Bayesian approaches to designing replication studies

Samuel Pawel[1], Guido Consonni[2], and Leonhard Held[1]

[1]Department of Biostatistics, Center for Reproducible Science, University of Zurich

[2]Dipartimento di Scienze Statistiche, Università Cattolica del Sacro Cuore

Author Note

Samuel Pawel https://orcid.org/0000-0003-2779-320X

Guido Consonni https://orcid.org/0000-0002-1252-5926

Leonhard Held https://orcid.org/0000-0002-8686-5325

Correspondence concerning this article should be addressed to Samuel Pawel, Epidemiology, Biostatistics and Prevention Institute, University of Zurich, Hirschengraben 84, 8001 Zurich, Switzerland. E-mail: samuel.pawel@uzh.ch

Abstract

Replication studies are essential for assessing the credibility of claims from original studies. A critical aspect of designing replication studies is determining their sample size; a too small sample size may lead to inconclusive studies whereas a too large sample size may waste resources that could be allocated better in other studies. Here we show how Bayesian approaches can be used for tackling this problem. The Bayesian framework allows researchers to combine the original data and external knowledge in a design prior distribution for the underlying parameters. Based on a design prior, predictions about the replication data can be made, and the replication sample size can be chosen to ensure a sufficiently high probability of replication success. Replication success may be defined through Bayesian or non-Bayesian criteria, and different criteria may also be combined to meet distinct stakeholders and allow conclusive inferences based on multiple analysis approaches. We investigate sample size determination in the normal-normal hierarchical model where analytical results are available and traditional sample size determination is a special case where the uncertainty on parameter values is not accounted for. An application to data from a multisite replication project of social-behavioral experiments illustrates how Bayesian approaches help to design informative and cost-effective replication studies. Our methods can be used through the R package BayesRepDesign.

Keywords: Bayesian design, design prior, multisite replication, sample size determination

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Introduction

The replicability of research findings is a cornerstone for the credibility of science. However, there is growing evidence that the replicability of many scientific findings is lower than expected (Camerer et al., 2018; Errington et al., 2021; Ioannidis, 2005; Open Science Collaboration, 2015). This "replication crisis" has led to methodological reforms in various fields of science, one of which is an increased conduct of replication studies (Munafò et al., 2017). Statistical methodology plays a key role in the evaluation of replication studies, and various methods have been proposed for quantifying how "successful" a replication study was in replicating the original finding (Anderson & Maxwell, 2016; Bayarri & Mayoral, 2002; Bonett, 2020; Etz & Vandekerckhove, 2016; Harms, 2019; Hedges & Schauer, 2019; Held, 2020; Held, Micheloud, & Pawel, 2022; Johnson, Payne, Wang, Asher, & Mandal, 2016; Ly, Etz, Marsman, & Wagenmakers, 2018; Mathur & VanderWeele, 2020; Patil, Peng, & Leek, 2016; Pawel & Held, 2020, 2022; Simonsohn, 2015; van Aert & van Assen, 2017; Verhagen & Wagenmakers, 2014, among others). Yet, as with ordinary studies, statistical methodology is not only important for analyzing replication studies but also for designing them, in particular for their *sample size determination* (SSD). Optimal SSD is important since too small sample sizes may lead to inconclusive studies, whereas too large sample sizes may waste resources which could have been allocated better in other research projects.

SSD for replication studies comes with unique opportunities and challenges; the data from the original study can be used to inform SSD, at the same time the analysis of replication success based on original and replication study is typically different from an analysis of a single study for which traditional SSD methodology was developed. Since analysis and design of replication studies should be in accordance, a relatively small literature has emerged which specifically deals with replication study power calculations and SSD (Anderson & Kelley, 2022; Anderson & Maxwell, 2017; Bayarri & Mayoral, 2002; Goodman, 1992; Hedges & Schauer, 2021; Held, 2020; Micheloud & Held, 2022; Pawel & Held, 2022; Senn, 2002; van Zwet & Goodman, 2022). However, most of these articles only deal with selected analysis methods and data models. An exception is the excellent article by Anderson and Kelley (2022)

which discusses more general principles of replication SSD in the context of psychological research, mostly from a frequentist perspective. As they state "the literature on Bayesian sample size planning is still nascent, particularly with respect to Bayes Factors (Schönbrodt & Wagenmakers, 2018), and has not yet been clearly optimized for the context of most replication goals" (Anderson & Kelley, 2022, p. 18). Our goal is therefore to complement their article by developing a unified framework of replication SSD (schematically illustrated in Figure 1) based on principles from Bayesian design approaches (De Santis, 2004; Gelfand & Wang, 2002; Grieve, 2022; Kunzmann et al., 2021; O'Hagan & Stevens, 2001; Park & Pek, 2022; Pek & Park, 2019; Schönbrodt & Wagenmakers, 2018; Spiegelhalter, Abrams, & Myles, 2004; Spiegelhalter & Freedman, 1986; Spiegelhalter, Freedman, & Blackburn, 1986; Weiss, 1997). We aim to provide both a theoretical basis for methodologists developing new methods for design and analysis methods of replication studies, and also to illustrate how Bayesian design approaches can practically be used by researchers planning a replication study.
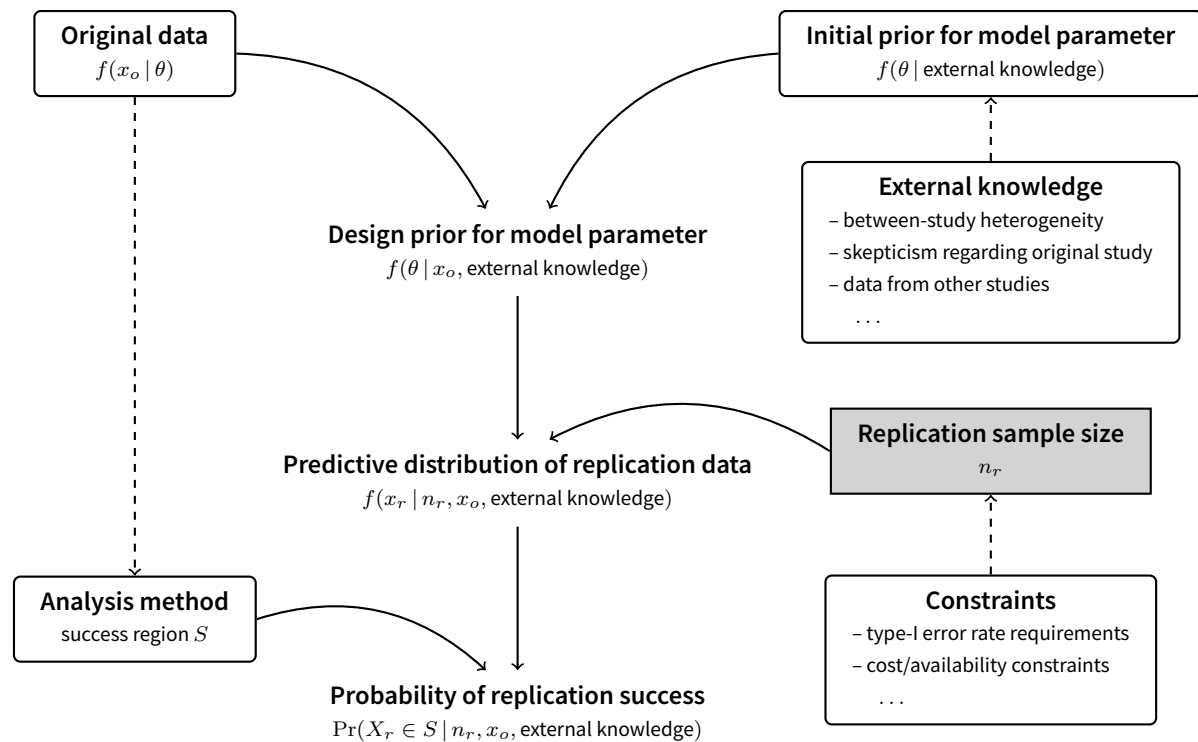
Figure 1

Schematic illustration of Bayesian sample size determination for replication studies. The original and replication data are denoted by x_o and x_r, respectively. Both are assumed to come from a distribution with density/probability mass function denoted by $f(x_i \mid \theta)$ for $i \in \{o, r\}$. An initial prior with density function $f(\theta \mid external\ knowledge)$ is assigned to the model parameter θ.

The design of replication studies is a natural candidate for Bayesian knowledge updating as it allows to combine uncertain information from different sources –for instance, the data from the original study and/or expert knowledge– in a so-called *design prior* distribution for the underlying model parameters. If the analysis of the replication data is also Bayesian, the design prior may be different from the so-called *analysis prior* which, unlike the design prior, is usually desired to be objective or "uninformative" (O'Hagan & Stevens, 2001). Based on the design prior, predictions about the replication data can then be made, and the sample size can be chosen such that the probability of replication success becomes sufficiently high. Importantly, Bayesian design approaches can also be used if the planned analysis of the replication study is non-Bayesian, which is the more common situation in practice. Bayesian design based on a frequentist analysis is known under various names, such as "hybrid classical-Bayesian design" (Spiegelhalter et al., 2004) or "Bayesian assurance" (O'Hagan, Stevens, & Campbell, 2005), and has also been used before for psychological applications (Park & Pek, 2022; Pek & Park, 2019) and replication studies (Anderson & Maxwell, 2017; Micheloud & Held, 2022).

This paper is structured as follows: We start with presenting a general framework for Bayesian SSD of replication studies which applies to any kind of data model and analysis method. We then investigate design priors and SSD in the normal-normal hierarchical model framework which provides sufficient flexibility for incorporating the original data and external knowledge in replication design. No advanced computational methods, such as (Markov Chain) Monte Carlo sampling, are required for conducting Bayesian SSD in this framework, and in many cases there are even simple formulae which generalize classical power and sample size calculations. We illustrate the methodology for several Bayesian and non-Bayesian analysis methods, and for both singlesite and multisite replication studies. Since multisite replication studies are becoming increasingly popular in psychology (e. g., Klein et al., 2018), we also discuss how to choose the optimum allocation of samples within and between sites from a Bayesian design point of view. As a running example we use data from a multisite replication project of social-behavioral experiments (Protzko et al., 2020). Finally, we close with concluding remarks, limitations, and open questions.

General framework

Suppose an original study has been conducted and resulted in a data set x_o. These data are assumed to come from a distribution characterized by an unknown parameter θ and with density function $f(x_o \mid \theta)$. To assess the replicability of a claim from the original study, an independent and identically designed (apart from the sample size) replication study is conducted, and the goal of the design stage is to determine its sample size n_r.

As the observed original data x_o, the yet unobserved replication data X_r are assumed to come from a distribution depending on the parameter θ. The parameter θ thus provides a link between the two studies, and the knowledge obtained from the original study can be used to make predictions about the replication. The central quantity for doing so is the so-called *design prior* of the parameter θ, which we write as the posterior distribution of θ based on the original data and an *initial prior* for θ

$$f(\theta \mid x_o, \text{external knowledge}) = \frac{f(x_o \mid \theta)\, f(\theta \mid \text{external knowledge})}{f(x_o \mid \text{external knowledge})}. \tag{1}$$

The initial prior of θ may depend on external knowledge (e. g., data from other studies) and it represents the uncertainty about θ before observing the original data. We will discuss common types of external knowledge in the replication setting in the next Section. The design prior (1) hence represents the state of knowledge and uncertainty about the parameter θ before the replication is conducted, and, along with an assumed replication sample size n_r, it can be used to compute a predictive distribution for the replication data

$$f(x_r \mid n_r, x_o, \text{external knowledge}) = \int f(x_r \mid n_r, \theta)\, f(\theta \mid x_o, \text{external knowledge})\, \mathrm{d}\theta. \tag{2}$$

After completion of the replication, the observed data x_r will be analyzed in some way to quantify to what extent the original result could be replicated. The analysis may involve the original data (for example, a meta-analysis of the two data sets) or it may only use the replication data. Typically, there is a *success region* S which implies that if the replication data fall within it ($x_r \in S$), the replication is successful. The *probability of replication success* can thus be computed by integrating the predictive density (2) over S. To ensure a sufficiently

conclusive replication design, the sample size n_r is determined such that the probability of replication success is at least as large as a desired target probability of success, here and henceforth denoted by $1 - \beta$. The required sample size n_r^* is then the smallest sample size which leads to a probability of replication success of at least $1 - \beta$, i. e.,

$$n_r^* = \inf \left\{ n_r : \Pr(X_r \in S \,|\, n_r, x_o, \text{external knowledge}) \geq 1 - \beta \right\}. \qquad (3)$$

Often, replication studies are analyzed using several methods which quantify different aspects of replicability, and which have different success regions (e. g., one method for quantifying parameter compatibility and another for quantifying evidence against a null hypothesis). In this case, the sample size may be chosen such that the probability of replication success is as large as desired for all planned analysis methods.

There may sometimes be certain constraints which the replication sample size needs to satisfy. For instance, in most cases there is an upper limit on the possible sample size due to limited resources and/or availability of samples. Moreover, funders and regulators may also require methods to be *calibrated* (Grieve, 2016), that is, to have appropriate type I error rate control. The sample size n_r^* may thus also need to satisfy a type I error rate not larger than some required level.

Sample size determination in the normal-normal hierarchical model

We will now illustrate the general methodology from the previous section in the *normal-normal hierarchical model* where predictive distributions and the probability of replication success can often be expressed in closed-form, permitting further insight. It is pragmatic to adopt a meta-analytic perspective and use only study level summary statistics instead of the raw study data since the raw data from the original study are not always available to the replicators. Typically, the underlying parameter θ is a univariate effect size quantifying the effect on the outcome variable (e. g., a mean difference, a log odds ratio, or a log hazard ratio). The original and replication study can then be summarized through an effect estimate $\hat{\theta}$, possibly the maximum likelihood estimate, and a corresponding standard error σ, i. e., $x_o = \{\hat{\theta}_o, \sigma_o\}$ and $x_r = \{\hat{\theta}_r, \sigma_r\}$. Effect estimates and standard errors are routinely

reported in research articles or can, under some assumptions, be computed from p-values and confidence intervals. As in the conventional meta-analytic framework (Sutton & Abrams, 2001), we further assume that for study $k \in \{o, r\}$ the (suitably transformed) effect estimate $\hat{\theta}_k$ is approximately normally distributed around a study specific effect size θ_k and with (known) variance equal to its squared standard error σ_k^2, here and henceforth denoted by $\hat{\theta}_k \mid \theta_k \sim \mathrm{N}(\theta_k, \sigma_k^2)$. The standard error σ_k is typically of the form $\sigma_k = \lambda / \sqrt{n_k}$ with λ^2 some unit variance and n_k the sample size. The ratio of the original to the replication variance is thus the ratio of the replication to the original sample size

$$c = \sigma_o^2 / \sigma_r^2 = n_r / n_o,$$

which is often the main focus of SSD as it quantifies how much the replication sample n_r size needs to be changed compared to the original sample size n_o. Depending on the effect size type, this framework might require slight modifications (see e. g., Spiegelhalter et al., 2004, ch. 2.4).

Assuming a normal sampling model for the effect estimates (4a), as described previously, and specifying an initial hierarchical normal prior for the study specific effect sizes (4b) and the effect size (4c), leads then to the normal-normal hierarchical model

$$\hat{\theta}_k \mid \theta_k \sim \mathrm{N}(\theta_k, \sigma_k^2) \tag{4a}$$

$$\theta_k \mid \theta \sim \mathrm{N}(\theta, \tau^2) \tag{4b}$$

$$\theta \sim \mathrm{N}(\mu_\theta, \sigma_\theta^2). \tag{4c}$$

By marginalizing over the study specific effects sizes, the model (4) can alternatively be expressed as

$$\hat{\theta}_k \mid \theta \sim \mathrm{N}(\theta, \sigma_k^2 + \tau^2) \tag{5a}$$

$$\theta \sim \mathrm{N}(\mu_\theta, \sigma_\theta^2) \tag{5b}$$

which is often more useful for derivations and computations. In the following we will explain

how the normal-normal hierarchical model can be used for SSD of the replication study.

Design prior and predictive distribution

The observed original data $x_o = \{\hat{\theta}_o, \sigma_o\}$ can be combined with the initial prior (5b) by standard Bayesian theory for normal prior and likelihood (Spiegelhalter et al., 2004, ch. 3.7) to obtain a posterior distribution for the effect size θ

$$\theta \mid \hat{\theta}_o, \sigma_o^2 \sim \mathrm{N}\left(\frac{\hat{\theta}_o}{1 + 1/g} + \frac{\mu_\theta}{1 + g}, \frac{\sigma_o^2 + \tau^2}{1 + 1/g}\right) \tag{6}$$

where $g = \sigma_\theta^2/(\sigma_o^2 + \tau^2)$ is the *relative prior variance*. This posterior serves then as the design prior for predicting the replication data.

It is interesting to contrast the design prior (6) to the "conditional" design prior (Micheloud & Held, 2022), that is, to assume that the unknown effect size θ corresponds to the original effect estimate $\hat{\theta}_o$. This is a standard approach in practice, for instance, Open Science Collaboration (2015) determined the sample sizes of its 100 replications under this assumption. In our framework it implies that the normal design prior (6) becomes a point mass at the original effect estimate $\hat{\theta}_o$, which can either be achieved through overwhelmingly informative original data ($\sigma_o^2 \downarrow 0$) along with no heterogeneity ($\tau^2 = 0$), or through an overwhelmingly informative initial prior ($g \downarrow 0$) centered around the original effect estimate ($\mu_\theta = \hat{\theta}_o$). Both cases show that from a Bayesian perspective the standard approach is unnatural as it either corresponds to making the standard error σ_o smaller than it actually was, or to cherry-picking the prior based on the data.

Based on the design prior (6), the predictive distribution of the replication effect estimate $\hat{\theta}_r$ can then be computed by assuming a replication standard error σ_r and integrating the marginal density of the replication effect estimate (5a) with respect to the prior density, leading to

$$\hat{\theta}_r \mid \hat{\theta}_o, \sigma_o^2, \sigma_r^2 \sim \mathrm{N}\left(\mu_{\hat{\theta}_r} = \frac{\hat{\theta}_o}{1 + 1/g} + \frac{\mu_\theta}{1 + g}, \sigma_{\hat{\theta}_r}^2 = \sigma_r^2 + \tau^2 + \frac{\sigma_o^2 + \tau^2}{1 + 1/g}\right) \tag{7}$$

which can again be shown using standard Bayesian theory (Spiegelhalter et al., 2004, ch. 3.13.3). The design prior (6) and the resulting predictive distribution (7) depend on the

parameters of the initial prior (τ^2, μ_θ, σ_θ^2). We will now explain how these parameters can be specified based on external knowledge.

Incorporating external knowledge in the initial prior

At least three common types of external knowledge can be distinguished in the replication setting: (i) expected heterogeneity between original and replication study due to differences in study design, execution, and population, (ii) prior knowledge about the effect size either from theory or from related studies, (iii) skepticism regarding the original study due to the possibility of exaggerated results.

Between-study heterogeneity

The expected degree of between-study heterogeneity can be incorporated via the variance τ^2 in (4b). As τ^2 decreases, the study specific effect sizes become more similar, whereas for increasing τ^2 they become more unrelated. If the replicators do not expect any heterogeneity they can thus set $\tau^2 = 0$ which will lead to the model collapsing to a fixed effects model.

If heterogeneity is expected, there are different approaches for specifying τ^2. A domain expert may subjectively assess how much heterogeneity is to be expected due to the change in laboratory, study population, and other factors. An alternative is to take an estimate from the literature, e. g., from multisite replication projects or from systematic reviews. Finally, one can also specify an upper limit of "tolerable heterogeneity". This approach is similar to specifying a minimal clinically relevant difference in classical power analysis in the sense that a true replication effect size which is intolerably heterogeneous from the original effect size is not relevant to be detected. An absolute (Spiegelhalter et al., 2004, ch. 5.7.3) and a relative approach (Held & Pawel, 2020) can be considered. In the absolute approach, a value of τ^2 is chosen such that a suitable range of study-specific effect sizes is not larger than an effect size difference considered negligible. For example, when 95% of the study specific effect sizes should not vary more than a small effect size e. g., $d = 0.2$ on standardized mean difference scale based on the Cohen (1992) effect size classification, this would lead to $\tau = d/(2 \cdot 1.96) \approx 0.05$. In the relative approach, τ^2 is specified relative to the variance of the original estimate σ_o^2 using field conventions for tolerable relative heterogeneity. For example,

in the Cochrane guidelines for systematic reviews (Deeks, Higgins, & Altman, 2019) a value of $I^2 = \tau^2/(\tau^2 + \sigma_o^2) = 40\%$ is classified as "negligible", which translates to $\tau^2 = \sigma_o^2/(1/I^2 - 1) = (2\sigma_o^2)/3$.

We note that one can also assign a prior distribution to τ^2 (for an overview of prior distributions for heterogeneity variances in the normal-normal hierarchical model see Röver et al., 2021). In this case there is no closed-form expression for the predictive distribution of the replication effect estimate but numerical or Monte Carlo integration need to be used. We illustrate in the supplement how the probability of replication success can in this case be computed. The derived closed-form expressions conditional on τ^2 are still useful as they enable computation of the predictive distribution up to a single one-dimensional integral which can be computed numerically.

Knowledge about the effect size

Prior knowledge about the effect size θ can be incorporated via the prior mean μ_θ and the prior variance σ_θ^2 in (4c). For instance, the parameters may be specified based on a meta-analysis of related studies (McKinney, Stefan, & Gronau, 2021) or based on expert elicitation (O'Hagan, 2019). The resulting design prior will then contain more information than what was provided by the original data alone, leading to potentially more efficient designs. If there is no prior knowledge available, a standard approach is to specify an (improper) flat prior by letting the variance go to infinity ($\sigma_\theta^2 \to \infty$). The resulting design prior will then only contain the information from the original study.

Exaggerated original results

Potentially exaggerated original results can be counteracted by setting $\mu_\theta = 0$ which shrinks the design prior towards smaller effect sizes (in absolute value) than the observed effect estimate $\hat{\theta}_o$. For instance, replicators could believe that the results from the original study are exaggerated because there is no pre-registered study protocol available. Even without such beliefs, weakly informative shrinkage priors may also be motivated from a "regularization" point of view as they can correct for statistical biases (Copas, 1983; Firth, 1993) or prevent unreasonable parameter values from taking over the posterior in settings with uninformative data (Gelman, 2009).

The amount of shrinkage is determined via the prior variance σ_θ^2. A diffuse prior $(\sigma_\theta^2 \to \infty)$ will lead to no shrinkage, while a highly concentrated prior $(\sigma_\theta^2 \downarrow 0)$ will completely shrink the design prior to a point mass on zero. One option for specifying σ_θ^2 is to use an estimate from a corpus of related studies. For instance, van Zwet, Schwab, and Senn (2021) used the Cochrane library of systematic reviews to specify design priors for hypothetical replication studies of RCTs. If no corpus is available, a pragmatic alternative is to use the empirical Bayes estimate based on the original data

$$\hat\sigma_\theta^2 = \max\{(\hat\theta_o - \mu_\theta)^2 - \tau^2 - \sigma_o^2, 0\}. \tag{8}$$

The estimate (8) will lead to adaptive shrinkage (Pawel & Held, 2020) in the sense that shrinkage is large for unconvincing original studies (those with small effect estimates in absolute value $|\hat\theta_o|$ and/or large standard errors σ_o), but disappears as the data become more convincing (through larger effect estimates in absolute value $|\hat\theta_o|$ and/or smaller standard errors σ_o).

Example: Cross-laboratory replication project

We will now illustrate the construction of design priors based on data from a recently conducted replication project (Protzko et al., 2020), see Figure 2 for a summary of the data. The data were collected in four laboratories which, over the course of five years, conducted their typical social-behavioral experiments on topics such as psychology, communication, or political science. From the experiments conducted in this period, each lab submitted four original findings to be replicated. For instance, the original finding from the "Labels" experiment was: "When a researcher uses a label to describe people who hold a certain opinion, he or she is interpreted as disagreeing with those attributes when a negative label is used and agreeing with those attributes when a positive label is used" (Protzko et al., 2020, p. 17), which was based on an effect estimate $\hat\theta_o = 0.205$ with 95% confidence interval from 0.11 to 0.3. For each submitted original finding, four replication studies were then carried out, one by the same lab (a *self-replication*) and three by the other three labs (three *external-replications*).
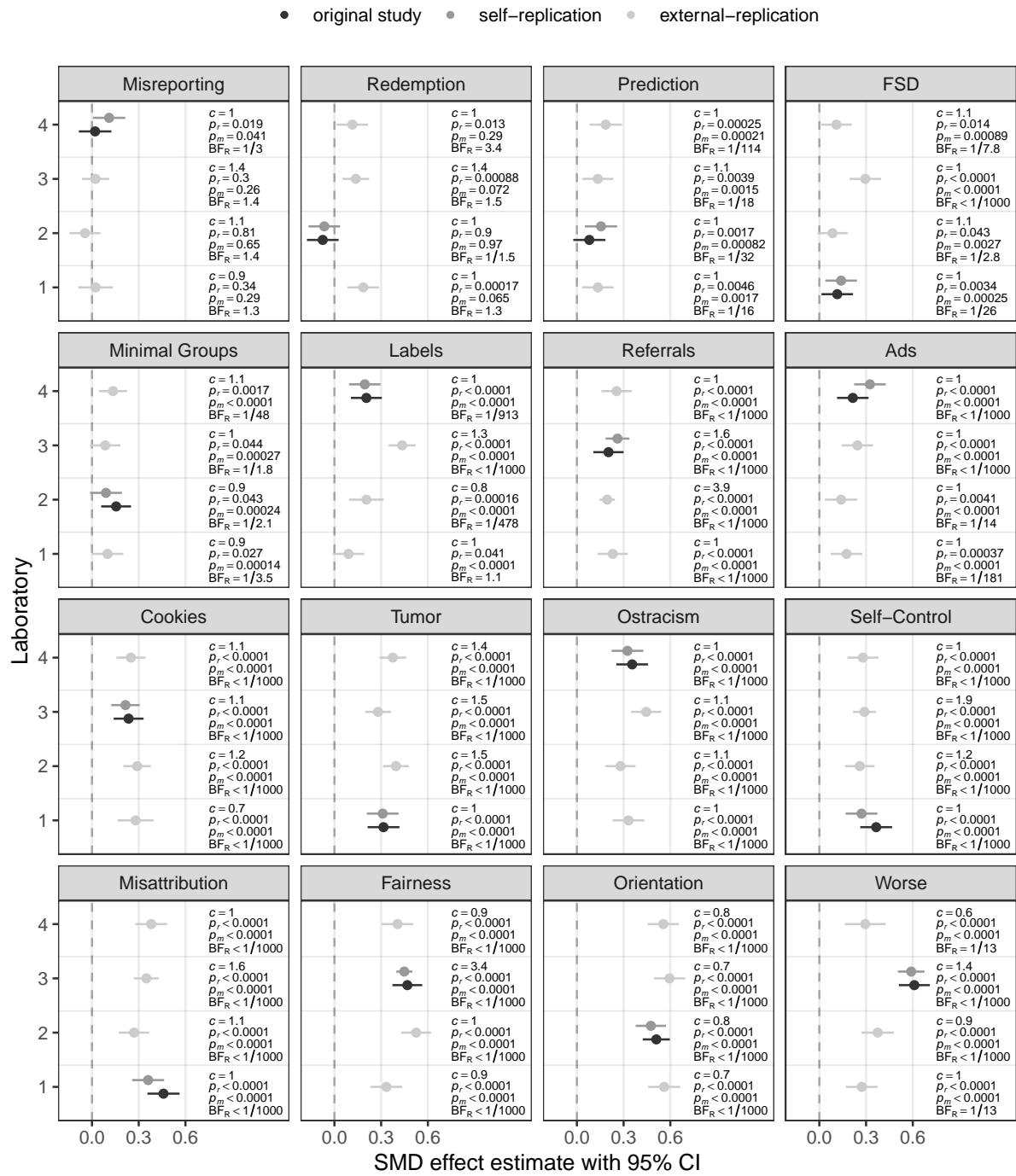
Figure 2

Data from cross-laboratory replication project by Protzko et al. (2020). Shown are standardized mean difference (SMD) effect estimates with 95% confidence intervals stratified by experiment and laboratory. For each replication study, the relative sample size $c = n_r/n_o$, the one-sided replication p-value p_r, the one-sided meta-analytic p-value p_m, and the replication Bayes factor BF_R are shown. Experiments are ordered (left to right, top to bottom) by their original one-sided p-value $p_o = 1 - \Phi(|\hat{\theta}_o|/\sigma_o)$

Most studies used simple between-subject designs with two groups and a continuous outcome so that for a study $i \in \{o, r\}$ the standardized mean difference (SMD) effect estimate $\hat{\theta}_i$ can be computed from the group means $\bar{y}_{i1}, \bar{y}_{i2}$, group standard deviations s_{i1}, s_{i2}, and group sample sizes n_{i1}, n_{i2} by

$$\hat{\theta}_i = \frac{\bar{y}_{i1} - \bar{y}_{i2}}{s_i}$$

with $s_i^2 = \{(n_{i1} - 1)s_{i1}^2 + (n_{i2} - 1)s_{i2}^2\}/(n_{i1} + n_{i2} - 2)$ the pooled sample variance. Under a normal sampling model and assuming equal variances in both groups, the approximate variance of $\hat{\theta}_i$ is

$$\sigma_i^2 = \frac{n_{i1} + n_{i2}}{n_{i1}n_{i2}} + \frac{\hat{\theta}_i^2}{2(n_{i1} + n_{i2})} \tag{9}$$

(Hedges, 1981). A cruder, but for SSD more useful, approximation $\sigma_i^2 \approx 4/n_i$ is obtained by assuming the same sample size in both groups $n_{i1} = n_{i2} = n_i/2$, with n_i the total sample size, and neglecting the second term in (9) which will be close to zero for small effect estimates and/or large sample sizes (Hedges & Schauer, 2021). We thus have the approximate unit variance $\lambda^2 = 4$ and the relative variance $c = \sigma_o^2/\sigma_r^2 = n_r/n_o$, which can be interpreted as the ratio of the replication to the original sample size.

Suppose now the original studies have been finished, and we want to conduct SSD for the not yet conducted replication studies. We start by specifying the design priors (one for each replication). Since the original studies have been preregistered, we do not expect an exaggeration of their effect estimates due to selective reporting or other questionable research practices. Therefore, we choose an uninformative initial prior ($g \to \infty$), which leads to design prior and predictive distribution both centered around the original effect estimate $\hat{\theta}_o$.

For specifying the between-study heterogeneity variance τ^2, a distinction needs to be made between self-replications and external-replications. For self-replications it is reasonable to set $\tau^2 = 0$ because we would expect no between-study heterogeneity as the experimental conditions will be nearly identical in both studies. In contrast, one would expect some between-study heterogeneity for external-replications as the experimental conditions may

slightly differ between the labs. In the following, we will use $\tau^2 = 0.05^2$ elicited via the "absolute" approach as discussed in previously, so that the range between the 2.5% and the 97.5% quantile of the study specific effect size distribution is equal to a small effect size $d = 0.2$.
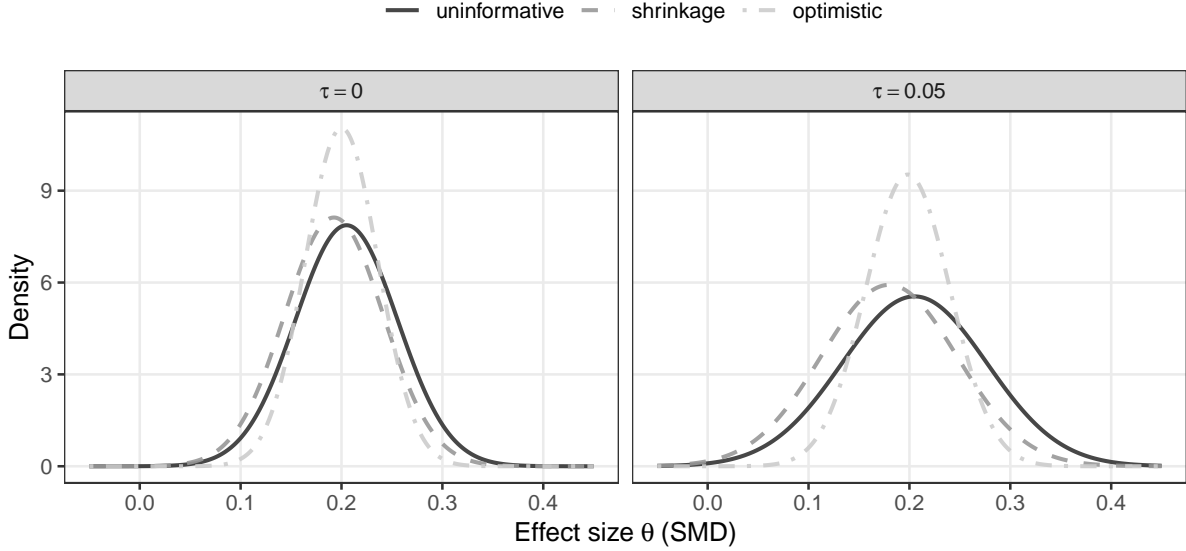
Figure 3

Design priors for the effect size θ (SMD) in the experiment "Labels" based on the original effect estimate $\hat{\theta}_o = 0.205$ with standard error $\sigma_o = 0.051$. Shown are different choices for the between-study heterogeneity τ and the initial prior for the effect size θ, "uninformative" corresponds to a flat prior, "shrinkage" corresponds to a zero-mean normal prior with empirical Bayes variance estimate (8), and "optimistic" corresponds to a flat prior updated by the data from a pilot study with effect estimate $\hat{\theta}_p = 0.195$ and standard error $\sigma_p = 0.052$.

Taken together, we obtain the design prior $\theta \mid \hat{\theta}_o, \sigma_o^2 \sim \mathrm{N}(\hat{\theta}_o, \sigma_o^2)$ for self-replications and the design prior $\theta \mid \hat{\theta}_o, \sigma_o^2 \sim \mathrm{N}(\hat{\theta}_o, \sigma_o^2 + \tau^2)$ with $\tau^2 = 0.05^2$ for external-replications. For example, for the experiment "Labels", the design prior would be centered around the original effect estimate $\hat{\theta}_o = 0.205$ with variance $\sigma_o^2 = 0.05^2$ for a self-replication, and with variance $\sigma_o^2 + \tau^2 = 0.05^2 + 0.05^2 \approx 0.07^2$ for an external-replication. Figure 3 (dark-gray solid lines) shows the densities of the two priors.

While these two priors seem sensible for the Protzko et al. (2020) data, it is interesting to think about alternative scenarios. If there had been reasons to believe that the original result might be exaggerated, we could have specified an initial shrinkage prior ($\mu_\theta = 0$ and

$\sigma_\theta^2 < \infty$). For instance, the empirical Bayes estimate for the prior variance σ_θ^2 from (8) leads to a prior whose mean and variance are shrunken towards zero by 12% (medium-gray dashed lines in Figure 3). In contrast, if we had prior knowledge about the effect size θ from another study, we could have specified an initial "optimistic" prior. For example, if the self-replication of the "Labels" experiment had been a pilot study and we used its effect estimate $\hat{\theta}_p = 0.195$ and standard error $\sigma_p = 0.05$ to specify the initial prior, this would lead to a design prior centered around the weighted mean of original and pilot study, and a prior precision equal to the sum of the precision of both estimates (light-gray dot-dashed lines in Figure 3). Due to the inclusion of the external data, this design prior is much more concentrated than the other two.

Probability of replication success and required sample size

To compute the probability of replication success one needs to select an analysis method and integrate the predictive distribution (7) over the associated success region S. There is no universally accepted method for quantifying replicability and here we do not intend to contribute to the debate about the most appropriate method. We will simply show the success regions of different methods, and how the replication sample size can be computed from them. Some methods depend on the direction of the original effect estimate $\hat{\theta}_o$ and throughout we will assume that it was positive ($\hat{\theta}_o > 0$). Functions for computing the probability of replication success and the required sample size are implemented in the R package **BayesRepDesign** (see Appendix) for all analysis methods discussed in the following.

The two-trials rule

The most common approach for the analysis of replication studies is to declare replication success when both the original and replication study lead to a p-value for testing the null hypothesis $H_0 \colon \theta = 0$ smaller than a pre-specified threshold α, usually $\alpha = 5\%$ for two-sided tests and $\alpha = 2.5\%$ for one-sided tests. This procedure is known as the *two-trials rule* in drug regulation (Senn, 2008).

We now assume that the one-sided original p-value was significant at some level α, i.e., $p_o = 1 - \Phi(\hat{\theta}_o/\sigma_o) \leq \alpha$. Replication success at level α is then achieved if the replication

p-value is also significant, i. e., $p_r = 1 - \Phi(\hat{\theta}_r/\sigma_r) \leq \alpha$, which implies a success region

$$S_{\text{2TR}} = [z_\alpha \, \sigma_r, \infty) \,,$$

where z_α is the $1 - \alpha$ quantile of the standard normal distribution. The probability of replication success is thus given by

$$\Pr(\hat{\theta}_r \in S_{\text{2TR}} \,|\, \hat{\theta}_o, \sigma_o, \sigma_r) = \Phi \left(\frac{\mu_{\hat{\theta}_r} - z_\alpha \, \sigma_r}{\sigma_{\hat{\theta}_r}} \right) \tag{10}$$

with $\Phi(\cdot)$ the standard normal cumulative distribution function and $\mu_{\hat{\theta}_r}$ and $\sigma_{\hat{\theta}_r}$ the mean and standard deviation of the predictive distribution (7). Importantly, by decreasing the standard error σ_r (through increasing the sample size n_r), the probability of replication success (10) cannot become arbitrarily large but is bounded from above by

$$\lim\Pr_{\text{2TR}} = \Phi \left(\frac{\mu_{\hat{\theta}_r}}{\sqrt{\tau^2 + (\sigma_o^2 + \tau^2)/(1 + 1/g)}} \right) . \tag{11}$$

The required replication standard error σ_r^* to achieve a target probability of replication success $1 - \beta < \lim\Pr_{\text{2TR}}$ can now be obtained by equating (10) to $1 - \beta$ and solving for σ_r. This leads to

$$\sigma_r^* = \frac{\mu_{\hat{\theta}_r} z_\alpha - z_\beta \sqrt{(z_\alpha^2 - z_\beta^2) \left\{ \tau^2 + (\sigma_o^2 + \tau^2)/(1 + 1/g) \right\} + \mu_{\hat{\theta}_r}^2}}{z_\alpha^2 - z_\beta^2} \tag{12}$$

for $\alpha < \beta$. The standard error σ_r^* can subsequently be translated in a sample size. The translation depends on the type of effect size, for instance, for SMD effect sizes we can use the approximation $n_r^* \approx 4/(\sigma_r^*)^2$ from earlier. Moreover, by assuming a standard error of the form $\sigma_r = \lambda/\sqrt{n_r}$ and plugging in the parameters of the "conditional" design prior ($\tau^2 = 0$, $\mu_\theta = \hat{\theta}_o$, $g \downarrow 0$), we obtain the well-known sample size formula (Matthews, 2006, ch. 3.3)

$$n_r^* = \frac{(z_\alpha + z_\beta)^2}{(\hat{\theta}_o/\lambda)^2}$$

for a one-sided significance test at level α with power $1 - \beta$ to detect the original effect

estimate $\hat{\theta}_o$. The formula (12) thus generalizes standard sample size calculation to take into account the uncertainty of the original estimate, between-study heterogeneity and other types of external knowledge.

Fixed effects meta-analysis

The data from the original and replication studies are sometimes pooled via fixed-effects meta-analysis. The pooled effect estimate $\hat{\theta}_m$ and standard error σ_m are then given by

$$\hat{\theta}_m = \left(\hat{\theta}_o/\sigma_o^2 + \hat{\theta}_r/\sigma_r^2\right)\sigma_m^2 \qquad \text{and} \qquad \sigma_m = \left(1/\sigma_o^2 + 1/\sigma_r^2\right)^{-1/2},$$

and they are also equivalent to the mean and standard deviation of a posterior distribution for the effect size θ based on the data from both studies and an initial flat prior for θ. The success region

$$S_{\text{MA}} = \left[\sigma_r z_\alpha \sqrt{1 + \sigma_r^2/\sigma_o^2} - (\hat{\theta}_o\sigma_r^2)/\sigma_o^2, \infty\right) \tag{13}$$

then corresponds to both replication success defined via a one-sided meta-analytic p-value being smaller than level α, i.e., $p_m = 1 - \Phi(\hat{\theta}_m/\sigma_m) \leq \alpha$, or to replication success defined via a Bayesian posterior probability $\Pr(\theta > 0 \,|\, \hat{\theta}_o, \hat{\theta}_r, \sigma_o, \sigma_r) \geq 1 - \alpha$. Based on the success region (13) and an assumed standard error σ_r, the probability of replication success can be computed by

$$\Pr(\hat{\theta}_r \in S_{\text{MA}} \,|\, \hat{\theta}_o, \sigma_o, \sigma_r) = \Phi\left(\frac{\mu_{\hat{\theta}_r} - \sigma_r z_\alpha \sqrt{1 + \sigma_r^2/\sigma_o^2} + (\hat{\theta}_o\sigma_r^2)/\sigma_o^2}{\sigma_{\hat{\theta}_r}}\right). \tag{14}$$

As for the two-trials rule, the probability (14) cannot be made arbitrarily large by decreasing the standard error σ_r but is bounded from above by $\text{limPr}_{\text{2TR}}$ defined in (11). The required standard error σ_r^* to achieve a target probability of replication success $1 - \beta < \text{limPr}_{\text{2TR}}$ can be computed numerically using root finding algorithms.

Effect size equivalence test

Anderson and Maxwell (2016) proposed a method for quantifying replicability based on effect size equivalence. Under normality, replication success at level α is achieved if the $(1 - \alpha)$ confidence interval for the effect size difference $\theta_r - \theta_o$

$$\hat{\theta}_r - \hat{\theta}_o \pm z_{\alpha/2}\sqrt{\sigma_r^2 + \sigma_o^2}$$

is fully inside an equivalence region $[-\Delta, \Delta]$ defined via the pre-specified margin $\Delta > 0$. This procedure corresponds to rejecting the null hypothesis $H_0 \colon |\theta_r - \theta_o| > \Delta$ in an equivalence test, and it implies a success region for the replication effect estimate $\hat{\theta}_r$ given by

$$S_{\mathrm{E}} = \left[\hat{\theta}_o - \Delta + z_{\alpha/2}\sqrt{\sigma_o^2 + \sigma_r^2}, \hat{\theta}_o + \Delta - z_{\alpha/2}\sqrt{\sigma_o^2 + \sigma_r^2} \right] \tag{15}$$

for $\Delta \geq z_{\alpha/2}\sqrt{\sigma_o^2 + \sigma_r^2}$. For too small margins ($\Delta < z_{\alpha/2}\sqrt{\sigma_o^2 + \sigma_r^2}$), the success region (15) becomes the empty set meaning that replication success is impossible. Assuming now that the margin is large enough, the probability of replication success can be computed by

$$\Pr(\hat{\theta}_r \in S_{\mathrm{E}} \,|\, \hat{\theta}_o, \sigma_o, \sigma_r) = \Phi\left(\frac{\hat{\theta}_o + \Delta - z_{\alpha/2}\sqrt{\sigma_o^2 + \sigma_r^2} - \mu_{\hat{\theta}_r}}{\sigma_{\hat{\theta}_r}} \right)$$
$$- \Phi\left(\frac{\hat{\theta}_o - \Delta + z_{\alpha/2}\sqrt{\sigma_o^2 + \sigma_r^2} - \mu_{\hat{\theta}_r}}{\sigma_{\hat{\theta}_r}} \right). \tag{16}$$

As with the previous methods, the probability (16) cannot be made arbitrarily large by decreasing the replication standard error σ_r, but is bounded by

$$\mathrm{limPr}_{\mathrm{E}} = \Phi\left(\frac{\hat{\theta}_o + \Delta - z_{\alpha/2}\sigma_o - \mu_{\hat{\theta}_r}}{\sqrt{\tau^2 + (\sigma_o^2 + \tau^2)/(1 + 1/g)}} \right) - \Phi\left(\frac{\hat{\theta}_o - \Delta + z_{\alpha/2}\sigma_o - \mu_{\hat{\theta}_r}}{\sqrt{\tau^2 + (\sigma_o^2 + \tau^2)/(1 + 1/g)}} \right).$$

The required replication standard error σ_r^* to achieve a target probability of replication success $1 - \beta < \mathrm{limPr}_{\mathrm{E}}$ can again be computed numerically.

The replication Bayes factor

A Bayesian hypothesis testing approach for assessing replication success was proposed by Verhagen and Wagenmakers (2014) and further developed by Ly et al. (2018). They define a "replication Bayes factor"

$$\mathrm{BF_R} = \frac{f(x_r \mid H_0)}{f(x_r \mid H_1)}$$

which is the ratio of the marginal likelihoods of the replication data x_r under the null hypothesis $H_0 \colon \theta = 0$ to the marginal likelihood of x_r under the alternative hypothesis $H_1 \colon \theta \sim f(\theta \mid x_o)$, that is the posterior of the effect size θ based on the original data x_o. If the original study provides evidence against the null hypothesis, replication Bayes factor values $\mathrm{BF_R} < 1$ indicate replication success, and the smaller the value the higher the degree of success.

Under normality and assuming no heterogeneity, the success region for achieving $\mathrm{BF_R} \leq \gamma$ is given by

$$S_{\mathrm{BF_R}} = \left(-\infty, -\sqrt{A} - (\hat{\theta}_o \sigma_r^2)/\sigma_o^2\right] \bigcup \left[\sqrt{A} - (\hat{\theta}_o \sigma_r^2)/\sigma_o^2, \infty\right) \tag{17}$$

with $A = \sigma_r^2(1 + \sigma_r^2/\sigma_o^2)\{\hat{\theta}_o^2/\sigma_o^2 - 2\log\gamma + \log(1 + \sigma_o^2/\sigma_r^2)\}$. Details of this calculation are given in the supplement. The fact that the success region (17) is defined on both sides around zero shows that replication success is also possible if the replication effect estimate goes in opposite direction of the original one, which is known as the "replication paradox" (Ly et al., 2018). The paradox can be avoided using a modified version of the replication Bayes factor but the success region is no longer available in closed-form (Pawel & Held, 2022, Appendix D). Based on the success region (17), the probability of replication success can be computed by

$$\Pr(\hat{\theta}_r \in S_{\mathrm{BF_R}} \mid \hat{\theta}_o, \sigma_o, \sigma_r) = \Phi\left(\frac{\mu_{\hat{\theta}_r} - \sqrt{A} + (\hat{\theta}_o \sigma_r^2)/\sigma_o^2}{\sigma_{\hat{\theta}_r}}\right) + \Phi\left(\frac{-\sqrt{A} - (\hat{\theta}_o \sigma_r^2)/\sigma_o^2 - \mu_{\hat{\theta}_r}}{\sigma_{\hat{\theta}_r}}\right).$$

$$\tag{18}$$

One may want to compute the probability of replication success only for the part of the success region with the same sign as the original effect estimate to avoid the replication paradox. As for the other methods, the probability (18) is bounded from above by a constant $\text{limPr}_{\text{BF}_\text{R}} = \lim_{\sigma_r \downarrow 0} \Pr(\hat{\theta}_r \in S_{\text{BF}_\text{R}} \,|\, \hat{\theta}_o, \sigma_o, \sigma_r)$, and root finding algorithms can be used to numerically determine the required standard error σ_r^* for achieving a target probability of replication success $1 - \beta < \text{limPr}_{\text{BF}_\text{R}}$.

The skeptical p-value

Held (2020) proposed a reverse-Bayes approach for quantifying replication success. The main idea is to determine the variance of a "skeptical" zero-mean normal prior for the effect size θ such that its posterior distribution based on the original study is no longer credible. Replication success is then achieved if the replication data are in conflict with the skeptical prior. The procedure can be summarized by a "skeptical p-value" p_s, and the lower the p-value the higher the degree of replication success. Held, Micheloud, and Pawel (2022, sec. 2.1) showed that the success region for replication success defined by $p_\text{s} \leq \alpha$ is given by

$$S_{p_\text{S}} = \left[z_\alpha \sqrt{\sigma_r^2 + \frac{\sigma_o^2}{(z_o^2/z_\alpha^2) - 1}}, \infty \right). \tag{19}$$

From the success region (19) the probability of replication success at level α is

$$\Pr(\hat{\theta}_r \in S_{p_\text{S}} \,|\, \hat{\theta}_o, \sigma_o, \sigma_r) = \Phi\left(\frac{\mu_{\hat{\theta}_r} - z_\alpha \sqrt{\sigma_r^2 + \sigma_o^2/\{(z_o^2/z_\alpha^2) - 1\}}}{\sigma_{\hat{\theta}_r}} \right),$$

and also bounded from above by a constant $\text{limPr}_{p_\text{S}} = \lim_{\sigma_r \downarrow 0} \Pr(\hat{\theta}_r \in S_{p_\text{S}} \,|\, \hat{\theta}_o, \sigma_o, \sigma_r)$. As for the two-trials rule, the required standard error σ_r^* to achieve a probability of replication success $1 - \beta < \text{limPr}_{p_\text{S}}$ can be computed analytically for $\alpha < \beta$:

$$\sigma_r^* = \sqrt{x^2 - \frac{\sigma_o^2}{(z_o/z_\alpha)^2 - 1}}$$

with

$$x = \frac{z_\alpha \mu_{\hat{\theta}_r} - z_\beta \sqrt{\mu_{\hat{\theta}_r}^2 - (z_\alpha^2 - z_\beta^2)[\tau^2 + (\sigma_o^2 + \tau^2)/(1 + 1/g) - \sigma_o^2/\{(z_o/z_\alpha)^2 - 1\}]}}{z_\alpha^2 - z_\beta^2}.$$

The skeptical Bayes factor

Pawel and Held (2022) modified the previously described reverse-Bayes assessment of replication success from Held (2020) to work with Bayes factors instead of tail probabilities as measures of evidence and prior data conflict. Again, the procedure can be summarized in a single measure termed the "skeptical Bayes factor" BF_s, with lower values of BF_s pointing to higher degrees of replication success. Also for this method, the success region and the probability of replication success can be expressed in closed-form but the derivations are more involved than for the other methods. For this reason, they are only given in the supplement.

Example: Cross-laboratory replication project (continued)

We will now revisit the experiment "Labels" and compute the probability of replication success. The parameters of the analysis methods are specified as follows: For the two-trials rule we use the conventional one-sided significance level $\alpha = 0.025$, while for meta-analysis we use the more stringent level $\alpha = 0.025^2$ as the method is based on two data sets rather than one. We use a $1 - \alpha = 90\%$ confidence interval which is conventionally used in equivalence testing, along with a margin $\Delta = 0.2$ corresponding to a small SMD effect size according to the classification from Cohen (1992). For the skeptical p-value we use the recommended "golden" level $\alpha = 0.062$ as it guarantees that for original studies which where just significant at $\alpha = 0.025$ replication success is only possible if the replication effect estimate is larger than the original one (Held, Micheloud, & Pawel, 2022). Finally, for the replication Bayes factor and the skeptical Bayes factor we use the "strong evidence" level $\gamma = 1/10$ from Jeffreys (1961).

Figure 4 shows the probability of replication success as a function of the relative sample size $c = n_r/n_o$ and for different initial priors. The left and middle plot are based on an uninformative prior for the effect size ($\sigma_\theta^2 = \infty$) without heterogeneity ($\tau^2 = 0$) and with heterogeneity ($\tau^2 = 0.05^2$), respectively. The right plot shows the prior corresponding to the
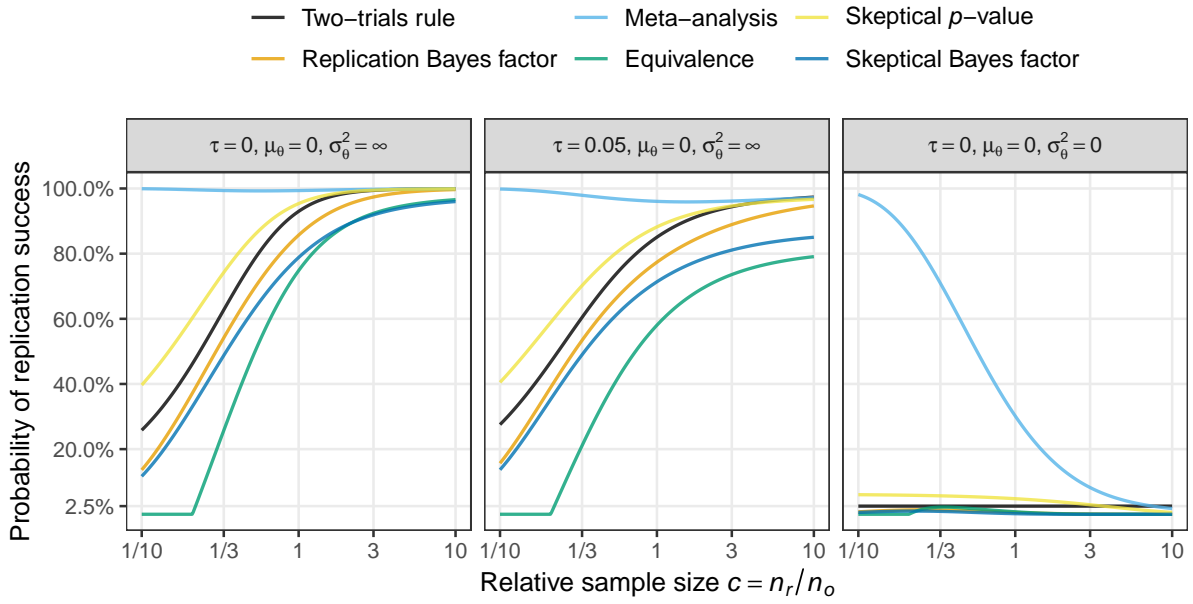
Figure 4

Probability of replication success as a function of relative sample size $c = n_r/n_o$ for experiment "Labels" with original effect estimate $\hat{\theta}_o = 0.205$ and standard error $\sigma_o = 0.051$ for different initial prior parameters $(\tau, \mu_\theta, \sigma_\theta^2)$. The probability of replication success with the design prior $\tau = 0, \mu_\theta = 0$, and $\sigma_\theta^2 = 0$ (right plot) corresponds to the type I error rate under the fixed effects null hypothesis ($H_0: \theta = 0, \tau^2 = 0$). Replication success is defined by the two-trials rule at level $\alpha = 0.025$, the replication Bayes factor at level $\gamma = 1/10$, fixed effects-meta analysis at level $\alpha = 0.025^2$, effect size equivalence based on 90% confidence interval and with margin $\Delta = 0.2$, skeptical p-value at level $\alpha = 0.062$, and skeptical Bayes factor at level $\gamma = 1/10$.

"fixed effects null hypothesis" $H_0: \theta = 0, \tau^2 = 0$, so that the probability of replication success is the type I error rate which some stakeholders might require to be "controlled" at some adequate level.

We see from the left and middle plots that increasing the relative sample size monotonically increases the probability of replication success for all methods but meta-analysis (light blue). Meta-analysis shows a non-monotone behavior because the original study was already highly significant so that the pooled effect estimate is significant even for replication studies with very small sample size (Micheloud & Held, 2022). The uncertainty regarding the replication effect estimate $\hat{\theta}_r$ may therefore even reduce the probability of replication success for meta-analysis. If heterogeneity is taken into account (middle plot) the probability of replication success becomes closer to 50% for all methods but

the equivalence test, reflecting the larger uncertainty about the effect size θ. To achieve 80% probability of replication success the fewest samples are required with meta-analysis, followed by the skeptical p-value, the two-trials rule, the replication Bayes factor, the skeptical Bayes factor, and lastly the equivalence test. If the chosen sample size should guarantee a sufficiently conclusive replication study with all these methods, the replication sample size has to be slightly larger than the original one in the situation of no heterogeneity ($\tau^2 = 0$), while it has to be increased more than ten-fold if there is heterogeneity ($\tau^2 = 0.05^2$). However, this is mostly due to the equivalence test which requires by far the most samples. If the equivalence test sample size is ignored, the relative sample size $c = 2.5$ ensures at least 80% probability of replication success with the remaining methods.

The right plot in Figure 4 shows that the type I error rate of the two-trials rule (black) stays constant at $\alpha = 0.025$, as expected by definition of the method. In contrast, the type I error rates of the other methods vary with the relative sample size c but most of them stay below $\alpha = 0.025$ for all c with the exception of meta-analysis and the skeptical p-value. Meta-analysis (light blue) has an extremely high type I error rate as the pooling with the highly significant original data leads to replication success if the replication sample size is not drastically increased. The type I error rate of the skeptical p-value (yellow) is only slightly larger than $\alpha = 0.025$ which is expected since the level $\alpha = 0.062$ is used for declaring replication success with the skeptical p-value, and its type I error rate is always smaller than the level for thresholding it (Held, 2020). The type I error rate of the skeptical p-value decreases to values smaller than $\alpha = 0.025$ of the two-trials rule at approximately $c = 3$.

We now conduct SSD for all studies from the replication project of Protzko et al. (2020). Figure 5 shows the required relative sample size and the associated type I error rates if a sample size can be computed for a probability of replication success of $1 - \beta = 80\%$. If there is no sample size for which a probability of 80% can be achieved the space is left blank. This is, for instance, the case for the meta-analysis method for all studies below the "Labels" experiment as the probability stays above 80% for any relative sample size.

We see that for all methods but the equivalence test the required relative sample size c decreases with decreasing original p-value p_o, and original studies with very small p-values
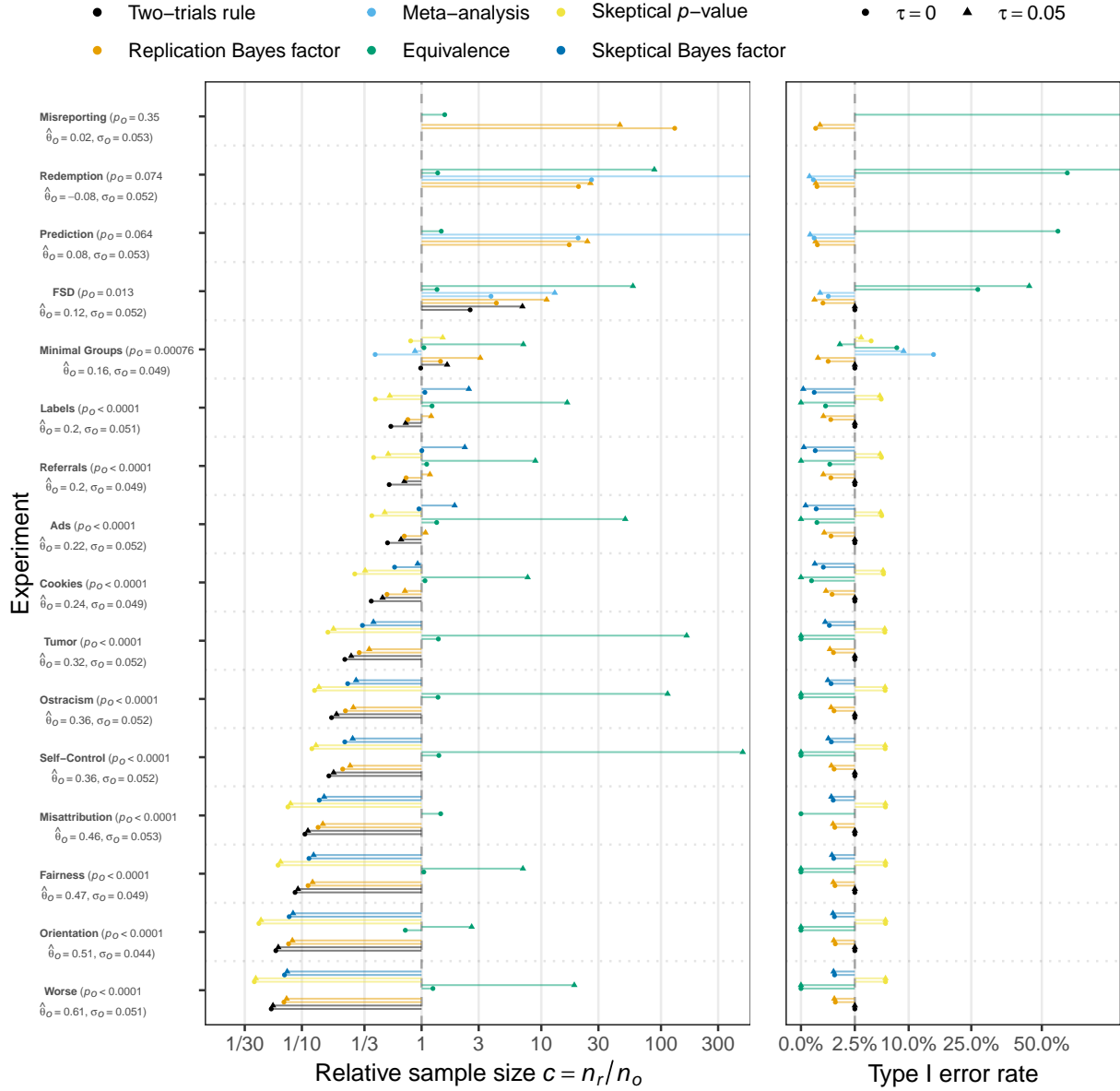
Figure 5

The left plot shows the required relative sample size $c = n_r/n_o$ to achieve a target probability of replication success of $1 - \beta = 80\%$ (if possible). Replication success is defined through the two-trials rule at level $\alpha = 0.025$, replication Bayes factor at level $\gamma = 1/10$, fixed effects-meta analysis at level $\alpha = 0.025^2$, effect size equivalence at level $\alpha = 0.1$ with margin $\Delta = 0.2$, skeptical p-value at level $\alpha = 0.062$, and skeptical Bayes factor at level $\gamma = 1/10$ for data from the replication project by Protzko et al. (2020). A flat initial prior ($\mu_\theta = 0, \sigma_\theta^2 = \infty$) is used for the effect size θ is used either without ($\tau = 0$) or with heterogeneity ($\tau = 0.05$). The right plot shows the type I error rate associated with the required sample size. Experiments are ordered (top to bottom) by their original one-sided p-value $p_o = 1 - \Phi(|\hat{\theta}_o|/\sigma_o)$.

require much fewer samples in the replication study. For the equivalence test, the required

sample size depends instead on the size of the original standard error σ_o, and smaller standard

errors leads to smaller required sample sizes in the replication. We also see that taking into account heterogeneity (triangle) increases the required sample size for all methods compared to not taking it into account (dot). At the same time, a larger required sample size reduces the type I error rate for most methods. We see again the pattern that the type I error rate of the equivalence test and the skeptical p-value is larger than the type I error rate 2.5% of the two-trials rule. However, while the type I error rate of the skeptical p-value decreases when replication studies require larger sample sizes, the type I error rate of the equivalence test may also be large if the replication requires very large sample sizes (e. g., for the experiment "FSD") since it depends on whether the original effect estimate $\hat{\theta}_o$ is sufficiently different from zero. If the original effect estimate $\hat{\theta}_o$ is close to zero, the type I error rate of the equivalence test is drastically increased as equivalence can also be established if the effect estimates from original and replication studies are close to zero (as under the null hypothesis).

Taken together, for most of the experiments all methods but the equivalence test require fewer samples in the replication than in the original study to achieve a target probability of replication success $1 - \beta = 80\%$. This is still the case if heterogeneity is taken into account in the design prior (triangles), which generally increases the required sample size compared to when heterogeneity is not taken into account (dots), especially for studies with large original p-value. Larger replication sample sizes are required for some original studies. In some cases these are unrealistically large (e. g., in the experiment "Prediction" an almost 30 times increase in sample size for the replication Bayes factor), but in other cases they seem more realistic and could be reallocated from the other studies which require fewer samples (e. g., in the experiment "Referrals" an almost three times increase for the skeptical Bayes factor). On the other hand, the equivalence test typically requires larger sample sizes in the replication because the original standard errors are large relative to the specified margin. If one anticipates to analyze the original and replication pair with an equivalence test, this should therefore be taken into account already at the design stage of the original study.

Sample size determination for multisite replication projects

So far we considered the situation where a pair of a single original and a single replication study are analyzed in isolation. However, if multiple replications per single original

study are conducted (so-called *multisite* replication studies) the ensemble of replications can also be analyzed jointly. In this case, some adaptations of the SSD methodology are required.

The replication effect estimate and its standard error are now vectors $\hat{\boldsymbol{\theta}}_r = (\hat{\theta}_{r1}, \ldots, \hat{\theta}_{rm})^\top$ and $\boldsymbol{\sigma}_r^2 = (\sigma_{r1}^2, \ldots, \sigma_{rm}^2)^\top$ consisting of m replication effect estimates, respectively, their standard errors. The normal hierarchical model for the replication estimates $\hat{\boldsymbol{\theta}}_r$ then becomes

$$\hat{\boldsymbol{\theta}}_r \,|\, \boldsymbol{\theta}_r \sim \mathrm{N}_m \left\{ \boldsymbol{\theta}_r, \mathrm{diag}\left(\boldsymbol{\sigma}_r^2\right) \right\} \tag{20a}$$

$$\boldsymbol{\theta}_r \,|\, \theta \;\sim \mathrm{N}_m \left\{ \theta\, \mathbf{1}_m, \tau^2 \mathrm{diag}(\mathbf{1}_m) \right\}, \tag{20b}$$

where $\boldsymbol{\theta}_r$ is a vector of m study specific effect sizes, $\mathbf{1}_m$ is a vector of m ones, and $\mathrm{N}_m(\boldsymbol{\mu}, \boldsymbol{\Sigma})$ denotes the m-variate normal distribution with mean vector $\boldsymbol{\mu}$ and covariance matrix $\boldsymbol{\Sigma}$. By marginalizing over the study specific effect size $\boldsymbol{\theta}_k$, the model can alternatively be expressed by

$$\hat{\boldsymbol{\theta}}_r \,|\, \theta \sim \mathrm{N}_m \left\{ \theta\, \mathbf{1}_m, \mathrm{diag}\left(\boldsymbol{\sigma}_r^2 + \tau^2 \mathbf{1}_m\right) \right\}, \tag{21}$$

so the predictive distribution of $\hat{\boldsymbol{\theta}}_r$ based on the design prior (6) is given by

$$\hat{\boldsymbol{\theta}}_r \,|\, \hat{\theta}_o, \sigma_o^2, \boldsymbol{\sigma}_r^2 \sim \mathrm{N}_m \left\{ \mu_{\hat{\theta}_r} \mathbf{1}_m, \mathrm{diag}\left(\boldsymbol{\sigma}_r^2 + \tau^2 \mathbf{1}_m\right) + \left(\frac{\tau^2 + \sigma_o^2}{1 + 1/g}\right) \mathbf{1}_m \mathbf{1}_m^\top \right\} \tag{22}$$

with $\mu_{\hat{\theta}_r}$ the mean of the predictive distribution of a single replication effect estimate from (7). Importantly, the replication effect estimates are correlated as the covariance matrix in (22) has $(\tau^2 + \sigma_o^2)/(1 + 1/g)$ in the off-diagonal entries.

Often the assessment of replication success can be formulated in terms of a weighted average of the replication effect estimates $\hat{\theta}_{r*} = (\sum_{i=1}^m w_i \hat{\theta}_{ri})/(\sum_{i=1}^m w_i)$. For instance, several multisite replication projects (e. g., Klein et al., 2018) have defined replication success by the fixed or random effects meta-analytic effect estimate of the replication effect estimates achieving statistical significance. Based on the predictive distribution of the replication effect

estimate vector (22), the predictive distribution of the weighted average $\hat{\theta}_{r*}$ is given by

$$\hat{\theta}_{r*} \mid \hat{\theta}_o, \sigma_o^2, \boldsymbol{\sigma}_r^2 \sim \mathrm{N}\left\{\mu_{\hat{\theta}_r}, \sigma_{\hat{\theta}_{r*}}^2 = \left(\sum_{i=1}^m w_i^2 \sigma_{\hat{\theta}_{ri}}^2 + \sum_{i=1}^m \sum_{\substack{j=1 \\ j \neq i}}^m w_i w_j \frac{\tau^2 + \sigma_o^2}{1 + 1/g}\right) \Big/ \left(\sum_{i=1}^m w_i\right)^2\right\} \quad (23)$$

with $\sigma_{\hat{\theta}_{ri}}^2$ the predictive variance of a single replication effect estimate with standard error σ_{ri} as in (7). In particular when the studies receive equal weights ($w_i = w$ for $i = 1, \ldots, m$) and the standard errors of the replication effect estimates are equal ($\sigma_{ri} = \sigma_r$ for $i = 1, \ldots, m$), the predictive variance becomes

$$\sigma_{\hat{\theta}_{r*}}^2 = \frac{\sigma_r^2 + \tau^2}{m} + \frac{\tau^2 + \sigma_o^2}{1 + 1/g}. \quad (24)$$

The probability of replication success can now be obtained by integrating (22), respectively (23), over the corresponding success region S. This may be more involved if the success region is defined in terms of the replication effect estimate vector $\hat{\boldsymbol{\theta}}_r$, whereas it is as simple as in the singlesite replication case if the success region is formulated in terms of the weighted average $\hat{\theta}_{r*}$.

Optimal allocation within and between sites

A key challenge in SSD for multisite replication studies is the optimal allocation of samples within and between sites, that is, how many sites m and how many samples n_{ri} per site i should be used. A similar problem exists in SSD for cluster randomized trials and we can adapt the common solution based on cost functions (Raudenbush, 1997). That is, the optimal configuration is determined so that the probability of replication success is maximized subject to a constrained cost function which accounts for the (typically different) costs of additional samples and sites.

For example, assume a balanced design ($n_{ri} = n_r$ for $i = 1, \ldots, m$) and that the standard errors of the replication effect estimates are inversely proportional to the square-root of the sample size $\sigma_{ri} = \lambda/\sqrt{n_r}$ for some unit variance λ^2. Further assume that maximizing the probability of replication success corresponds to minimizing the variance of the weighted average $\sigma_{\hat{\theta}_{r*}}^2$ in (24). Let K_s denote the cost of an additional site, and K_c the cost of an

additional sample/case. The total cost of the project is then $K = m(K_c\, n_r + K_s)$, and constrained minimization of the predictive variance (24) leads to the optimal sample size per site

$$n_r^* = \frac{\lambda}{\tau} \sqrt{\frac{K_s}{K_c}}$$

which is equivalent to the optimal cluster sample size known from cluster randomized trials (Raudenbush & Liu, 2000). Note that the optimal sample size per site may be different for other analysis approaches where maximizing the probability of replication success does not correspond to minimizing the variance of the weighted average. Moreover, there are also practical considerations which affect the choice of how many sites should be included in a project. For instance, there may simply not be enough labs available with the required expertise to perform the replication experiments.

Example: Cross-laboratory replication project (continued)

Figure 6 illustrates multisite SSD for the experiment "Labels" from Protzko et al. (2020) for planned analyses based on the two-trials rule and the replication Bayes factor (see the supplement for details on the multisite extension of these two methods). As for singlesite SSD, we use the design prior based on an initial flat prior for the effect size and taking into account heterogeneity ($\tau = 0.05$). The top plots show the probability of replication success as a function of the total sample size $m \times n_r$ for different number of sites m. We see that for the same total sample size a larger number of sites increases the probability of replication success. For instance, a total sample size of roughly 3000 is required to achieve an 80% target probability with one site for the two-trials rule, whereas only approximately half as many samples are required for two sites.

However, focusing only on the total sample size ignores the fact that the cost of an additional site is usually larger than the cost of an additional observation. The bottom plot shows the total cost K of a design (relative to the cost of one sample K_c) whose sample size is determined for a target probability of replication success $1 - \beta = 80\%$. We see that if the cost of an additional site K_s is not much larger than the cost of an additional sample K_c, e. g.,
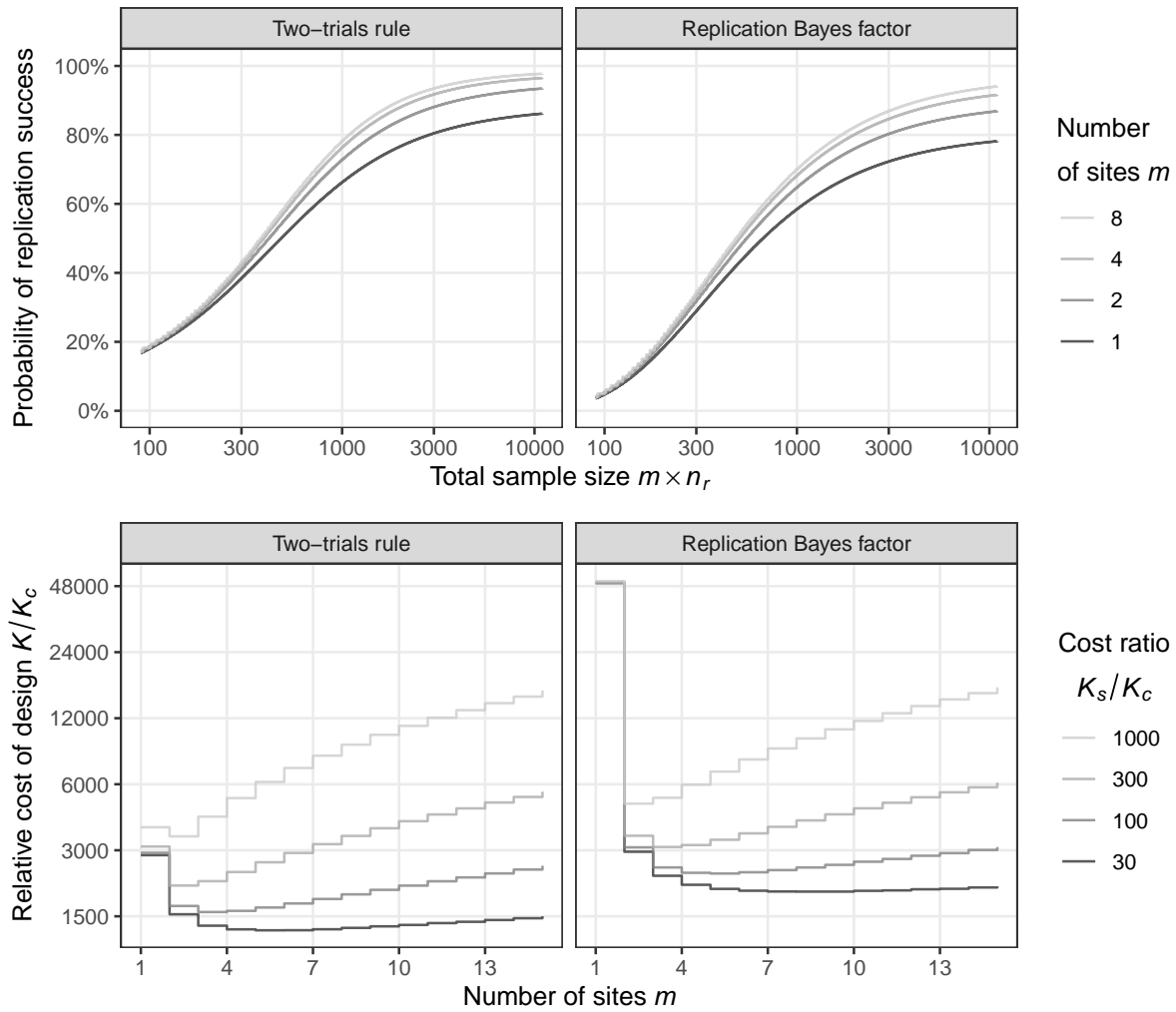
Figure 6

Top plots show the probability of replication success based on the replication Bayes factor at level $\gamma = 1/10$ (left) and the two-trials rule at level $\alpha = 0.025$ (right) as a function of the total sample size and for different number of sites m for data from the experiment "Labels". A design prior with heterogeneity $\tau = 0.05$ and flat initial prior for the effect size θ is used. The same heterogeneity value is assumed in the analysis of the replications. design (relative to the cost of a single sample K_c) as a function of the number of sites m and for different site costs K_s. The sample size of each design corresponds to a target probability of replication success $1 - \beta = 80\%$.

$K_S/K_c = 30$ the optimal number of sites is $m = 5$ for the two-trials rule and $m = 8$ for the replication Bayes factor. If an additional site is more costly the optimal number of sites is lower, e. g., if the cost ratio is $K_S/K_c = 300$, the optimal number of sites is $m = 2$ for the two trials rule and $m = 3$ for the replication Bayes factor, while the number of sites in the actually used by Protzko et al. (2020) was $m = 3$ (counting only external-replications), respectively, $m = 4$ (counting also the internal-replication).

Discussion

We showed how Bayesian approaches can be used to determine the sample size of replication studies based on all the available information and the associated uncertainty. A key strength of the approach is that it can be applied to any kind of replication analysis method, Bayes or non-Bayes, as long as there is a well-defined success region for the replication effect estimate. Methods for assessing replication success which have not yet been adapted to Bayesian design approaches in the normal-normal hierarchical model (or which have not even been proposed) can thus benefit from our methodology. For instance, our methods could be straightforwardly applied to the "dual-criterion" from Rosenkranz (2021) which defines replication success via simultaneous statistical significance and practical relevance of the effect estimates from original and replication study.

There are some limitations and possible extensions: we have developed the methodology for "direct" replication studies (Simons, 2014) which attempt to replicate the conditions from the original study as closely as possible; yet SSD methodology is also needed for "conceptual" replication and for "generalization" studies which may show systematic deviations from the original study. While the heterogeneity variance in the design prior allows to take effect size heterogeneity into account for SSD, to some extent, further research is needed for investigating how systematic study deviations and external knowledge can be incorporated. Furthermore, as is standard in meta-analysis we assumed that the variances of the effect estimates are known, which can sometimes be inadequate (Jackson & White, 2018). Specifying priors also for the variances could better reflect the available uncertainty but would come at the price of lower interpretability and higher computational complexity. We did also not consider designs where the replication data are analyzed in a sequential manner. Ideas from the Bayesian sequential design (Schönbrodt & Wagenmakers, 2018; Stefan, Gronau, & Wagenmakers, 2022) or from the adaptive trials literature (Bretz, Koenig, Brannath, Glimm, & Posch, 2009) could be adapted to the replication setting as in Micheloud and Held (2022). A sequential analysis of the replication data could possibly increase the efficiency of the replication. An additional point is that we assumed that the original study has been completed when planning the replication study. One could also consider a scenario where both the

original and replication study are planned simultaneously and adopt a "project" perspective (Held, Micheloud, & Pawel, 2022; Maca, Gallo, Branson, & Maurer, 2002). However, in this case no information from the original study is available and the design prior needs to be specified entirely based on external knowledge. Finally, researchers have only limited resources and it may happen that they cannot afford a large enough sample size to obtain their desired probability of replication success. In this situation a reverse-Bayes approach (Held, Matthews, Ott, & Pawel, 2022) could be applied in order to determine the prior for the effect size which is required to achieve the desired probability of replication success based on the maximally possible sample size. Researchers can then judge whether or not such prior beliefs are scientifically sensible, and decide whether they should conduct the replication study with their limited resources.

Appendix: The BayesRepDesign R package

```r
library("BayesRepDesign")


## design prior (flat initial prior for effect size + heterogeneity)
dp <- designPrior(to = 0.2, so = 0.05, tau = 0.08)
plot(dp)
```
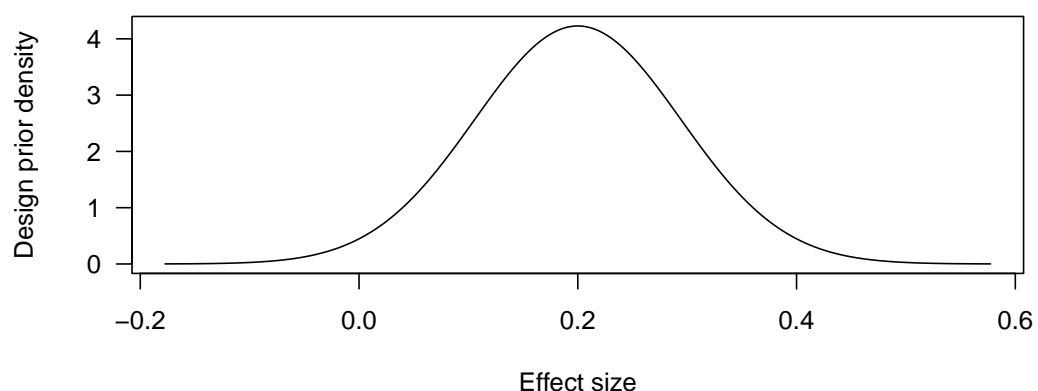


```r
## compute replication standard error for achieving significance at 2.5%
ssdSig(level = 0.025, dprior = dp, power = 0.8)

##        Bayesian sample size calculation for replication studies
```

```
##          ========================================================
##
## success criterion and computation
## ----------------------------------------------------------------
##    replication p-value <= 0.025 (exact computation)
##
## original data and initial prior for effect size
## ----------------------------------------------------------------
##    to = 0.2 : original effect estimate
##    so = 0.05 : standard error of original effect estimate
##    tau = 0.08 : assumed heterogeneity standard deviation
##    N(mean = 0, sd = Inf) : initial normal prior
##
## design prior for effect size
## ----------------------------------------------------------------
##    N(mean = 0.2, sd = 0.094) : normal design prior
##
## probability of replication success
## ----------------------------------------------------------------
##    PoRS = 0.8 : specified
##    PoRS = 0.8 : recomputed with sr
##
## required sample size
## ----------------------------------------------------------------
##    sr = 0.045 : required standard error of replication effect estimate
##    c = so^2/sr^2 ~= nr/no = 1.2 : required relative variance / sample size

## compute numerically via success region and method agnostic function
sregFun <- function(sr) {
    ## success region is [1.96*sr, Inf)
    successRegion(intervals = cbind(qnorm(p = 0.975)*sr, Inf))
}
ssd(sregionfun = sregFun, dprior = dp, power = 0.8)

##          Bayesian sample size calculation for replication studies
##          ========================================================
```

```
##
## success criterion and computation
## ------------------------------------------------------------------------
##   method agnostic success region (numerical computation)
##
## original data and initial prior for effect size
## ------------------------------------------------------------------------
##   to = 0.2 : original effect estimate
##   so = 0.05 : standard error of original effect estimate
##   tau = 0.08 : assumed heterogeneity standard deviation
##   N(mean = 0, sd = Inf) : initial normal prior
##
## design prior for effect size
## ------------------------------------------------------------------------
##   N(mean = 0.2, sd = 0.094) : normal design prior
##
## probability of replication success
## ------------------------------------------------------------------------
##   PoRS = 0.8 : specified
##   PoRS = 0.8 : recomputed with sr
##
## required sample size
## ------------------------------------------------------------------------
##   sr = 0.045 : required standard error of replication effect estimate
##   c = so^2/sr^2 ~= nr/no = 1.2 : required relative variance / sample size
```

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Computational details

```
sessionInfo()

## R version 4.2.1 (2022-06-23)
## Platform: x86_64-pc-linux-gnu (64-bit)
## Running under: Ubuntu 20.04.5 LTS
##
## Matrix products: default
## BLAS:   /usr/lib/x86_64-linux-gnu/blas/libblas.so.3.9.0
## LAPACK: /usr/lib/x86_64-linux-gnu/lapack/liblapack.so.3.9.0
##
## locale:
##  [1] LC_CTYPE=en_US.UTF-8       LC_NUMERIC=C
##  [3] LC_TIME=de_CH.UTF-8        LC_COLLATE=en_US.UTF-8
##  [5] LC_MONETARY=de_CH.UTF-8    LC_MESSAGES=en_US.UTF-8
##  [7] LC_PAPER=de_CH.UTF-8       LC_NAME=C
##  [9] LC_ADDRESS=C               LC_TELEPHONE=C
## [11] LC_MEASUREMENT=de_CH.UTF-8 LC_IDENTIFICATION=C
##
## attached base packages:
## [1] stats     graphics  grDevices utils     datasets  methods   base
##
## other attached packages:
##  [1] BayesRepDesign_0.42   BayesRep_0.1          ggpubr_0.4.0
##  [4] colorspace_2.0-3      scales_1.2.1          ggplot2_3.3.6
##  [7] tidyr_1.2.0           dplyr_1.0.9           ReplicationSuccess_1.2
## [10] knitr_1.39
##
## loaded via a namespace (and not attached):
##  [1] Rcpp_1.0.9        highr_0.9         pillar_1.8.1      compiler_4.2.1
##  [5] tools_4.2.1       digest_0.6.29     evaluate_0.15     lifecycle_1.0.3
##  [9] tibble_3.1.8      gtable_0.3.1      pkgconfig_2.0.3   rlang_1.0.6
## [13] DBI_1.1.2         cli_3.4.1         xfun_0.31         gridExtra_2.3
## [17] withr_2.5.0       stringr_1.4.0     generics_0.1.2    vctrs_0.4.2
## [21] cowplot_1.1.1     grid_4.2.1        tidyselect_1.1.2  glue_1.6.2
```

```
## [25] R6_2.5.1          lamW_2.1.1        rstatix_0.7.0     fansi_1.0.3
## [29] carData_3.0-5     farver_2.1.1      purrr_0.3.4       car_3.0-13
## [33] magrittr_2.0.3    ellipsis_0.3.2    backports_1.4.1   assertthat_0.2.1
## [37] abind_1.4-5       ggsignif_0.6.3    labeling_0.4.2    utf8_1.2.2
## [41] stringi_1.7.6     RcppParallel_5.1.5 munsell_0.5.0    broom_0.8.0
```