

Replication of “null results” – Absence of evidence or evidence of absence?

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Abstract In several large-scale replication projects, statistically non-significant results in both the original and the replication study have been interpreted as a “replication success”. Here we discuss the logical problems with this approach. Non-significance in both studies does not ensure that the studies provide evidence for the absence of an effect and “replication success” can virtually always be achieved if the sample sizes of the studies are small enough. In addition, the relevant error rates are not controlled. We show how methods, such as equivalence testing and Bayes factors, can be used to adequately quantify the evidence for the absence of an effect and how they can be applied in the replication setting. Using data from the Reproducibility Project: Cancer Biology we illustrate that many original and replication studies with “null results” are in fact inconclusive. We conclude that it is important to also replicate studies with statistically non-significant results, but that they should be designed, analyzed, and interpreted appropriately.

Introduction

Absence of evidence is not evidence of absence – the title of the 1995 paper by Douglas Altman and Martin Bland has since become a mantra in the statistical and medical literature (*Altman and Bland, 1995*). Yet, the misconception that a statistically non-significant result indicates evidence for the absence of an effect is unfortunately still widespread (*Makin and de Xivry, 2019*). Such a “null result” – typically characterized by a p -value of $p > 0.05$ for the null hypothesis of an absent effect – may also occur if an effect is actually present. For example, if the sample size of a study is chosen to detect an assumed effect with a power of 80%, null results will incorrectly occur 20% of the time when the assumed effect is actually present. Conversely, if the power of the study is lower, null results will occur more often. In general, the lower the power of a study, the greater the ambiguity of a null result. To put a null result in context, it is therefore critical to know whether the study was adequately powered and under what assumed effect the power was calculated (*Hoenig and Heisey, 2001; Greenland, 2012*). However, if the goal of a study is to explicitly quantify the evidence for the absence of an effect, more appropriate methods designed for this task, such as equivalence testing (*Wellek, 2010*) or Bayes factors (*Kass and Raftery, 1995*), should be used from the outset.

The contextualization of null results becomes even more complicated in the setting of replication studies. In a replication study, researchers attempt to repeat an original study as closely as possible in order to assess whether similar results can be obtained with new data (*National Academies of Sciences, Engineering, and Medicine, 2019*). There have been various large-scale replication projects in the biomedical and social sciences in the last decade (*Prinz et al., 2011; Begley and Ellis, 2012; Klein et al., 2014; Open Science Collaboration, 2015; Camerer et al., 2016, 2018;*

Klein et al., 2018; Cova et al., 2018; Errington et al., 2021, among others). Most of these projects reported alarmingly low replicability rates across a broad spectrum of criteria for quantifying replicability. While most of these projects restricted their focus on original studies with statistically significant results (“positive results”), the *Reproducibility Project: Psychology* (RPP, *Open Science Collaboration, 2015*), the *Reproducibility Project: Experimental Philosophy* (RPEP, *Cova et al., 2018*), and the *Reproducibility Project: Cancer Biology* (RPCB, *Errington et al., 2021*) also attempted to replicate some original studies with null results.

The RPP excluded the original null results from its overall assessment of replication success, but the RPCB and the RPEP explicitly defined null results in both the original and the replication study as a criterion for “replication success”. There are several logical problems with this “non-significance” criterion. First, if the original study had low statistical power, a non-significant result is highly inconclusive and does not provide evidence for the absence of an effect. It is then unclear what exactly the goal of the replication should be – to replicate the inconclusiveness of the original result? On the other hand, if the original study was adequately powered, a non-significant result may indeed provide some evidence for the absence of an effect when analyzed with appropriate methods, so that the goal of the replication is clearer. However, the criterion does not distinguish between these two cases. Second, with this criterion researchers can virtually always achieve replication success by conducting two studies with very small sample sizes, such that the p -values are non-significant and the results are inconclusive. This is because the null hypothesis under which the p -values are computed is misaligned with the goal of inference, which is to quantify the evidence for the absence of an effect. We will discuss methods that are better aligned with this inferential goal. Third, the criterion does not control the error of falsely claiming the absence of an effect at some predetermined rate. This is in contrast to the standard replication success criterion of requiring significance from both studies (also known as the two-trials rule, see chapter 12.2.8 in *Senn, 2008*), which ensures that the error of falsely claiming the presence of an effect is controlled at a rate equal to the squared significance level (for example, $5 \times 5\% = 0.25\%$ for a 5% significance level). The non-significance criterion may be intended to complement the two-trials rule for null results, but it fails to do so in this respect, which may be important to regulators, funders, and researchers. We will now demonstrate these issues and potential solutions using the null results from the RPCB.

Null results from the Reproducibility Project: Cancer Biology

Figure 1 shows standardized mean difference effect estimates with 95% confidence intervals from two RPCB study pairs. Both are “null results” and meet the non-significance criterion for replication success (the two-sided p -values are greater than 0.05 in both the original and the replication study), but intuition would suggest that these two pairs are very much different.

The original study from *Dawson et al. (2011)* and its replication both show large effect estimates in magnitude, but due to the small sample sizes, the uncertainty of these estimates is very large, too. If the sample sizes of the studies were larger and the point estimates remained the same, intuitively both studies would provide evidence for a non-zero effect. However, with the sample sizes that were actually used, the results seem inconclusive. In contrast, the effect estimates from *Goetz et al. (2011)* and its replication are much smaller in magnitude and their uncertainty is also smaller because the studies used larger sample sizes. Intuitively, these studies seem to provide some evidence for a zero (or negligibly small) effect. While these two examples show the qualitative difference between absence of evidence and evidence of absence, we will now discuss how the two can be quantitatively distinguished.

Methods for assessing replicability of null results

There are both frequentist and Bayesian methods that can be used for assessing evidence for the absence of an effect. *Anderson and Maxwell (2016)* provide an excellent summary of both ap-

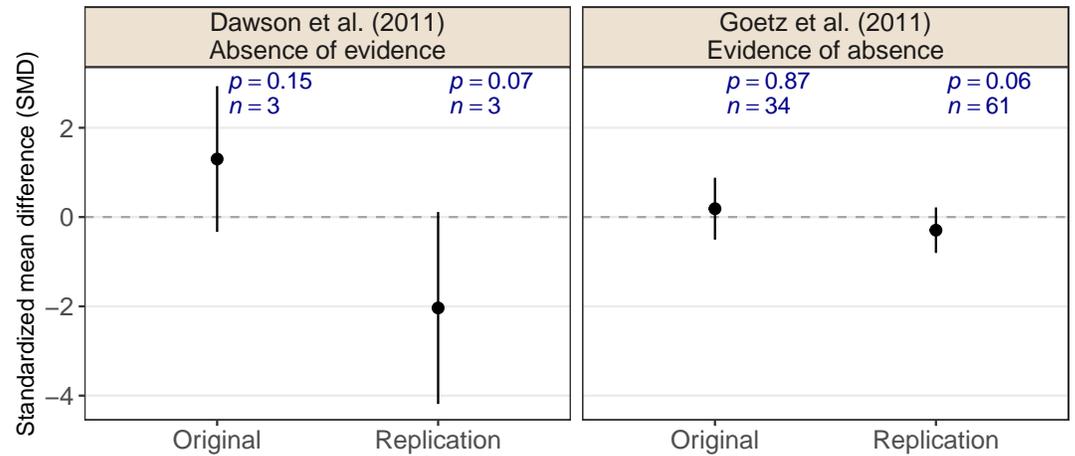


Figure 1. Two examples of original and replication study pairs which meet the non-significance replication success criterion from the Reproducibility Project: Cancer Biology (Errington et al., 2021). Shown are standardized mean difference effect estimates with 95% confidence intervals, sample sizes, and two-sided p -values for the null hypothesis that the effect is absent.

proaches in the context of replication studies in psychology. We now briefly discuss two possible approaches – frequentist equivalence testing and Bayesian hypothesis testing – and their application to the RPCB data.

Equivalence testing

Equivalence testing was developed in the context of clinical trials to assess whether a new treatment – typically cheaper or with fewer side effects than the established treatment – is practically equivalent to the established treatment (Wellek, 2010). The method can also be used to assess whether an effect is practically equivalent to the value of an absent effect, usually zero. Using equivalence testing as a remedy for non-significant results has been suggested by several authors (Hauck and Anderson, 1986; Campbell and Gustafson, 2018). The main challenge is to specify the margin $\Delta > 0$ that defines an equivalence range $[-\Delta, +\Delta]$ in which an effect is considered as absent for practical purposes. The goal is then to reject the null hypothesis that the true effect is outside the equivalence range. This is in contrast to the usual null hypothesis of a superiority test which states that the effect is zero, see Figure 2 for an illustration.

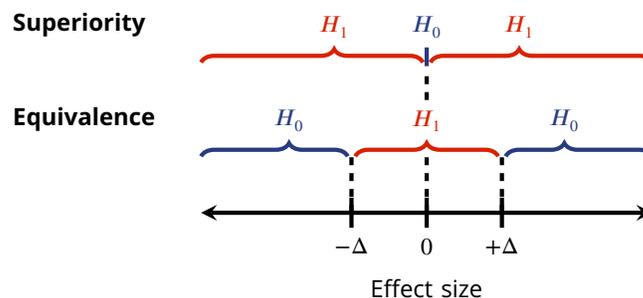


Figure 2. Null hypothesis (H_0) and alternative hypothesis (H_1) for superiority and equivalence tests (with equivalence margin $\Delta > 0$).

To ensure that the null hypothesis is falsely rejected at most $\alpha \times 100\%$ of the time, the standard approach is to declare equivalence if the $(1-2\alpha) \times 100\%$ confidence interval for the effect is contained

within the equivalence range, for example, a 90% confidence interval for $\alpha = 5\%$ (Westlake, 1972). The procedure is equivalent to two one-sided tests (TOST) for the null hypotheses of the effect being greater/smaller than $+\Delta$ and $-\Delta$ being significant at level α (Schuirmann, 1987). A quantitative measure of evidence for the absence of an effect is then given by the maximum of the two one-sided p -values (the TOST p -value). A reasonable replication success criterion for null results may therefore be to require that both the original and the replication TOST p -values be smaller than some level α (e.g., 0.05), or, equivalently, that their $(1-2\alpha)\times 100\%$ confidence intervals are included in the equivalence region. In contrast to the non-significance criterion, this criterion controls the error of falsely claiming replication success at level α^2 when there is a true effect outside the equivalence margin, thus complementing the usual two-trials rule.

Returning to the RPCB data, Figure 3 shows the standardized mean difference effect estimates with 90% confidence intervals for the 15 effects which were treated as quantitative null results by the RPCB.¹ Most of them showed non-significant p -values ($p > 0.05$) in the original study, but there are two effects in paper 48 which the original authors regarded as null results despite their statistical significance. We see that there are 11 “success” according to the non-significance criterion (with $p > 0.05$ in original and replication study) out of total 15 null effects, as reported in Table 1 from Errington et al. (2021).

We will now apply equivalence testing to the RPCB data. The dotted red lines represent an equivalence range for the margin $\Delta = 0.74$, which Wellek (2010, Table 1.1) classifies as “liberal”. However, even with this generous margin, only 4 of the 15 study pairs are able to establish replication success at the 5% level, in the sense that both the original and the replication 90% confidence interval fall within the equivalence range (or, equivalently, that their TOST p -values are smaller than 0.05). For the remaining 11 studies, the situation remains inconclusive and there is no evidence for the absence or the presence of the effect. For instance, the previously discussed example from Goetz et al. (2011) marginally fails the criterion ($p_{\text{TOST}} = 0.06$ in the original study and $p_{\text{TOST}} = 0.04$ in the replication), while the example from Dawson et al. (2011) is a clearer failure ($p_{\text{TOST}} = 0.75$ in the original study and $p_{\text{TOST}} = 0.88$ in the replication).

The post-hoc determination of the equivalence margins is controversial. Ideally, the margin should be determined on a case-by-case basis before the studies are conducted by researchers familiar with the subject matter. In the social and medical sciences, the conventions of Cohen (1992) are typically used to classify SMD effect sizes (SMD = 0.2 small, SMD = 0.5 medium, SMD = 0.8 large). While effect sizes are typically larger in preclinical research, it seems unrealistic to specify margins larger than 1 to represent effect sizes that are absent for practical purposes. It could also be argued that the chosen margin $\Delta = 0.74$ is too lax compared to margins commonly used in clinical research; for instance, in oncology, a margin of $\Delta = \log(1.3)$ is commonly used for log odds/hazard ratios, whereas in bioequivalence studies a margin of $\Delta = \log(1.25)$ is the convention. These margins would translate into much more stringent margins of $\Delta = 0.14$ and $\Delta = 0.12$ on the SMD scale, respectively, using the $\text{SMD} = (\sqrt{3/\pi}) \log \text{OR}$ conversion (Cooper et al., 2019, p. 233). Therefore, we report a sensitivity analysis in Figure 4. The top plot shows the number of successful replications as a function of the margin Δ and for different TOST p -value thresholds. Such an “equivalence curve” approach was first proposed by Hauck and Anderson (1986). We see that for realistic margins between 0 and 1, the proportion of replication successes remains below 50%. To achieve a success rate of $11/15 = 73.3\%$, as with the non-significance criterion, unrealistic margins of $\Delta > 2$ are required, highlighting the paucity of evidence provided by these studies.

¹There are four original studies with null effects for which two or three “internal” replication studies were conducted, leading in total to 20 replications of null effects. As in the RPCB main analysis (Errington et al., 2021), we aggregated their SMD estimates into a single SMD estimate with fixed-effect meta-analysis and recomputed the replication p -value based on a normal approximation. For the original studies and single replication studies we report the p -values as provided by the RPCB.

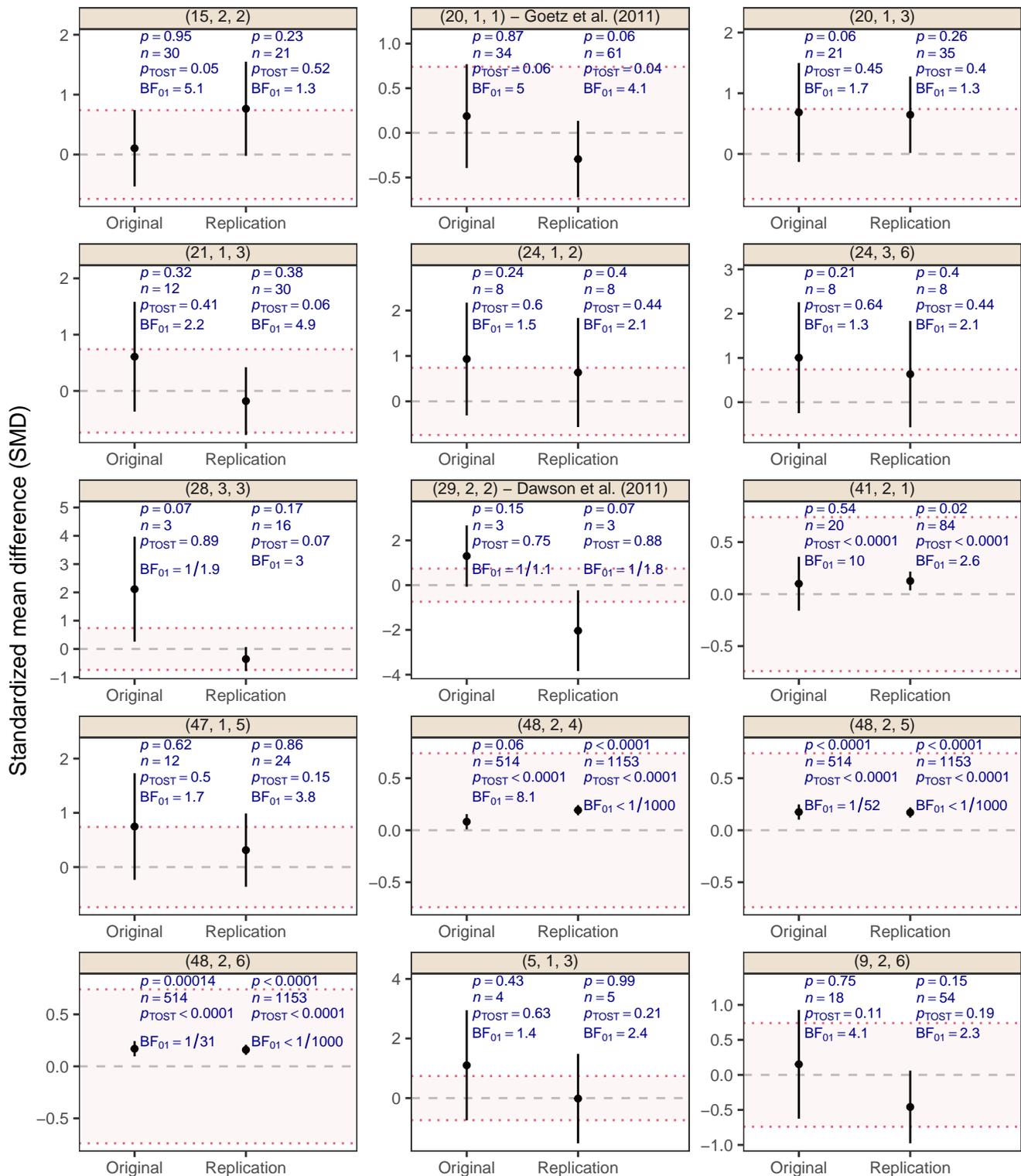


Figure 3. Standardized mean difference (SMD) effect estimates with 90% confidence interval for the “null results” and their replication studies from the Reproducibility Project: Cancer Biology (Errington et al., 2021). The identifier above each plot indicates (original paper number, experiment number, effect number). Two original effect estimates from original paper 48 were statistically significant at $p < 0.05$, but were interpreted as null results by the original authors and therefore treated as null results by the RPCB. The two examples from Figure 1 are indicated in the plot titles. The dashed gray line represents the value of no effect (SMD = 0), while the dotted red lines represent the equivalence range with a margin of $\Delta = 0.74$, classified as “liberal” by Wellek (2010, Table 1.1). The p -values p_{TOST} are the maximum of the two one-sided p -values for the effect being less than or greater than $+\Delta$ or $-\Delta$, respectively. The Bayes factors BF_{01} quantify the evidence for the null hypothesis $H_0 : SMD = 0$ against the alternative $H_1 : SMD \neq 0$ with normal unit-information prior assigned to the SMD under H_1 .

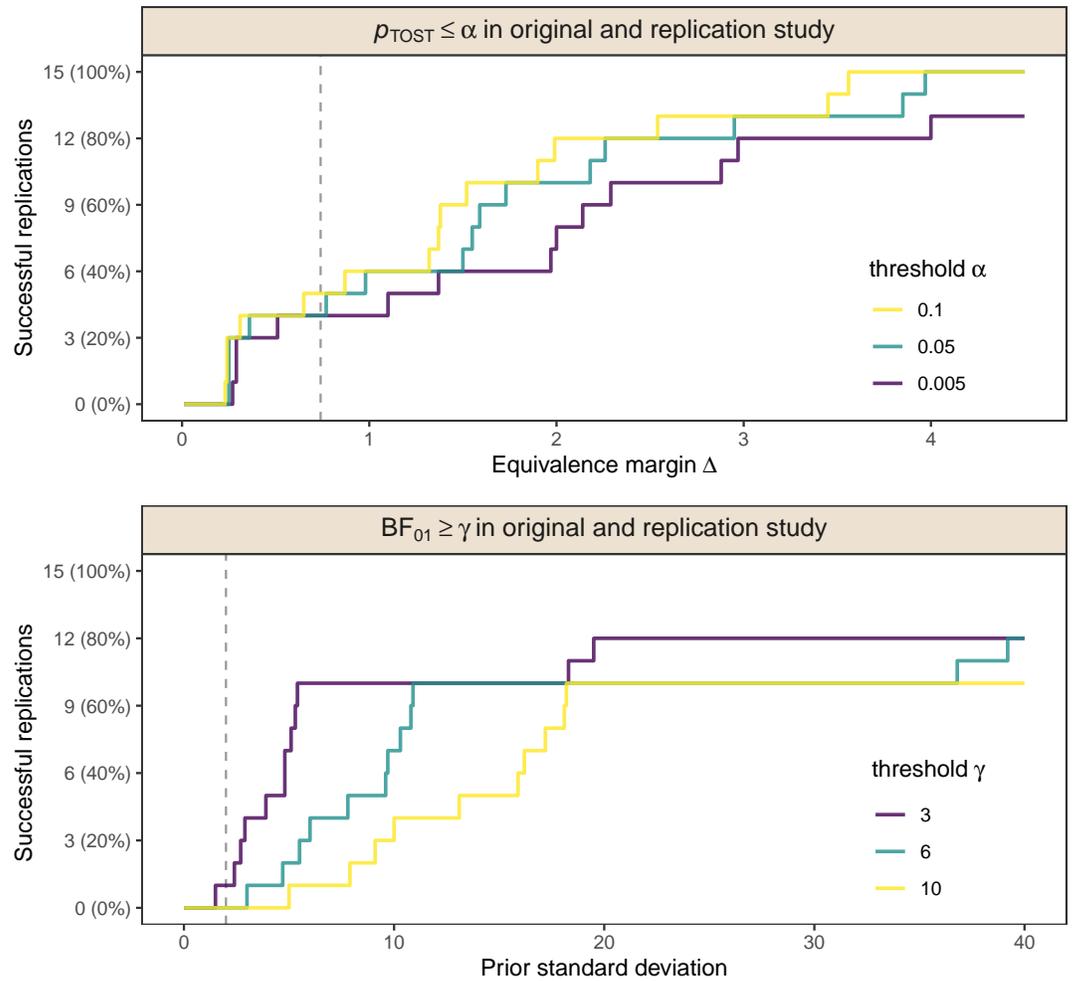


Figure 4. Number of successful replications of original null results in the RPCB as a function of the margin Δ of the equivalence test ($p_{TOST} \leq \alpha$ in both studies) or the standard deviation of the normal prior distribution for the SMD effect size under the alternative H_1 of the Bayes factor test ($BF_{01} \geq \gamma$ in both studies). The dashed gray lines represent the margin and standard deviation used in the main analysis shown in Figure 3.

Bayesian hypothesis testing

The distinction between absence of evidence and evidence of absence is naturally built into the Bayesian approach to hypothesis testing. A central measure of evidence is the Bayes factor (*Kass and Raftery, 1995*), which is the updating factor of the prior odds to the posterior odds of the null hypothesis H_0 versus the alternative hypothesis H_1

$$\underbrace{\frac{\Pr(H_0 | \text{data})}{\Pr(H_1 | \text{data})}}_{\text{Posterior odds}} = \underbrace{\frac{\Pr(H_0)}{\Pr(H_1)}}_{\text{Prior odds}} \times \underbrace{\frac{p(\text{data} | H_0)}{p(\text{data} | H_1)}}_{\text{Bayes factor } BF_{01}}.$$

The Bayes factor quantifies how much the observed data have increased or decreased the probability of the null hypothesis H_0 relative to the alternative H_1 . If the null hypothesis states the absence of an effect, a Bayes factor greater than one ($BF_{01} > 1$) indicates evidence for the absence of the effect and a Bayes factor smaller than one indicates evidence for the presence of the effect ($BF_{01} < 1$), whereas a Bayes factor not much different from one indicates absence of evidence for either hypothesis ($BF_{01} \approx 1$). A reasonable criterion for successful replication of a null result may hence be to require a Bayes factor larger than some level $\gamma > 1$ from both studies, for example, $\gamma = 3$ or

$\gamma = 10$ which are conventional levels for “substantial” and “strong” evidence, respectively (Jeffreys, 1961). In contrast to the non-significance criterion, this criterion provides a genuine measure of evidence that can distinguish absence of evidence from evidence of absence.

When the observed data are dichotomized into positive ($p < 0.05$) or null results ($p > 0.05$), the Bayes factor based on a null result is the probability of observing $p > 0.05$ when the effect is indeed absent (which is 95%) divided by the probability of observing $p > 0.05$ when the effect is indeed present (which is one minus the power of the study). For example, if the power is 90%, we have $BF_{01} = 95\%/10\% = 9.5$ indicating almost ten times more evidence for the absence of the effect than for its presence. On the other hand, if the power is only 50%, we have $BF_{01} = 95\%/50\% = 1.9$ indicating only slightly more evidence for the absence of the effect. This example also highlights the main challenge with Bayes factors – the specification of the alternative hypothesis H_1 . The assumed effect under H_1 is directly related to the power of the study, and researchers who assume different effects under H_1 will end up with different Bayes factors. Instead of specifying a single effect, one therefore typically specifies a “prior distribution” of plausible effects. Importantly, the prior distribution, like the equivalence margin, should be determined by researchers with subject knowledge and before the data are observed.

In practice, the observed data should not be dichotomized into positive or null results, as this leads to a loss of information. Therefore, to compute the Bayes factors for the RPCB null results, we used the observed effect estimates as the data and assumed a normal sampling distribution for them, as in a meta-analysis. The Bayes factors BF_{01} shown in Figure 3 then quantify the evidence for the null hypothesis of no effect ($H_0 : SMD = 0$) against the alternative hypothesis that there is an effect ($H_1 : SMD \neq 0$) using a normal “unit-information” prior distribution² (Kass and Wasserman, 1995) for the effect size under the alternative H_1 . We see that in most cases there is no substantial evidence for either the absence or the presence of an effect, as with the equivalence tests. For instance, with a lenient Bayes factor threshold of 3, only 1 of the 15 replications are successful, in the sense of having $BF_{01} > 3$ in both the original and the replication study. The Bayes factors for the two previously discussed examples are consistent with our intuitions – in the Goetz et al. (2011) example there is indeed substantial evidence for the absence of an effect ($BF_{01} = 5$ in the original study and $BF_{01} = 4.1$ in the replication), while in the Dawson et al. (2011) example there is even weak evidence for the presence of an effect, though the Bayes factors are very close to one due to the small sample sizes ($BF_{01} = 1/1.1$ in the original study and $BF_{01} = 1/1.8$ in the replication).

As with the equivalence margin, the choice of the prior distribution for the SMD under the alternative H_1 is debatable. The normal unit-information prior seems to be a reasonable default choice, as it implies that small to large effects are plausible under the alternative, but other normal priors with smaller/larger standard deviations could have been considered to make the test more sensitive to smaller/larger true effect sizes. We therefore report a sensitivity analysis with respect to the choice of the prior standard deviation in the bottom plot of Figure 4. It is uncommon to specify prior standard deviations larger than the unit-information standard deviation of 2, as this corresponds to the assumption of very large effect sizes under the alternatives. However, to achieve replication success for a larger proportion of replications than the observed $1/15 = 6.7\%$, unreasonably large prior standard deviations have to be specified. For instance, a standard deviation of roughly 5 is required to achieve replication success in 50% of the replications, and the standard deviation needs to be almost 20 so that the same success rate $11/15 = 73.3\%$ as with the non-significance criterion is achieved.

Of note, among the 15 RPCB null results, there are three interesting cases (the three effects from paper 48) where the Bayes factor is qualitatively different from the equivalence test, revealing a

²For SMD effect sizes, a normal unit-information prior is a normal distribution centered around the null value with a standard deviation corresponding to one observation. Assuming that the group means are normally distributed $\bar{X}_1 \sim N(\theta_1, 2\sigma^2/n)$ and $\bar{X}_2 \sim N(\theta_2, 2\sigma^2/n)$ with n the total sample size and σ the known data standard deviation, the distribution of the SMD is $SMD = (\bar{X}_1 - \bar{X}_2)/\sigma \sim N((\theta_1 - \theta_2)/\sigma, 4/n)$. The standard deviation of the SMD based on one unit ($n = 1$) is hence 2, just as the unit standard deviation for log hazard/odds/rate ratio effect sizes (Spiegelhalter et al., 2004, Section 2.4).

fundamental difference between the two approaches. The Bayes factor is concerned with testing whether the effect is *exactly zero*, whereas the equivalence test is concerned with whether the effect is within an *interval around zero*. Due to the very large sample size in the original study ($n = 514$) and the replication ($n = 1153$), the data are incompatible with an exactly zero effect, but compatible with effects within the equivalence range. Apart from this example, however, both approaches lead to the same qualitative conclusion – most RPCB null results are highly ambiguous.

Conclusions

We showed that in most of the RPCB studies with “null results”, neither the original nor the replication study provided conclusive evidence for the presence or absence of an effect. It seems logically questionable to declare an inconclusive replication of an inconclusive original study as a replication success. While it is important to replicate original studies with null results, our analysis highlights that they should be analyzed and interpreted appropriately.

For both the equivalence testing and the Bayes factor approach, it is critical that the parameters of the procedure (the equivalence margin and the prior distribution) are specified independently of the data, ideally before the studies are conducted. Typically, however, the original studies were designed to find evidence for the presence of an effect, and the goal of replicating the “null result” was formulated only after failure to do so. It is therefore important that margins and prior distributions are motivated from historical data and/or field conventions (*Campbell and Gustafson, 2021*), and that sensitivity analyses regarding their choice are reported.

While the equivalence test and the Bayes factor are two principled methods for analyzing original and replication studies with null results, they are not the only possible methods for doing so. For instance, the reverse-Bayes approach from *Micheloud and Held (2022b)* specifically tailored to equivalence testing in the replication setting may lead to more appropriate inferences as it also takes into account the compatibility of the effect estimates from original and replication studies. In addition, there are various other Bayesian methods which could potentially improve upon the considered Bayes factor approach. For example, Bayes factors based on non-local priors (*Johnson and Rossell, 2010*) or based on interval null hypotheses (*Morey and Rouder, 2011; Liao et al., 2020*), methods for equivalence testing based on effect size posterior distributions (*Kruschke, 2018*), or Bayesian procedures that involve utilities of decisions (*Lindley, 1998*). Finally, the design of replication studies should align with the planned analysis (*Anderson and Maxwell, 2017; Anderson and Kelley, 2022; Micheloud and Held, 2022a; Pawel et al., 2022*). If the goal of the study is to find evidence for the absence of an effect, the replication sample size should also be determined so that the study has adequate power to make conclusive inferences regarding the absence of the effect.

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Conflict of interest

We declare no conflict of interest.

Software and data

The code and data to reproduce our analyses is openly available at <https://gitlab.uzh.ch/samuel.pawel/rsAbsence>. A snapshot of the repository at the time of writing is available at <https://doi.org/10.5281/zenodo.XXXXXX>. We used the statistical programming language R version 4.2.3 (*R Core Team, 2022*) for analyses. The R packages *ggplot2* (*Wickham, 2016*), *dplyr* (*Wickham et al., 2022*), *knitr* (*Xie, 2022*), and *reporttools* (*Rufibach, 2009*) were used for plotting, data preparation, dynamic reporting, and formatting, respectively. The data from the RPCB were obtained by downloading

the files from <https://github.com/mayamathur/rpcb> (commit a1e0c63) and extracting the relevant variables as indicated in the R script `preprocess-rpcb-data.R` which is available in our git repository.

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Computational details

```
cat(paste(Sys.time(), Sys.timezone(), "\n"))

## 2023-04-04 14:40:26 Etc/UTC

sessionInfo()

## R version 4.2.3 (2023-03-15)
## Platform: x86_64-pc-linux-gnu (64-bit)
## Running under: Ubuntu 22.04.2 LTS
##
## Matrix products: default
## BLAS: /usr/lib/x86_64-linux-gnu/openblas-pthread/libblas.so.3
## LAPACK: /usr/lib/x86_64-linux-gnu/openblas-pthread/libopenblas-p-r0.3.20.so
##
## locale:
## [1] LC_CTYPE=en_US.UTF-8 LC_NUMERIC=C
## [3] LC_TIME=en_US.UTF-8 LC_COLLATE=en_US.UTF-8
## [5] LC_MONETARY=en_US.UTF-8 LC_MESSAGES=en_US.UTF-8
## [7] LC_PAPER=en_US.UTF-8 LC_NAME=C
## [9] LC_ADDRESS=C LC_TELEPHONE=C
## [11] LC_MEASUREMENT=en_US.UTF-8 LC_IDENTIFICATION=C
##
## attached base packages:
## [1] stats graphics grDevices utils datasets methods base
##
## other attached packages:
## [1] reporttools_1.1.3 xtable_1.8-4 dplyr_1.1.1 gridExtra_2.3
## [5] ggplot2_3.4.1 knitr_1.42
##
## loaded via a namespace (and not attached):
## [1] magrittr_2.0.3 tidyselect_1.2.0 munsell_0.5.0 viridisLite_0.4.1
## [5] colorspace_2.1-0 R6_2.5.1 rlang_1.1.0 fansi_1.0.4
## [9] highr_0.10 tools_4.2.3 grid_4.2.3 gtable_0.3.3
## [13] xfun_0.38 utf8_1.2.3 cli_3.6.1 withr_2.5.0
## [17] tibble_3.2.1 lifecycle_1.0.3 farver_2.1.1 vctrs_0.6.1
## [21] glue_1.6.2 evaluate_0.20 labeling_0.4.2 compiler_4.2.3
## [25] pillar_1.9.0 generics_0.1.3 scales_1.2.1 pkgconfig_2.0.3
```