeds the complexity.

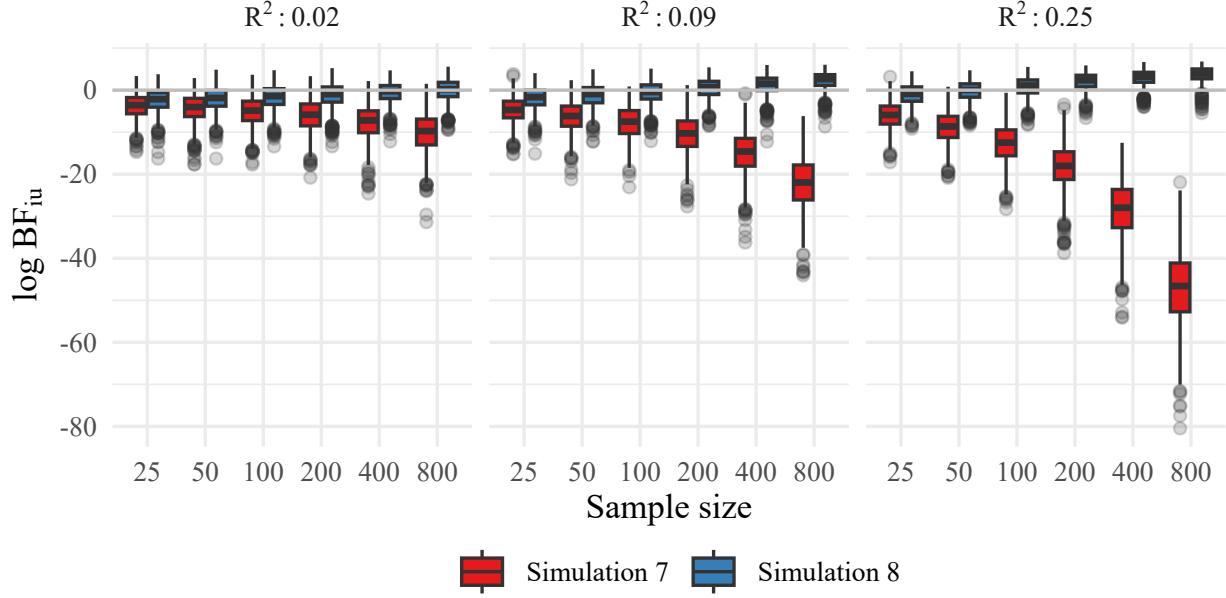


Figure 7: Aggregated Bayes factors for *incorrect* and *partially incorrect* hypotheses $H_7 : \{\beta_2, \beta_3, \beta_4\} < 0$ and $H_8 : \{\beta_1, \beta_2, \beta_3\} > 0$ versus H_u in simulation 7 and 8 over three studies (with linear, logistic and probit models).

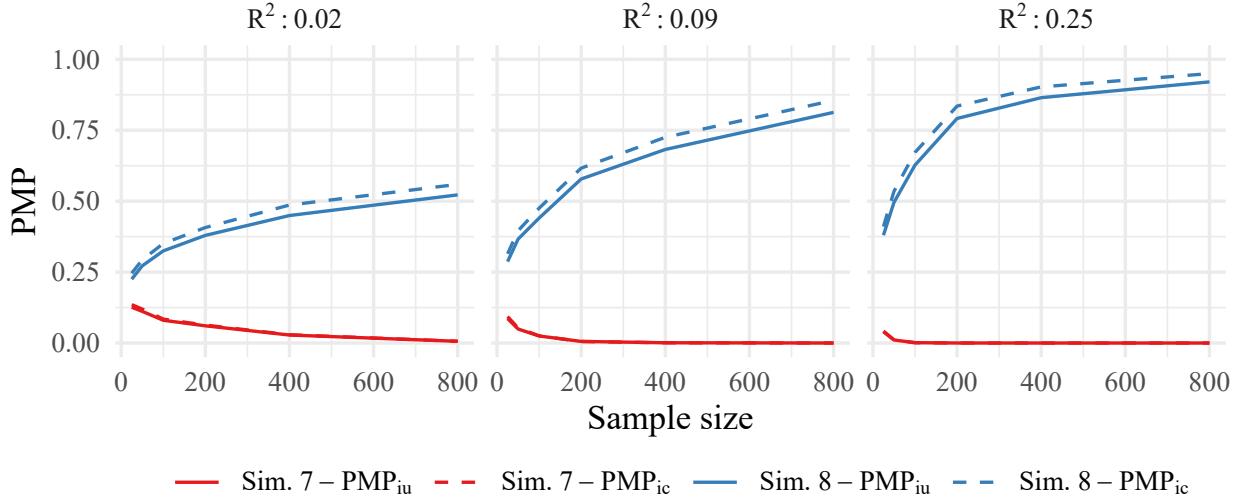


Figure 8: Aggregated *PMPs* for *incorrect* and *partially incorrect* hypotheses $H_7 : \{\beta_2, \beta_3, \beta_4\} < 0$ and $H_8 : \{\beta_1, \beta_2, \beta_3\} > 0$ versus H_u or H_c in simulation 7 and 8 over three studies (with linear, logistic and probit models). Note that the lines of $PMP_{i,u}$ and $PMP_{i,c}$ are almost completely overlapping in simulation 7.

Figure 8 tells a similar story. The average aggregated *PMPs* for the incorrect H_7 render very strong support against this hypothesis, for all sample sizes and effect sizes and for both alternative hypotheses (the red solid and dashed lines, note that these are almost completely overlapping). In simulation 8 (the blue solid and dashed lines in Figure 8), the average aggregated *PMPs* are indecisive under a small effect size, but favor the partially incorrect hypothesis H_8 over the correct unconstrained and partially incorrect complement hypothesis when the effect and sample size increase. The difference between evaluating against H_u and H_c is negligible, although H_u is the only correct hypothesis in this simulation.

3.1.5 Discussion simulations part 1

The first simulations show that *BES* performs adequately, in the sense that the aggregated support for correct hypotheses increases with the sample and effect size (simulations 1-6). Moreover, simulation 7 shows that incorrect hypotheses obtain less support when the sample and effect size increase. The main focus of these simulations was on how the complexity of the hypotheses affects the performance of *BES*. Especially in the context of conceptual replications, researchers might want to aggregate the support for hypotheses with different complexities, due to different operationalizations or measurement instruments. Unlike conventional research synthesis approaches, *BES* allows to aggregate support for equivalent hypotheses with different complexities and, as simulations 3 to 6 show, renders satisfactory results when the individual studies have sufficient power. Hence, the support for an overarching theory over studies can be quantified with *BES*, even when studies use different operationalizations. Our findings may even insinuate that, with sufficient power, common procedures in data handling that result in hypotheses with smaller complexities, such as categorizing continuous variables or assessing individual indicators rather than scale scores, can lead to more support on the aggregate level. After all, the hypotheses with the smallest complexities within a set of studies have the highest upper bound for the Bayes factor against the unconstrained hypothesis. However, this conclusion should be drawn with caution, if at all, because it only holds when evaluating against the unconstrained, but not when evaluating against the complement hypothesis. The Bayes factor against the complement tends to infinity when the data fits the hypothesis perfectly, and thus does not benefit from evaluating more specific hypotheses.

In simulation 8, *BES* functions unsatisfactorily, because with increasing sample and effect sizes, it renders increasing support for the partially incorrect hypothesis. However, this hypothesis is very close to the truth (Bayes factors have been proven to prefer the model that is closest, in terms of Kullback-Leibler divergence, to the true data-generating model; see [Ly et al., 2016](#); [Berger, 2013](#)). When evaluating inequality-constrained hypotheses against the unconstrained or complement alternative, support for incorrect hypotheses can only keep increasing with the sample and effect size when no parameters truly violate the hypothesis of interest (i.e., all parameters are on or within the boundary values of the hypothesis). If at least one of the parameters falls outside the constraints imposed by the hypothesis, the fit will tend to zero for sufficiently large sample sizes. Yet, the fact that partially incorrect hypotheses can obtain substantial support warrants that researchers not only assess the aggregated support for complex hypotheses, but also consider the support for the parameters in the individual studies. If some of the estimated parameters are typically in line with the hypothesis while others fluctuate around the hypothesized value between studies, further investigation is required, potentially leading to theoretical refinements, that should be evaluated with new data, regardless of the aggregated support.

Similarly to previous work (e.g., [Klugkist and Volker, 2023](#)), our simulations confirm that if individual studies lack statistical power, *BES* has difficulties finding support for the correct hypothesis. Specifically, including a single underpowered study, as in simulation 1 and 2, reduces the performance of *BES*. Moreover, aggregating over three adequately powered studies provides more support for the correct hypothesis than aggregating over two adequately powered studies and a single underpowered study, even if the total number of observations is higher in the latter scenario. Whereas conventional approaches for research synthesis as meta-analysis or Bayesian sequential updating can overcome such power issues, *BES* typically does not, because it answers a different question. Whereas these conventional approaches assess whether the pooled estimate is in line with the hypothesis, *BES* questions to what extent each study provides support for the hypothesis and combines this into a single measure of evidence. Hence, some support against the hypothesis of interest in each study will accumulate when aggregating over studies, and considerable support against this hypothesis in one study is not necessarily counterbalanced by support for this hypothesis in multiple other studies.

Such power issues are amplified under hypotheses with smaller complexities. With insufficient power, sampling variability increases and parameters may not be estimated accurately. If a hypothesis places constraints on multiple parameters, it becomes increasingly likely that at least one constraint is violated, reducing the fit of the hypothesis. Common procedures in data handling that reduce the complexity of the hypothesis (e.g., categorizing continuous variables), and simultaneously the power of the analysis, may thus have adverse consequences for *BES*. Although our previous simulations show the vulnerability of *BES* to power issues, substantial uncertainty remains about the extent to which these problems depend on (i) the number of studies included, (ii) the complexity of the hypothesis of interest, and (iii) the choice of alternative hypothesis. Evaluating against the complement hypothesis yields a more powerful evaluation of the hypothesis of interest, which may reduce the severity of power issues. Our findings also suggest that including more studies with insufficient power may lead to decreasing support for the correct hypothesis. We address these questions in the subsequent section.

3.2 Simulations part 2 - power issues of *BES*

Part two of the simulations zooms in on the extent to which *BES* renders support for *correct* hypotheses as the number of studies increases, while comparing between studies with and without sufficient power. Moreover, we further assess the influence of the complexity of the hypothesis and the choice of alternative hypothesis (i.e., an unconstrained or

a complement alternative hypothesis) on the amount of aggregated support. We consider the same data-generating mechanism as in previous simulations, but restrict the simulations to OLS regression for the sake of brevity (although other models yield equivalent results). To keep the results tractable, only one effect size and two sample sizes are considered ($R^2 = 0.09$ and $n \in \{25, 200\}$), representing underpowered and adequately powered studies, respectively. The number of studies is varied from 1 to 150, and the number of iterations is again set to 1000. In all three simulations in part two, the cumulative aggregated Bayes factor is assessed for the hypotheses of interest against an unconstrained and a complement alternative hypothesis for the two sample sizes considered.

3.2.1 Simulations 9, 10 and 11

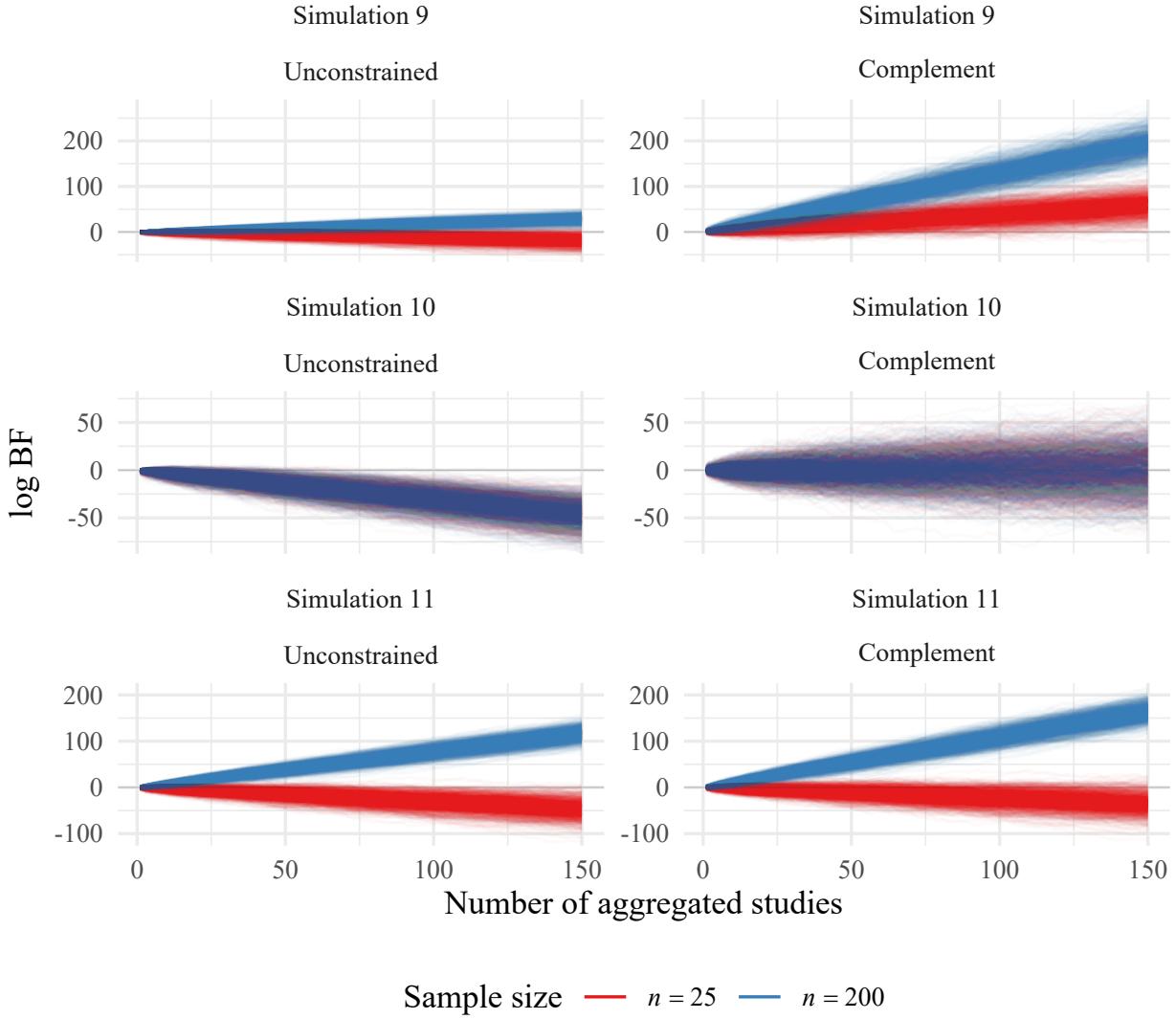


Figure 9: Cumulative aggregated Bayes factors for the correct hypothesis $H_9 : \beta_2 > 0$ in simulation 9, $H_{10} : \beta_1 > 0$ in simulation 10 and $H_{11} : \{\beta_2, \beta_3, \beta_4\} > 0$ in simulation 11, versus an unconstrained and complement alternative hypothesis over 1 to 150 studies, based on OLS regression models. Note that (i) the scale of the y-axis differs between the simulations, and (ii) the lines for the two sample sizes are almost completely overlapping in simulation 10.

In simulation 9, we evaluate $H_9 : \beta_2 > 0$ against the unconstrained and complement hypotheses and assess the influence of the alternative hypothesis for underpowered and adequately powered studies. Recall that H_9 is correct, as the population value of the coefficient equals $\beta_2 = 0.054$ (Table 1). For studies with a small sample size, Figure 9 shows that evaluating against H_u renders decreasing support for the correct hypothesis H_9 when the number of studies increases. After conducting 150 studies, H_u is clearly preferred over H_9 . For larger samples (i.e., $n = 200$), the

correct hypothesis H_9 is consistently preferred over H_u . Evaluating H_9 against the complement hypothesis consistently renders support for H_9 for both sample sizes, although the support increases faster for larger samples. Additionally, the absence of an upper bound yields that the support for H_9 increases at a faster rate when evaluated against H_c instead of against H_u .

In simulation 10, we investigate a similar, but incorrect, hypothesis $H_{10} : \beta_1 > 0$ ($\beta_1 = 0$ in the population; Table 1). This renders H_u the only true hypothesis, because H_c and H_{10} are equally incorrect. Evaluating against the unconstrained hypothesis renders more support for H_u than for H_{10} , regardless of the sample size. When the true parameter value is on the boundary of the hypothesis of interest, comparing against the complement renders substantial variability in the support for H_{10} and H_c , regardless of the sample size (note that the lines for the two sample sizes are almost completely overlapping). Although H_{10} and H_c obtain, on average, the same support, the individual iterations show considerable support for either of the two. Hence, there is a downside to evaluating against the complement hypothesis, because it is possible to find considerable support for or against a hypothesis on the aggregate level, while there is no effect in reality.

Simulation 11 evaluates the *correct* $H_{11} : \{\beta_2, \beta_3, \beta_4\} > 0$ ($\beta_2 = \beta_3 = \beta_4 = 0.054$ in the population; Table 1). Whereas the hypotheses of interest in simulations 9 and 10 were balanced with their respective complements (i.e., both have a complexity of $c_i = 1/2$), H_{11} is not. Also note that the size of the coefficients here is the same as the size of the coefficient in simulation 9, so the only difference is the complexity of the hypothesis due to considering three parameters. Simulation 11 in Figure 9 shows that the support for the correct hypothesis H_{11} decreases when more small sample studies are included, regardless of the alternative hypothesis, although there is somewhat more support on average when evaluating against the complement. Hence, when the complexity of the hypothesis of interest and its complement are not balanced, the aggregated Bayes factor does not necessarily tend to the *correct* hypothesis, but favors the more general alternatives. When the sample size is sufficiently large, the issue dissolves, and the support for H_{11} increases substantially when more studies are added, for both alternative hypotheses.

Informative hypotheses on multiple parameters can be separated in multiple, simpler hypotheses. For example, the hypothesis H_{11} can be deconstructed into three simpler hypotheses such that each component is balanced with its complement ($H_{11_a} : \beta_2 > 0$, $H_{11_b} : \beta_3 > 0$ and $H_{11_c} : \beta_4 > 0$). Evaluating each sub-hypothesis against the unconstrained hypothesis and its complements yields an identical outcome as in simulation 9. Each sub-hypothesis obtains more support than the unconstrained hypothesis when the sample size is large, but H_u obtains more support for small sample sizes. When evaluating each hypothesis against its complement, the sub-hypotheses obtain considerable support, regardless of the sample size. Thus, whereas evaluating a multifaceted hypothesis as H_{11} leads to support against the true hypothesis of interest for smaller sample sizes, evaluating the separate components of this hypothesis provides support for each component.

3.2.2 Discussion simulation part 2

Part two of the simulations further assessed the sensitivity of BES to power issues. When the studies are adequately powered, the support for the true hypothesis is consistently found over the situations. However, when evaluating a correct hypothesis on a single regression parameter in studies that lack statistical power, the aggregated support for the unconstrained hypothesis increases with the number of studies. If the hypothesis of interest is balanced with its complement, such that both have the same complexity, evaluating against the complement hypothesis is not sensitive to the power within the studies. This difference is due to the fact that evaluating against the unconstrained hypothesis weighs evidence against the hypothesis of interest heavier than evidence for this hypothesis. That is, the Bayes factor for a hypothesis evaluated against the unconstrained has an upper bound, while the lower bound of 0 corresponds to infinite support for the unconstrained. Note that when studies have little power, parameter estimates vary considerably. If a single study finds, by chance, substantial support against the hypothesis of interest (e.g., a fit of $f_i = 0.1$ and a complexity of $c_i = 0.5$, rendering a Bayes factor of $BF_{iu} = 0.2$), adding two new studies that perfectly fit the hypothesis of interest (resulting in a Bayes factor of $BF_{iu}^T = 1/0.5 = 2$ per study) yields an aggregate Bayes factor of $BF_{iu}^T = 0.8$. On the aggregate level, there thus is still more support for the unconstrained hypothesis. Evaluating against the complement weighs the evidence for both hypotheses equally heavy under these circumstances, and thus provides increasing support for the correct hypothesis.

When the hypothesis of interest places constraints on multiple parameters, evaluating against the complement results in somewhat more power than evaluating against the unconstrained, but both provide support against the correct hypothesis of interest when the studies are underpowered. Whereas part one already showed that evaluating hypotheses with smaller complexities is only appropriate when studies are adequately powered, part two of the simulations shows that adding more studies provides no solution. The hypothesis of interest constrained three parameters, resulting in 8 possible parameter orderings of which only one was correct. When the individual studies have little power, the probability that at least one of the estimated parameters falls outside the constraints imposed by the hypothesis is considerable, due to the fact that the parameters are generally estimated inaccurately. Regardless of which constraint is

violated, the hypothesis may fit poorly in each study, resulting in aggregated support against the hypothesis that further increases with the number of studies. This issue can be remedied by deconstructing the hypothesis space in smaller regions, and evaluating each part separately against each complement. This approach has two advantages. First, less statistical power is required to find support for the correct hypothesis. Especially, if each sub-hypothesis is balanced with its complement, *BES* will provide support for the correct hypothesis if enough studies are included. Second, this approach allows to evaluate whether each component of the hypothesis obtains support after aggregation. If the overall hypothesis is incorrect, evaluating the sub-hypotheses highlights which parts of the overall hypothesis are not supported. In this sense, deconstructing the hypothesis space can help to further refine the theory.

Although evaluating hypotheses against their balanced complements has advantages, it is no panacea. Simulation 10 shows that if the true parameter value is on the boundary of the hypothesis of interest and its complement, it is possible to find overwhelming support for either of the two. The implications hereof reach beyond *BES*, because already within individual studies strong support can be obtained for incorrect hypotheses. *BES* further amplifies this problem. The variability of the aggregated support increases with the number of studies, such that overwhelming support for one of the two hypotheses regularly occurs. This problem can be dealt with in three ways. First, one could consider an equality-constrained hypothesis (e.g., $H_i : \beta_1 = 0$). In contrast to evaluating inequality-constrained hypotheses (Klugkist and Hoijtink, 2007), however, evaluating equality-constrained hypotheses with a Bayes factor is sensitive to the scale (i.e., variance) of the prior distribution within a study (Hoijtink, 2021; Tendeiro and Kiers, 2019). Accordingly, a sensitivity analysis of the Bayes factor within a study is generally required (Hoijtink, 2021), which induces uncertainty regarding the “correct” Bayes factor within a study, let alone when aggregated over studies. Additionally, Bayes factors on equality-constrained hypotheses versus unconstrained alternatives tend to provide overly strong evidence in favor of the former, especially when the power of the study is relatively small (e.g., Tendeiro and Kiers, 2019). This approach might induce more severe power issues when using *BES*. Future research should address these considerations in more detail. Second, one could evaluate a hypothesis with a boundary on the smallest effect of interest (Lakens, 2014) versus its complement. Ultimately, when support for the hypothesis is found, one can be sure that there is an effect that is relevant. If no support is found, either there is no effect, or the effect is too small to be relevant. Third, the hypothesis of interest can be evaluated against both the unconstrained and the complement hypothesis. If both render support for (or against) the hypothesis of interest, one can conclude that this hypothesis provides an accurate description of the data. If the results are contradictory, researchers may have to acknowledge that considerable uncertainty remains, and that more (adequately powered) studies on the topic are required for a robust conclusion.

4 Conclusion

In multiple simulations, we examined the performance of Bayesian Evidence Synthesis when evaluating hypotheses with different complexities against different alternatives under various sample and effect sizes. Part one of the simulations showed that *BES* is applicable regardless of differences in analysis models in the set of studies under consideration, and renders satisfactory results if the individual studies have sufficient statistical power. The simulations emphasized the importance of power when aggregating evidence over studies, especially when evaluating more specific hypotheses. For small sample and effect sizes, evaluating a hypothesis with a relatively small complexity generally results in support for the unconstrained and complement hypotheses. If the Bayes factors within the studies yield more support for the alternatives than for the specific correct hypothesis, adding more studies that are also underpowered will not solve the issue. Hence, *BES* differs from conventional research synthesis methods as (Bayesian) meta-analysis and Bayesian sequential updating. Whereas the former two approaches increase the statistical power when incorporating evidence from additional studies, *BES*, in general, does not. As said, *BES* answers a somewhat different question than these conventional methods, as it aggregates the extent to which each study supports the overall hypothesis, rather than to what extend the pooled parameter estimate supports the hypothesis of interest. In this sense, *BES* can be seen as a joint Bayes factor that measures the support for an overall hypothesis that states that there is support for each study-specific hypothesis.

Part two of the simulations underscored that adding more underpowered studies only amplifies power issues. Aggregating the evidence for a hypothesis with a small complexity consistently renders support against this hypothesis when the studies lack power, regardless of the alternative hypothesis. If the hypothesis of interest and its complement are balanced, however, the aggregated Bayes factor will eventually show support for the correct hypothesis, if either the hypothesis of interest or its complement is correct. As a consequence, it can be worthwhile to separate a specific hypothesis with multiple parameter constraints into multiple sub-hypotheses that are all balanced with their complements. If either the sub-hypothesis or its complement is correct, the aggregated Bayes factor will provide support for the correct hypothesis. Moreover, evaluating sub-hypotheses allows to assess which constraints imposed by the overall hypothesis are supported by the data and which are not, providing a more detailed overview of the support in the studies for the overarching hypothesis.

Overall, evaluating against the complement has advantages over evaluating against the unconstrained. Evaluating against the complement results in more statistical power, and the resulting Bayes factor has no upper bound. Additionally, in a practical research setting, some studies may have insufficient power, while others have sufficient power. In such instances, adequately powered studies will eventually provide decisive support when evaluated against the complement hypothesis. When evaluating against the unconstrained, the underpowered studies may jeopardize the aggregated evidence. That is, if underpowered studies occasionally render support against the hypothesis of interest, the adequately powered studies may not be able to compensate because of the upper bound. A disadvantage of evaluating against the complement occurs if the true parameter value is on the boundary of the hypothesis of interest and its complement, because the aggregated support becomes highly variable. We discussed several approaches to deal with this issue, but future research should compare the advantages and disadvantages of these approaches.

There are other aspects that this research did not address. First of all, we focused solely on inequality constrained hypotheses, whereas equality constrained hypotheses (such as the classical null hypothesis) are still standard practice in research. As said, evaluating null hypotheses may amplify power issues and thus affect the performance of *BES*, therefore warranting future research. Additionally, we considered only three members of the GLM family and used relatively simple data generating models with few parameters. When considering many parameters, each individual parameter may only contribute little when predicting the outcome, which would reduce the power of the analyses, enlarging the issues we already discussed. Lastly, we varied the hypothesis of interest only between the set of simulations, but not within a set of studies, to easily compare between different hypotheses. In practice, it seems more realistic that researchers want to use *BES* especially because specifications of the overall hypothesis differ within the set of studies. Under these circumstances, the aggregated support may be driven by results in one or two studies. Future research should address to what extent this behavior is problematic, and methodological developments might be required to make *BES* less sensitive to the results of “outlying” studies. Potentially, weighting the studies in terms of power might be one way forward. To some extent, this already happens implicitly when evaluating against the complement hypothesis by allowing the Bayes factor to tend to 0 or infinity, but not so much when evaluating against the unconstrained hypothesis.

Overall, *BES* has strengths that conventional methods for research synthesis lack, in the sense that *BES* is capable of aggregating the support for hypotheses over studies with different designs (i.e., experimental, cross-sectional or longitudinal) or different methodologies. However, there are situations in which solely evaluating the aggregated evidence can fall short, for example when the hypothesis of interest is too specific for the statistical power of a study, or when the hypothesis under evaluation is partially incorrect. Hence, researchers should not only blindly follow the results after aggregation, but also assess the results in the individual studies. The results on the level of the individual studies may give an additional sense of the robustness of the results over different situations, while simultaneously signifying potential moderating circumstances of the effect of interest. Such additional information might raise doubts about the robustness of the conclusions in specific scenarios, but can also hint towards interesting new areas of research or corroborate the conclusions from the synthesis. Overall, *BES* provides great opportunities to aggregate scientific evidence over heterogeneous studies, but solely relying on this aggregate derogates from the wealth of information in the individual studies.

5 Acknowledgements

We gratefully acknowledge stimulating discussions with Vincent Buskens and Werner Raub.

References

- Jens B. Asendorpf, Mark Conner, Filip de Fruyt, Jan De Houwer, Jaap J. A. Denissen, Klaus Fiedler, Susann Fiedler, David C. Funder, Reinholt Kliegl, Brian A. Nosek, Marco Perugini, Brent W. Roberts, Manfred Schmitt, Marcel A. G. van Aken, Hannelore Weber, and Jelte M. Wicherts. *Recommendations for increasing replicability in psychology. Methodological issues and strategies in clinical research*, 4th ed. American Psychological Association, 2016. doi: 10.1037/14805-038.
- Monya Baker. Reproducibility crisis. *Nature*, 533(26):353–66, 2016. doi: 10.1038/533452a.
- Daniel J. Bauer and Patrick J. Curran. *The discrepancy between measurement and modeling in longitudinal data analysis.*, pages 3–38. CILVR series on latent variable methodology. IAP Information Age Publishing, Charlotte, NC, US, 2016.
- Caroline Bennette and Andrew Vickers. Against quantiles: categorization of continuous variables in epidemiologic research, and its discontents. *BMC Medical Research Methodology*, 12(1):21, 2012. doi: 10.1186/1471-2288-12-21.
- James O Berger. *Statistical decision theory and Bayesian analysis*. Springer Science & Business Media, New York, NY, 2013.

- Mark J Brandt, Hans IJzerman, Ap Dijksterhuis, Frank J Farach, Jason Geller, Roger Giner-Sorolla, James A Grange, Marco Perugini, Jeffrey R Spies, and Anna Van't Veer. The replication recipe: What makes for a convincing replication? *Journal of Experimental Social Psychology*, 50:217–224, 2014. doi: 10.1016/j.jesp.2013.10.005.
- Colin F Camerer, Anna Dreber, Eskil Forsell, Teck-Hua Ho, Jürgen Huber, Magnus Johannesson, Michael Kirchler, Johan Almenberg, Adam Altmejd, Taizan Chan, et al. Evaluating replicability of laboratory experiments in economics. *Science*, 351(6280):1433–1436, 2016. doi: 10.1126/science.aaf0918.
- Colin F Camerer, Anna Dreber, Felix Holzmeister, Teck-Hua Ho, Jürgen Huber, Magnus Johannesson, Michael Kirchler, Gideon Nave, Brian A Nosek, Thomas Pfeiffer, et al. Evaluating the replicability of social science experiments in nature and science between 2010 and 2015. *Nature Human Behaviour*, 2(9):637–644, 2018. doi: 10.1038/s41562-018-0399-z.
- J. Cohen. *Statistical power analysis for the behavioral sciences*. Lawrence Erlbaum Associates, New York, NY, 2nd edition, 1988.
- Jacob Cohen. Things I have learned (so far). *American Psychologist*, 45(12):1304–1312, 1990. doi: 10.1037/0003-066X.45.12.1304.
- Jacob Cohen. The earth is round ($p < .05$). *American Psychologist*, 49(12):997–1003, 1994. doi: 10.1037/0003-066X.49.12.997.
- Harris Cooper, Larry Vernon Hedges, and Jeffrey C Valentine. *The Handbook of Research Synthesis and Meta-Analysis*. Russell Sage Foundation, New York, NY, 2nd edition, 2009.
- Christian S. Crandall and Jeffrey W. Sherman. On the scientific superiority of conceptual replications for scientific progress. *Journal of Experimental Social Psychology*, 66:93–99, 2016. doi: 10.1016/j.jesp.2015.10.002.
- Geoff Cumming. The new statistics: Why and how. *Psychological science*, 25(1):7–29, 2014. doi: 10.1177/0956797613504966.
- Jamie DeCoster, Marcello Gallucci, and Anne-Marie R. Iselin. Best practices for using median splits, artificial categorization, and their continuous alternatives. *Journal of Experimental Psychopathology*, 2(2):197–209, 2011. doi: 10.5127/jep.008310.
- Alfred DeMaris. Explained variance in logistic regression: A monte carlo study of proposed measures. *Sociological Methods & Research*, 31(1):27–74, 2002. doi: 10.1177/0049124102031001002.
- James M. Dickey. The Weighted Likelihood Ratio, Linear Hypotheses on Normal Location Parameters. *The Annals of Mathematical Statistics*, 42(1):204 – 223, 1971. doi: 10.1214/aoms/1177693507.
- Andrew Gelman, John B. Carlin, Hal S. Stern, Aki Vehtari, and Donald B. Rubin. *Bayesian Data Analysis*. Chapman and Hall/CRC, New York, NY, 3rd edition, 2004. doi: 10.1201/b16018.
- Steven N. Goodman, Daniele Fanelli, and John P. A. Ioannidis. What does research reproducibility mean? *Science Translational Medicine*, 8(341):341ps12–341ps12, 2016. doi: 10.1126/scitranslmed.aaf5027.
- Xin Gu, Joris Mulder, and Herbert Hoijtink. Approximated adjusted fractional bayes factors: A general method for testing informative hypotheses. *British Journal of Mathematical and Statistical Psychology*, 71(2):229–261, 2018. doi: 10.1111/bmsp.12110.
- Timothy M. Hagle and Glenn E. Mitchell. Goodness-of-fit measures for probit and logit. *American Journal of Political Science*, 36(3):762–784, 1992. doi: 10.2307/2111590.
- Herbert Hoijtink. *Informative Hypotheses: Theory and Practice for Behavioral and Social Scientists*. CRC Press, New York, NY, 2012.
- Herbert Hoijtink. Prior sensitivity of null hypothesis bayesian testing. *Psychological Methods*, 2021. doi: 10.1037/met0000292.
- Herbert Hoijtink, Joris Mulder, Caspar van Lissa, and Xin Gu. A tutorial on testing hypotheses using the Bayes factor. *Psychological methods*, 24(5):539, 2019. doi: 10.1037/met0000201.
- Harold Jeffreys. *Theory of probability*. Oxford University Press, Oxford, 3rd ed. edition, 1961.
- Robert E. Kass and Adrian E. Raftery. Bayes factors. *Journal of the American Statistical Association*, 90(430):773–795, 1995. doi: 10.1080/01621459.1995.10476572.
- Sofieke T. Kevenaar, Maria A.J. Zondervan-Zwijnenburg, Elisabet Blok, Heiko Schmengler, M. (Ties) Fakkel, Eveline L. de Zeeuw, Elsje van Bergen, N. Charlotte Onland-Moret, Margot Peeters, Manon H.J. Hillegers, Dorret I. Boomsma, and Albertine J. Oldehinkel. Bayesian evidence synthesis in case of multi-cohort datasets: An illustration by multi-informant differences in self-control. *Developmental Cognitive Neuroscience*, 47:100904, 2021. doi: 10.1016/j.dcn.2020.100904.

Richard A. Klein, Kate A. Ratliff, Michelangelo Vianello, Reginald B. Adams, Štěpán Bahník, Michael J. Bernstein, Konrad Bocian, Mark J. Brandt, Beach Brooks, Claudia Chloe Brumbaugh, Zeynep Cemalcilar, Jesse Chandler, Winnee Cheong, William E. Davis, Thierry Devos, Matthew Eisner, Natalia Frankowska, David Furrow, Elisa Maria Galliani, Fred Hasselman, Joshua A. Hicks, James F. Hovermale, S. Jane Hunt, Jeffrey R. Huntsinger, Hans IJzerman, Melissa-Sue John, Jennifer A. Joy-Gaba, Heather Barry Kappes, Lacy E. Krueger, Jaime Kurtz, Carmel A. Levitan, Robyn K. Mallett, Wendy L. Morris, Anthony J. Nelson, Jason A. Nier, Grant Packard, Ronaldo Pilati, Abraham M. Rutchick, Kathleen Schmidt, Jeanine L. Skorinko, Robert Smith, Troy G. Steiner, Justin Storbeck, Lyn M. Van Swol, Donna Thompson, A. E. van 't Veer, Leigh Ann Vaughn, Marek Vranka, Aaron L. Wichman, Julie A. Woodzicka, and Brian A. Nosek. Investigating variation in replicability. *Social Psychology*, 45(3):142–152, 2014. doi: 10.1027/1864-9335/a000178.

Irene Klugkist and Herbert Hoijtink. The bayes factor for inequality and about equality constrained models. *Computational Statistics & Data Analysis*, 51(12):6367–6379, 2007. doi: 10.1016/j.csda.2007.01.024.

Irene Klugkist and Thom Benjamin Volker. Bayesian Evidence Synthesis for informative hypotheses: An introduction. *Psychological Methods*, Advance online publication, 2023. doi: 10.1037/met0000602.

Irene Klugkist, Olav Laudy, and Herbert Hoijtink. Inequality constrained analysis of variance: a bayesian approach. *Psychological methods*, 10(4):477–493, 2005. doi: 10.1037/1082-989X.10.4.477.

Rebecca M. Kuiper, Vincent Buskens, Werner Raub, and Herbert Hoijtink. Combining statistical evidence from several studies: A method using bayesian updating and an example from research on trust problems in social and economic exchange. *Sociological Methods & Research*, 42(1):60–81, 2013. doi: 10.1177/0049124112464867.

Daniël Lakens. Performing high-powered studies efficiently with sequential analyses. *European Journal of Social Psychology*, 44(7):701–710, 2014. doi: 10.1002/ejsp.2023.

Debbie A. Lawlor, Kate Tilling, and George Davey Smith. Triangulation in aetiological epidemiology. *International Journal of Epidemiology*, 45:1866–1886, 2017. doi: 10.1093/ije/dyw314.

Mark W Lipsey and David B Wilson. *Practical Meta-Analysis*. SAGE Publications, Thousand Oaks, CA, 2001.

Peter Lipton. *Inference to the best explanation*. Routledge, New York, NY, 2003.

Alexander Ly, Josine Verhagen, and Eric-Jan Wagenmakers. Harold Jeffreys's default Bayes factor hypothesis tests: Explanation, extension, and application in psychology. *Journal of Mathematical Psychology*, 72:19–32, 2016. doi: 10.1016/j.jmp.2015.06.004.

David T. Lykken. What's wrong with psychology, anyway? In D. Chiccetti and W. Grove, editors, *Thinking Clearly About Psychology*, pages 3–39. University of Minnesota Press, 1991.

Scott M. Lynch. *Introduction to Applied Bayesian Statistics and Estimation for Social Scientists*. Springer, New York, 2007. doi: 10.1007/978-0-387-71265-9.

Scott M. Lynch and Bryce Bartlett. Bayesian statistics in sociology: Past, present, and future. *Annual Review of Sociology*, 45(1):47–68, 2019. doi: 10.1146/annurev-soc-073018-022457.

Sandra Mathison. Why triangulate? *Educational researcher*, 17(2):13–17, 1988. doi: 10.3102/0013189X017002013.

Richard D. McKelvey and William Zavoina. A statistical model for the analysis of ordinal level dependent variables. *The Journal of Mathematical Sociology*, 4(1):103–120, 1975. doi: 10.1080/0022250X.1975.9989847.

J. Mulder and A. Olsson-Collentine. Simple bayesian testing of scientific expectations in linear regression models. *Behavior Research Methods*, 51(3):1117–1130, 2019. doi: 10.3758/s13428-018-01196-9.

Joris Mulder. Prior adjusted default Bayes factors for testing (in)equality constrained hypotheses. *Computational Statistics & Data Analysis*, 71:448–463, 2014. doi: 10.1016/j.csda.2013.07.017.

Joris Mulder and Xin Gu. Bayesian testing of scientific expectations under multivariate normal linear models. *Multivariate Behavioral Research*, pages 1–29, 2021. doi: 10.1080/00273171.2021.1904809.

Joris Mulder, Herbert Hoijtink, and Irene Klugkist. Equality and inequality constrained multivariate linear models: Objective model selection using constrained posterior priors. *Journal of Statistical Planning and Inference*, 140(4): 887–906, 2010. doi: 10.1016/j.jspi.2009.09.022.

Joris Mulder, Caspar van Lissa, Donald R. Williams, Xin Gu, Anton Olsson-Collentine, Florian Boeing-Messing, and Jean-Paul Fox. *BFPack: Flexible Bayes Factor Testing of Scientific Expectations*, 2021. URL <https://CRAN.R-project.org/package=BFPack>. R package version 0.3.2.

Marcus R. Munafò, Brian A. Nosek, Dorothy V. M. Bishop, Katherine S. Button, Christopher D. Chambers, Nathalie Percie du Sert, Uri Simonsohn, Eric-Jan Wagenmakers, Jennifer J. Ware, and John P. A. Ioannidis. A manifesto for reproducible science. *Nature Human Behaviour*, 1(1):0021, 2017. doi: 10.1038/s41562-016-0021.

- Marcus R. Munafò and George Davey Smith. Robust research needs many lines of evidence. *Nature*, 553(7689):399–401, 2018. doi: 10.1038/d41586-018-01023-3.
- Brian A. Nosek, Jeffrey R. Spies, and Matt Motyl. Scientific utopia: II. restructuring incentives and practices to promote truth over publishability. *Perspectives on Psychological Science*, 7(6):615–631, 2012. doi: 10.1177/1745691612459058.
- Brian A. Nosek, Tom E. Hardwicke, Hannah Moshontz, Aurélien Allard, Katherine S. Corker, Anna Dreber, Fiona Fidler, Joe Hilgard, Melissa Kline Struhl, Michèle B. Nuijten, Julia M. Rohrer, Felipe Romero, Anne M. Scheel, Laura D. Scherer, Felix D. Schönbrodt, and Simine Vazire. Replicability, robustness, and reproducibility in psychological science. *Annual Review of Psychology*, 73(1):null, 2021. doi: 10.1146/annurev-psych-020821-114157.
- NWO. Replication studies, Mar 2020. URL <https://www.nwo.nl/en/researchprogrammes/replication-studies>.
- Anthony O'Hagan. Fractional bayes factors for model comparison. *Journal of the Royal Statistical Society: Series B (Methodological)*, 57(1):99–118, 1995. doi: 10.1111/j.2517-6161.1995.tb02017.x.
- Open Science Collaboration. Estimating the reproducibility of psychological science. *Science*, 349(6251), 2015. doi: 10.1126/science.aac4716.
- R Core Team. *R: A Language and Environment for Statistical Computing*. R Foundation for Statistical Computing, Vienna, Austria, 2021. URL <https://www.R-project.org/>.
- Richard Royall. *Statistical Evidence: A Likelihood Paradigm*. Routledge, New York, NY, 1997.
- Stefan Schmidt. Shall we really do it again? the powerful concept of replication is neglected in the social sciences. *Review of General Psychology*, 13(2):90–100, 2009. doi: 10.1037/a0015108.
- Felix D Schönbrodt, Eric-Jan Wagenmakers, Michael Zehetleitner, and Marco Perugini. Sequential hypothesis testing with bayes factors: Efficiently testing mean differences. *Psychological methods*, 22(2):322, 2017. doi: 10.1037/met0000061.
- Alex J Sutton and Keith R Abrams. Bayesian methods in meta-analysis and evidence synthesis. *Statistical methods in medical research*, 10(4):277–303, 2001. doi: 10.1177/096228020101000404.
- Jorge N. Tendeiro and Henk A. L. Kiers. A review of issues about null hypothesis bayesian testing. *Psychological Methods*, 24(6):774–795, 2019. doi: 10.1037/met0000221.
- David Trafimow, Valentin Amrhein, Corson N. Areshenkoff, Carlos J. Barrera-Causil, Eric J. Beh, Yusuf K. Bilgiç, Roser Bono, Michael T. Bradley, William M. Briggs, Héctor A. Cepeda-Freyre, Sergio E. Chaingneau, Daniel R. Ciocca, Juan C. Correa, Denis Cousineau, Michiel R. de Boer, Subhra S. Dhar, Igor Dolgov, Juana Gómez-Benito, Marian Grendar, James W. Grice, Martin E. Guerrero-Gimenez, Andrés Gutiérrez, Tania B. Huedo-Medina, Klaus Jaffe, Armina Janyan, Ali Karimnezhad, Fränzi Korner-Nievergelt, Koji Kosugi, Martin Lachmair, Rubén D. Ledesma, Roberto Limongi, Marco T. Liuzza, Rosaria Lombardo, Michael J. Marks, Gunther Meinschmidt, Ladislas Nalborczyk, Hung T. Nguyen, Raydonal Ospina, Jose D. Perezgonzalez, Roland Pfister, Juan J. Rahona, David A. Rodríguez-Medina, Xavier Romão, Susana Ruiz-Fernández, Isabel Suarez, Marion Tegethoff, Mauricio Tejo, Rens Van de Schoot, Ivan I. Vankov, Santiago Velasco-Forero, Tonghui Wang, Yuki Yamada, Felipe C. M. Zoppino, and Fernando Marmolejo-Ramos. Manipulating the alpha level cannot cure significance testing. *Frontiers in Psychology*, 9, 2018. doi: 10.3389/fpsyg.2018.00699.
- Rens Van de Schoot, Herbert Hoijtink, and Jan-Willem Romeijn. Moving beyond traditional null hypothesis testing: Evaluating expectations directly. *Frontiers in Psychology*, 2:24, 2011. doi: 10.3389/fpsyg.2011.00024.
- Rens Van de Schoot, Sonja D Winter, Oisín Ryan, Mariëlle Zondervan-Zwijnenburg, and Sarah Depaoli. A systematic review of Bayesian articles in psychology: The last 25 years. *Psychological Methods*, 22(2):217, 2017. doi: 10.1037/met0000100.
- Thom Benjamin Volker. The future is made today: Concerns for reputation foster trust and cooperation. Master's thesis, Utrecht University, Department of Sociology, 2022. Unpublished.
- Eric-Jan Wagenmakers, Maarten Marsman, Tahira Jamil, Alexander Ly, Josine Verhagen, Jonathon Love, Ravi Selker, Quentin F. Gronau, Martin Šmíra, Sacha Epskamp, Dora Matzke, Jeffrey N. Rouder, and Richard D. Morey. Bayesian inference for psychology. part i: Theoretical advantages and practical ramifications. *Psychonomic Bulletin & Review*, 25(1):35–57, 2018. doi: 10.3758/s13423-017-1343-3.
- A. Zellner and A. Siow. Posterior odds ratios for selected regression hypotheses. *Trabajos de Estadística Y de Investigacion Operativa*, 31(1):585–603, 1980. doi: 10.1007/BF02888369.
- M. A. J. Zondervan-Zwijnenburg, Sabine A.M. Veldkamp, Alexander Neumann, Stefania A. Barzeva, Stefanie A. Nelemans, Catharina E.M. van Beijsterveldt, Susan J.T. Branje, Manon H.J. Hillegers, Wim H.J. Meeus, Henning Tiemeier, Herbert J.A. Hoijtink, Albertine J. Oldehinkel, and Dorret I. Boomsma. Parental age and offspring

childhood mental health: A multi-cohort, population-based investigation. *Child Development*, 91(3):964–982, 2020a. doi: 10.1111/cdev.13267.

M.A.J. Zondervan-Zwijnenburg, J.S. Richards, S.T. Kevenaar, A.I. Becht, H.J.A. Hoijtink, A.J. Oldehinkel, S. Branje, W. Meeus, and D.I. Boomsma. Robust longitudinal multi-cohort results: The development of self-control during adolescence. *Developmental Cognitive Neuroscience*, 45:100817, 2020b. doi: 10.1016/j.dcn.2020.100817.