

A call for replications of addiction research: Which studies should we replicate & what constitutes a “successful” replication?

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Robert M. Heirene ([robert.heirene@sydney.edu.au](mailto:robert.heirene@sydney.edu.au))

Brain & Mind Centre, University of Sydney, Science Faculty, 94 Mallett St, Camperdown, NSW,  
2050.

ORCID: [orcid.org/0000-0002-5508-7102](https://orcid.org/0000-0002-5508-7102)

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A call for replications of addiction research: Which studies should we replicate & what constitutes a “successful” replication?

**Abstract**

Several prominent researchers in the problem gambling field have recently called for high-quality replications of existing gambling studies. This call should be extended to the entire field of addiction research: there is a need to focus on ensuring that the understanding of addiction and related phenomena gained through the extant literature is robust and replicable. This article discusses two important questions addictions researchers should consider before proceeding with replication studies: [1] which studies should we attempt to replicate? And: [2] how should we interpret the findings of a replication study in relation to the original study? In answering these questions, a focus is placed on experimental research, though the discussion may still serve as a useful introduction to the topic of replications for addictions researchers using any methodology.

**Keywords:** replication; open science; reproducibility; addiction; methods.

## Introduction

Perhaps more than ever, scientists are spending their time replicating existing studies in addition to conducting research that is novel or exploratory. The most well-known examples of this include the Open Science Collaboration's (2015) replication of 100 psychological studies and Camerer and colleagues' (2018) replication of 21 social science experiments published in *Nature* and *Science*. The failure to replicate many of the original findings in these projects has generated concerns regarding the reproducibility of scientific research and highlighted the importance of undertaking replication studies. In the addictions field, there has been little direct recognition of this importance until recently when, in a series editorials published in *International Gambling Studies*, the journal's Editors (Blaszczynski & Gainsbury, 2019) and others in the field (LaPlante, 2019; Wohl et al., 2019) called for good quality replications of existing gambling research. This call should be extended to the entire field of addiction studies: there is a need to focus on ensuring that the understanding of addiction and related phenomena gained through the existing literature is robust and replicable. The aim of this article is to discuss two important questions addictions researchers should consider *before* proceeding on this replication crusade: [1] which studies should we attempt to replicate? And: [2] how should we interpret the findings of replication research?<sup>1</sup> In answering these questions, a focus is placed on experimental research, though the discussion may still serve as a useful introduction to the topic of replications for addictions researchers using any methodology.

### Which studies should we replicate?

Addictions researchers, like all others, are unavoidably constrained by their available resources and therefore prioritising studies for replication attempts is desirable (Coles et al., 2018).

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<sup>1</sup> A focus is placed on answering these questions and not the "how?" and "why?" of replication as these questions have received considerable attention in recent discussions (e.g., Wohl et al., 2019; Zwaan et al., 2018).

Intuitively, we might consider the studies that have greatly influenced our thinking about addiction<sup>2</sup> as most worth replicating. This approach is logical and consistent with Makel and colleagues' (2012) cautiously proposed heuristic of replicating all studies that have received  $\geq 100$  citations to prevent flawed or fraudulent findings from remaining unchallenged. Isager (2018) has found replication authors are often motivated by different types of impact, including theoretical, academic, and societal forms. Within the addictions field, theoretically impactful studies requiring replication, for example, might include those investigating nascent developments such as network theories of addictive disorders (e.g., Rhemtulla et al., 2016). The identification of so called “bridge symptoms” and symptoms with high “centrality” (see Fried et al., 2017) by such studies may help us better understand the relationship between addiction and comorbid psychopathologies and identify target symptoms for intervention.

An additional priority for addictions researchers considering replications is clinical impact. Studies evaluating novel interventions or screening procedures, for example, should be candidates for replication whenever the original studies observe promising findings. This would streamline the testing process whilst still ensuring the efficacy of the procedures before their implementation in clinical practice. In context of intervention research, direct—as opposed to conceptual—replications (see Nosek & Errington, 2017 for details of this distinction) should be prioritised to ensure the reliability of the original finding under the same protocol for administration (Lilienfeld, 2018). In reality, however the process of evaluating a novel intervention or screening method does not always follow such clearly demarcated and linear phases in which a *first* study can be easily identified. In such cases, researchers may wish to first undertake meta-analysis of the existing research to obtain an understanding of the effect, before then—if the line of enquiry appears promising and/or conflicting findings are observed—considering which of the available studies is the most plausible and desirable (based on methodological rigour) to replicate, thereby further

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<sup>2</sup> I use “addiction” here as shorthand for all addiction-related phenomena of interest, including harms, interventions, associated cognitions and so on.

contributing to the estimate of the effect. While this approach could add additional burden, researchers would contribute substantially to the understanding of addiction-related phenomena by offering a replication study and meta-analytic synthesis of the literature pre- and post-replication.

Isager (2019) provides a simple formula that can be used to quantify replication value (RV):

$$RV = \frac{\textit{impact}}{\textit{corroboration}}$$

This, Isager suggests, could be operationalised in any number of different ways to suit the needs of researchers within a given field; for example, as:

$$RV = \frac{\textit{citations}}{\textit{sample size}}$$

Addictions researchers could optimise this formula in several ways to increase its value in identifying and comparing the studies most worth replicating. First, if using the operationalisation above, using a more liberal citation count—such as that provided by Google Scholar—which includes an article’s mentions in blogs and websites may be desirable if wishing to detect articles with the widest impact. Second, if considering societal influence, researchers could attempt to further quantify this by investigating the number of times an article is referred to on social media platforms<sup>3</sup>. For example, adding an indicator of societal impact in this way raises the RV of the now famous Rat Park study (Alexander et al., 1978) from  $\frac{339}{140(n)} = 2.42$ , to  $\frac{339 + 101(\textit{mentions on Twitter \& YouTube}^4)}{140(n)} = 3.14$ . The original Rat Park study and follow ups by Alexander and colleagues (for an overview of this research line, see: Gage & Sumnall, 2019) showed reduced opiate consumption in rats living in enriched environments compared to those in standard environments. Subsequent research supports the value of environmental enrichment for

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<sup>3</sup> This could be more formally achieved using the Altmetric service: <https://www.altmetric.com>.

<sup>4</sup> The exact details of these calculations are shared on Open Science Framework: <https://osf.io/5r7a9/>

addiction recovery (e.g., Imperio et al., 2018; Pooriamehr et al., 2017), though the only direct replication of the Rat Park experiment by an external researcher did not support the original findings (Petrie, 1996). Thus, despite subsequent replications lowering the RV of the study as the total participant number has increased, the widespread attention the study [still receives online and in media](#) cannot be ignored when considering its RV. However, whether societal impact (and any other form) can be accurately quantified remains uncertain and sound scientific and clinical judgment should also be used to determine RV.

While we should aim to replicate impactful addiction studies such as the Rat Park experiments, we should also be cautious to not exclude studies reporting null findings from those deemed worthy of replication that have—as a result of their negative findings—been less influential and received fewer citations (see Fanelli, 2010). Discussions of reproducibility to date have mostly focused on the concerning number of false positives or Type-I errors that are potentially published in the addiction (Wohl et al., 2019) and scientific literature more broadly (Simmons et al., 2011). These concerns are certainly legitimate (see Ioannidis, 2005) and underpin the need to replicate statistically significant, impactful findings in the addictions field to determine whether they represent false positives. Yet convincing evidence also exists to suggest a considerable number of published articles (~66.7% in psychology) contain at least one Type-II error (Hartgerink et al., 2017). This is unsurprising: when a true effect of any size exists, there is still a 36% probability that one or more non-significant results will be observed in any two studies where power = 80% and  $\alpha = .05^5$ . Mixed results should be expected (Lakens & Etz, 2017).

It logically follows that there may exist a proportion of addiction-related studies which have asked important or influential research questions that report false negatives. Thus, it may be appropriate to ask: which studies had the *potential* to greatly influence our thinking about addiction,

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<sup>5</sup> Calculation:  $(100 - [.80 \times .80 \times 100])$ . The probability of null effects can be easily calculated for different scenarios using the Shiny app provided by Lakens & Etz, 2017: [http://shiny.ieis.tue.nl/mixed\\_results\\_likelihood/](http://shiny.ieis.tue.nl/mixed_results_likelihood/)

regardless of outcome? This distinction is only likely to become more pertinent given the increasing acceptance of publishing null findings—preliminary evidence indicates that 56-61% of preregistered and registered report studies are reporting null results, compared with ~4-20% of traditional studies (Allen & Mehler, 2019; Scheel et al., 2020).

Finally, although the emphasis of this discussion has been placed on considering a study's (potential) impact when determining its RV, several other selection criteria can be used. For example, there may be concerns regarding the suitability or rigour of the original study's methodology (Isager, 2018; Mackey, 2012), or the existing evidence for an effect in a given line of research may be weak (see Field et al., 2019). Nonetheless, these concerns may be insufficient to motivate replication efforts if the research question(s) under study have limited scope to influence our understanding of addiction. Plausibility is also likely to substantially influence decisions regarding replication, with larger and more complex studies potentially being excluded based on implausibility alone. In such cases, addiction researchers could overcome the limitations of any single research team by considering multi-lab collaborations (e.g., Klein et al., 2014).

### **How should we interpret the findings of a replication study?**

Once we decide which addiction-related studies to replicate, it is important to clearly define what criteria will be used to determine the extent to which the findings of the replication corroborate those of the original study *before* commencing data analysis<sup>6</sup>. Four broad approaches available to addiction researchers for determining this are discussed below<sup>7</sup>. However, before considering which of these approaches to use, researchers may wish gauge their confidence in the original outcomes—which may also influence the decision as to whether to replicate. For example, was the study preregistered and are data and materials shared (increasing confidence that results

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<sup>6</sup> Coles et al. (2018) recommend doing this in a pre-registered format in collaboration with the authors of the original study, wherever possible.

<sup>7</sup> As an extensive review of all approaches is beyond the scope of this article, the interested reader is referred to an annotated list of relevant articles and resources shared on this project's Open Science Framework (OSF) page: <https://osf.io/5r7a9/>

were not selectively reported or  $p$ -hacked)? Are the statistical procedures used clearly reported? Are outcomes reported accurately according to screening tools such as statcheck (Nuijten et al., 2016) and the GRIM test (Brown & Heathers, 2016)? Where data are shared, can the findings be independently reanalysed and reproduced? Asking these questions before choosing to replicate may prevent wasting time when the original finding(s) cannot be relied upon for comparison (Nuijten et al., 2018).

### ***Approach 1: $p$ -values in NHST***

The criterion for “successful” replication most consistent with common statistical thinking would be  $p < \alpha$  when using traditional Null Hypothesis Significance Testing (NHST). That is, the data are surprising if the null hypothesis is true—that no difference between groups or association exists. Using  $p$ -values from traditional NHST to determine replication success is appealing as this is likely to be the approach used by the authors of the original study to interpret their findings, yet this approach has several limitations.

First, as referred to above,  $p$ -values can be an unreliable indicator of outcome (Cumming, 2008, 2014). The average level of power achieved in psychological studies historically (Fraley & Vazire, 2014) is ~50%. Thus, if we were to use  $p < 0.05$  as the criterion for replication success in a typical addiction study where a true population effect size exists, we would only expect ~50% of studies to replicate the original finding<sup>8</sup>. Second,  $p$ -values are not comparable. A  $p$  of 0.03 in both an original and replication study do not represent similar findings in terms of the magnitude of the effect studied (Sullivan & Feinn, 2012). Indeed, in large samples one could find an effect size one tenth the magnitude of an original study and still find a similar  $p$ . Whilst we would be reluctant to label this finding as corroborating the original, using  $p$ -values as our only method of inference could permit such a conclusion. Third, and following on from the previous point, if we find a  $p$

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<sup>8</sup> Cumming (2008) has further calculated that only 12% of the variance in replication  $p$ s can be explained by the original value, with only smaller  $p$ s (i.e.,  $p < .001$ ) providing useful predictive information about future replications.



less than our specified  $\alpha$  (and are confident that this is not a Type-I error), this tells us little other than an effect is greater than 0 (Murphy et al., 2014). This is evident when samples are large enough to reach statistical significance despite negligible effects (e.g.,  $d = 0.001$ ; see Kramer et al., 2014), and is problematic for evaluating replication outcomes as researchers are often interested in whether the *magnitude* of the effect is (approximately) replicable.

### ***Approach 2: Effect sizes and interval estimates***

A common way to overcome some of the aforementioned limitations of  $p$ -values is to report effect sizes and their confidence intervals alongside, or in place of, NHST outcomes. Effect sizes inform us of the magnitude of an effect and therefore convey practical information in an intuitive format (Cumming, 2014; Sullivan & Feinn, 2012), particularly when unstandardized versions are reported (Pek & Flora, 2018). By providing an indication of where a study's finding exists along a scale of possible outcomes, effect sizes avoid a dichotomous approach to thinking about study outcomes in which an effect is either true or false depending on whether a value crosses a threshold, as with  $p$ -values (Cumming & Fidler, 2009). In relation to replication, this allows us to determine the *degree* to which findings from a replication study mirror the original (Camerer et al., 2018; Piper et al., 2019) by calculating point and interval estimates for the difference. For details on how to compare effect sizes using this method, see Zou (2007; correlation coefficients) and Pfister and Janczyk (2013; mean difference scores).

Effect sizes can also be compared in a significance testing paradigm using the  $Q$ -statistic used to study the heterogeneity of effect sizes in meta-analyses, although this approach may lack statistical power to detect meaningful levels of heterogeneity if comparing small numbers of effects (see Hedges & Schauer, 2019). Effect sizes can also be combined meta-analytically to provide an estimate of the true effect based on the outcomes from both the replication(s) and the original study/studies (Cumming, 2008); although, a consideration of the study quality, as discussed above, should inform how meta-analytic techniques are used and interpreted.

Confidence intervals for effect sizes and other parameters such as means, medians, probabilities (denoted by  $\theta$ ) provide additional information by illustrating the margin of error for the parameter ( $\theta$ ) estimate, offering a range of plausible values based on the *observed* data (Cumming & Fidler, 2009). Put simply, 95% confidence intervals will, on average and over time, contain the true population value of  $\theta$  95% of the time (Morey et al., 2016)<sup>9</sup>. Seeing whether a replication study's  $\theta$  fits within the confidence interval boundaries of the original study has been used as an indicator of replication success (Camerer et al., 2018; Open Science Collaboration, 2015). However, in replication terms, confidence intervals are not indicators that 95% of future  $\theta$ s will fall within the original interval boundaries<sup>10</sup>. Using simulated data, Cumming and Maillardet, (2006) have calculated that the capture percentage for 95% confidence intervals (i.e., the % of replication  $\theta$ s which lay within the original  $\theta$ 's confidence intervals) around sample means is approximately 83.4%; although, replication studies were assumed to have the same sample size as the original. Spence and Stanley (2016) have calculated the capture percentage of 95% confidence intervals for different parameter estimates by using data simulations and randomly selecting subsamples of varying sizes for both an original and replication study. The capture percentage ranged between 33 and 95% for sample mean confidence intervals, 34 and 95% for  $d$  value confidence intervals ( $d$ s of 0.2, 0.5, & 0.8), and 44 and 94% for correlation coefficient confidence intervals ( $r$ s of 0.1, 0.3, & 0.5). These findings caution against using the confidence intervals around the  $\theta$  from the original or replication study to interpret the outcomes of replication research. Using confidence intervals formed around the point difference between parameter estimates may be more informative for this purpose, as discussed above. Overall, the predictive value of any individual parameter estimate and its confidence interval will depend on how well it reflects the true population value (which increases with sample size and reliable measurement).

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<sup>9</sup> See Morey et al. (2016) for an important discussion of the fallacies that surround confidence intervals.

<sup>10</sup> This is because the original parameter estimate and its confidence intervals may be a poor indication of the true population value, particularly if the sample size(s) studied were small.

While using effect sizes and their confidence intervals to interpret replication outcomes may be more informative than  $p$ -values, there are some important considerations and limitations associated with their use in this context. Effect sizes in the existing literature appear to be inflated estimates. Camerer et al. (2018) found successful replication studies (as determined by  $p < .05$ ) produced effect sizes 74.5% the size of the original, whilst the Open Science Collaboration (2015) found the average effect size across 99 replications (successful & unsuccessful) was half (48.9%) that of the originals. Publication and reporting biases are thought to explain the inflation of effect sizes (Open Science Collaboration, 2015), and thus it may be wise to anticipate smaller effects in replication studies and to consider how these biases could lead to over-estimating effects in meta-analysis (see Kvarven et al., 2019).

It is also important to consider how sampling variation, contextual factors (e.g., experimenters, location, date), measurement error, and hidden moderators may have influenced the effect size of a study (Stanley & Spence, 2014); as well as how these factors and deviations from the original design may influence a replication's effect size. There is increasing recognition that heterogeneity of effect sizes in the published literature is not only large, but larger than would be expected with sampling variation alone (Stanley et al., 2018); particularly when larger and more consistent effects are studied (Klein et al., 2018). Based on simulated research scenarios, Kenny and Judd (2019) have found when heterogeneity of effect sizes exists, multiple smaller  $N$  studies ( $N = 100 \times 5$ ) may produce more precise estimates (i.e., narrower CIs) of an effect than one large  $N$  study ( $N = 500$ ) even when only moderate power (69%) is achieved; though the importance of achieving sufficient sample sizes in replications should not be downplayed (Maxwell et al., 2015; Piper et al., 2019). Nonetheless, addiction researchers planning replications may want to consider the possibility of running multiple, multi-site studies to provide the most precise estimate of an effect and thereby test the robustness of the effect to minor variations in protocol and setting. Indeed, evidence of significant heterogeneity in effects has led to concerns regarding the utility of

any single replication study<sup>11</sup> (Kenny & Judd, 2019; Maxwell et al., 2015) and the proposal that researchers conducting replications should not be focused on *verifying* an existing finding (particularly when the original sample size was small and unlikely to provide an accurate estimate), but rather on contributing to the overall *estimation* of the true underlying effect (Stanley & Spence, 2014). Thus, from this perspective the terms “successful” and “unsuccessful” replication are misleading (Gelman, 2018).

One approach to accounting for *some* of the heterogeneity in study outcomes when interpreting replication findings is to use the prediction interval method propounded by Patil et al. (2016). Prediction intervals have similar, although not identical, properties to confidence intervals—given the original  $\theta$ , a prediction interval can be calculated that is such that 95% of exact replications will produce  $\theta$ s that fall within the interval boundaries. Calculations for prediction intervals incorporate variation in both the original study and replication. For example, for prediction intervals around  $r$ , the following equation is used:

$$r_{orig} \pm Z_{0.975} \sqrt{\frac{1}{n_{orig}-3} + \frac{1}{n_{rep}-3}}$$

Additional information and calculations are provided by Patil and colleagues (2016). A similar approach, also involving the calculations of “prediction intervals” (but using different calculations that estimate the variance of the sampling distribution for the original study), is offered by Spence and Stanley (2016). Using data simulations, Spence and Stanley have found the capture percentage for prediction intervals around sample means,  $d$  values, and correlation coefficients all ranged between 94.3 and 95.2% (contrast with capture percentage values for confidence intervals discussed above). Prediction intervals can be specified prior to undertaking the replication study

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<sup>11</sup> This is not to say that addictions researchers should not conduct single replication studies, but rather that replication outcomes—like those of the original study—can be subject to a variety of influences (e.g., questionable research practices, biases [conscious and unconscious], contextual factors, statistical power, sampling variation etc.). Each study is valuable in the same way that individual pieces contribute to a puzzle—giving some but not complete insight into what the overall picture looks like. The more pieces (studies), the clearer the overall picture (the true effect).

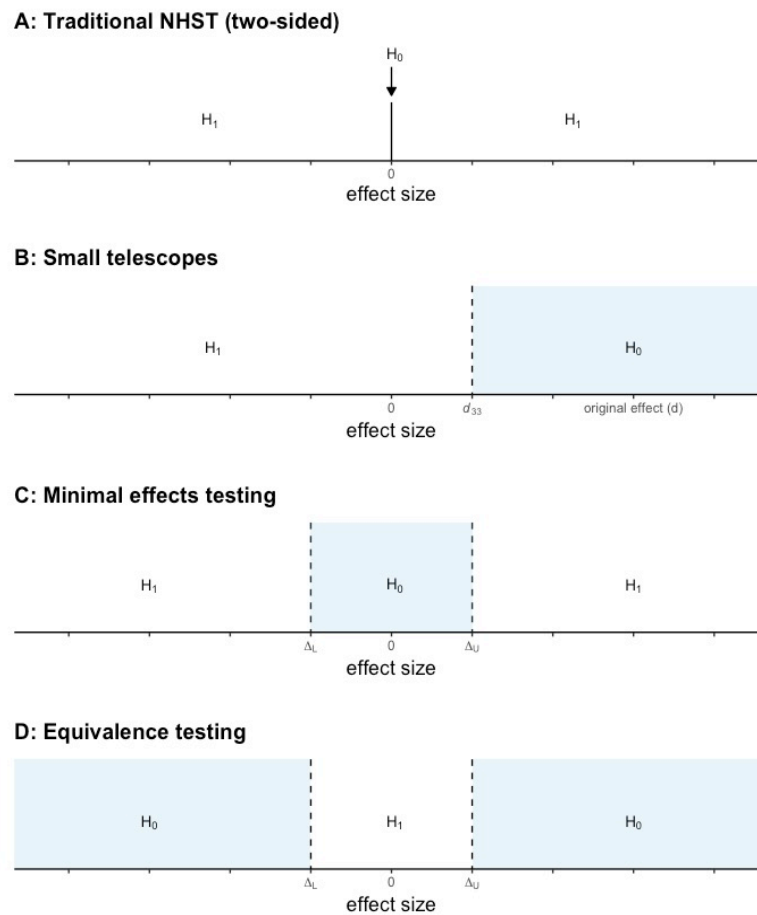
(provided  $N$  is known in advance), avoiding post hoc biases affecting interpretation, and Spence and Stanley (2016) provide open source software and R code for calculating prediction intervals for means, correlations, and Cohen's  $d$ . Using prediction intervals (via either computation method) to determine whether a replication's findings are consistent with the original can overcome the criticism of using confidence intervals for this purpose, namely that they do not account for sampling error (Kenny & Judd, 2019).

### ***Approach 3: Combining $p$ -values and effect sizes***

Significance testing may be more useful for interpreting replication outcomes when used in reference to a specific effect size. This approach encompasses several different methods including small telescopes, minimum effects testing, and equivalence testing. Each of these involve, in varying ways, testing against a Smallest Effect Size of Interest (SESOI). Testing against a SESOI may always be preferential over testing against an effect of zero (i.e.,  $H_0$  in traditional NHST) as any effect under study is highly unlikely to be exactly zero given random variation. The corollary of this is that one can theoretically always find support for  $H_1$  with sufficiently large sample sizes (Murphy et al., 2014), rendering significant outcomes in such circumstances uninformative. Thus, testing against a SESOI better enables falsifiability.

***Small telescopes:*** Proposed by Simonsohn (2015), the small telescopes approach labels a replication as a failure if it rejects an effect that the original study was 33% powered to detect ( $d_{33\%}$ ). For example, if a study has a sample size of 50 participants for a paired-sample  $t$ -test, it has 33% power (or roughly a 1:2 chance) to detect an effect of  $d_{33\%} = 0.22$ . If a replication study's effect is statistically smaller than  $d_{33\%}$ , then we can conclude the original evidence to suggest a theoretically interesting effect existed is not convincing and was likely a false positive,  $p$ -hacked, or fraudulent (Simonsohn, 2015). To use the small telescopes approach, a typical one-sided significance test is used with the null hypothesis ( $H_0$ ) equivalent to  $\geq d_{33\%}$  and an alternative hypothesis ( $H_1$ ) that  $d < d_{33\%}$  (see Figure 1). One limitation of the Small telescopes approach is that it requires a sample size

roughly 2.5 times the original to detect  $d_{33\%}$  and thus may not be applicable when the original study used a large sample. However, as Simonsohn notes, when the sample size of an original study was large, we may be less interested in knowing whether we could achieve  $d_{33\%}$  as this could be negligibly small in such circumstances (e.g.,  $d_{33\%}$  for an independent  $t$ -test [ $N = 4,000$ ] = 0.049).



**Figure 1.** Different approaches to statistical testing: A) Traditional NHST, B) Small telescopes, C) Minimal effects testing, and D) Equivalence testing<sup>12</sup>.  $\Delta_L$  and  $\Delta_U$  are lower and upper equivalence boundaries, respectively.

**Minimum effects testing:** Using this method, the SESOI is a range that becomes  $H_0$  (e.g.,  $d = -.05 - .05$ ), and effects larger than the SESOI range in either direction (e.g.,  $d < -.05$  or  $> .05$ ) are set as  $H_1$  (see Figure 1). Thus, we test whether an effect lays statistically within ( $H_0$ ) or outside

<sup>12</sup> The R code provided by Lakens et al. (2018) was adapted (with permissions) to create this figure. The exact code used to create Figure 1 is shared on Open Science Framework: <https://osf.io/5r7a9/>

( $H_1$ ) a range of effects that would be so small so as to be of no interest if they were found, regardless of whether they reached significance using traditional NHST (Murphy et al., 2014). In replication terms, this option enables researchers to select a SESOI that, if achieved, would represent the minimal level of evidence required to support the original study. Addiction researchers should determine their SESOI based on a consideration of the theoretical or clinical importance of different effect sizes (Lakens, Scheel, & Isager, 2018).

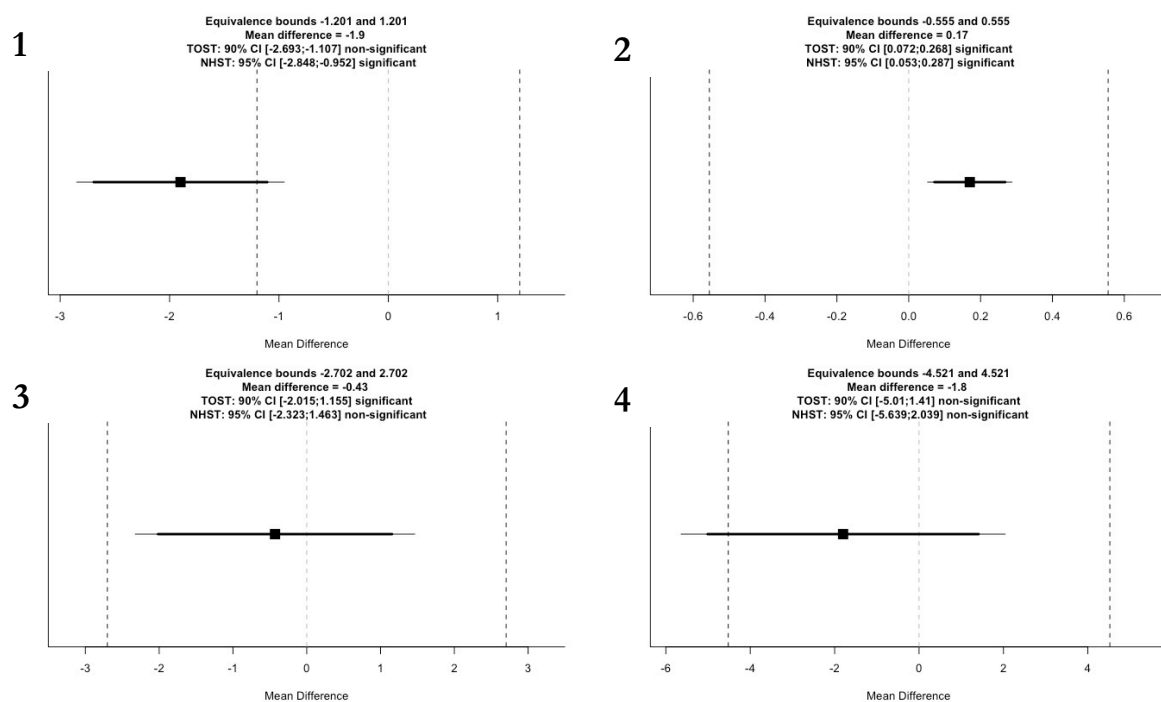
***Equivalence testing:*** This approach can be viewed as the inverse of minimum effects testing. It involves testing whether an effect is statistically within upper and lower equivalence boundaries that represent the SESOI (Lakens et al., 2018). In equivalence testing,  $H_0$  represents a meaningful effect (i.e., larger than the SESOI range in either direction), while effects within the boundaries of the SESOI support  $H_1$  (see Figure 1). Choosing between minimal effects and equivalence testing may depend on whether one wishes to place the burden of proof on replication (when  $H_1$  is supported, findings support the original study) or non-replication (when  $H_1$  is supported, findings do not support the original study; see Hedges & Schauer, 2019). Lakens and colleagues (2018) have developed the 'TOST' (two one-sided significance tests) which can allow addiction researchers to easily include equivalence testing in their studies<sup>13</sup> ([available for R, jamovi, excel](#)).

Lakens et al. (2018) recommend combining equivalence testing with traditional NHST, resulting in four possible outcomes (see corresponding panels in Figure 2): [1] clear evidence of effect: not statistically equivalent and significantly different from zero (i.e.,  $H_0$  in NHST), [2] the effect is smaller than the SESOI, though is greater than zero: statistically equivalent and significantly different, [3] no evidence of effect: statistically equivalent and not statistically different [4] the SESOI cannot be rejected, but the effect is not significant using NHST: not statistically equivalent and not significantly different. Figure 2 plots the findings from a reanalysis of the outcomes of three randomised control trials published in addiction journals. A medium effect size

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<sup>13</sup> The TOST can also be adapted for use by those wanting to employ the small telescopes approach.

$(d = 0.5)^{14}$  was used for upper and lower boundaries for ease of illustration, though addictions researchers should give proper consideration to the equivalence boundaries that would be appropriate for their research (see Anvari & Lakens, 2019). Each of the outcomes presented in Figure 2 is more informative than using NHST alone, and outcome 4 may be of particular interest in the context of replication. This outcome indicates that, although not statistically significant using NHST, the effect *may* be equal to or larger than the SESOI—which in this case is a noteworthy  $0.5(d)$ —and therefore the original study may have lacked sufficient power to detect a meaningful effect. Going forward, addictions researchers can use the TOST, as done here, to determine whether other null findings reported in their subfields may require further investigation (e.g., Quintana, 2018).



**Figure 2.** Examples of all possible equivalence testing outcomes from Randomised Control Trials (RCTs) published in *Addiction* (panels 1, 3, & 4) & a recent study published in *International Journal of*

<sup>14</sup> Cohen’s  $d$  effect size was used to set boundaries here (hence the difference in scales along the x-axes in the plots), though raw difference scores can also be used for this purpose.



Mental health & Addiction (panel 2; no example of this outcome could be identified in RCTs published in addiction journals)

[1] Lintzeris et al. (2002), comparison of self-reported heroin use after 8 days between intervention (buprenorphine) & control groups; [2] Bener et al. (2019), difference between students with internet addiction disorder vs. "normal" students on question 4 of the 14-item Fatigue Scale ("Do you have problems starting things?"); [3] Petrakis et al. (2017), comparison of mean no. of drinks per day during 30-day follow-up between intervention (Mecamylamine) & placebo groups; [4] Grønbaek & Nielsen (2007), drinks per day comparison between Minnesota day clinic patients vs. standard public psychotherapy treatment patients in the 30-days post intervention.

Maxwell et al. (2015) contend that equivalence testing can be used to determine a range of values that, if a  $\theta$  and its CI were observed within (they propose ranges for  $d$  of -0.10 to 0.10 or -0.05 to 0.05), would support the conclusion that the null hypothesis is "*for all intents and purposes essentially true*". Thus, if the range of parameter values observed in a replication study is within the equivalence boundaries, then strong evidence for the absence of an effect has been found and any effect(s) observed in previous investigations should be questioned. However, to classify an effect as equivalent to null based on such small ranges would require inordinately large sample sizes for addiction research. For example, to achieve 80% power for a between groups  $t$ -test using equivalence boundaries of -0.10 and 0.10 ( $d$ ) and assuming the true effect is 0, a total of 3,426 participants would be required (rising to 13,704 participants with boundaries of -0.05–0.05). The solution to this issue as recommended by Maxwell and colleagues (2015) echoes a recommendation made earlier in this article: addiction researchers must work collaboratively, undertaking multiple replication studies across multiple labs. Only through such collaborations can we achieve the necessary sample sizes required to provide convincing evidence for the absence of an effect.

#### ***Approach 4: Bayesian statistics***

A fourth approach is the use of Bayesian statistics and the calculation of Bayes Factors (BFs). BFs quantify the relative evidence for  $H_0$ , and  $H_1$  using average likelihood ratios and therefore may be favoured over  $p$ -values. BFs also incorporate researchers' prior beliefs or predictions about the effect under study (based on theory or existing evidence) which is included into the statistical

model as a *prior*, or more accurately the *prior* distribution of the parameter (e.g., one's prior belief about the distribution of coin flips landing on heads is likely to be tightly centred around a chance value of 0.5). Once the data are observed, one's degree of belief in the theory is then updated accordingly into a *posterior belief* in the form of the resulting BF; thus, BFs can be viewed as the degree to which the observed data require an update to prior beliefs about the relative plausibility of the models specified by our hypotheses ( $H_0$  &  $H_1$ ). According to convention, BFs  $>3$  represent support for  $H_1$  over  $H_0$ , BFs  $<0.333$  (or  $1/3$ ) represent support for  $H_0$  over  $H_1$ , and BFs between 0.333 and 3 reflect insufficient evidence for either hypothesis. While using a Bayesian approach to statistical inference was once seen as a complicated and unusual approach, such methods are becoming increasingly more common (e.g., Pisklak et al., 2019) and user-friendly software (e.g., JASP) has been developed that can now easily calculate BFs. Indeed, the journal *Addiction* now recommends all authors report BFs in studies reporting null findings, with the suggestion that only BFs  $< 0.3$  should be interpreted as representing no evidence of effect (i.e., support for  $H_0$  over  $H_1$ ).

Bayesian methods and BFs possess a number of properties that make them useful for interpreting replication outcomes (Dienes, 2014, 2016). Unlike traditional NHST, researchers can clearly specify  $H_0$  and  $H_1$ , potentially specifying  $H_1$  as the original study's (or meta-analysis) finding for comparison. Such an approach has been advocated by Verhagen and Wagenmakers (2014) and then updated by Ly et al. (2018), who propose evaluating replication outcomes from the perspective of both a sceptic (i.e., no effect exists) and proponent (i.e., the original study's finding reflects the true effect). The relevant evidence for the sceptic's perspective ( $H_0$ ) over the proponent's ( $H_1$ ) can then be calculated.

In addition to model comparisons, Bayesian statistics can also be used for estimating parameters and their variance in a similar way to frequentist approaches (Kruschke, 2011). The 95% *credible* interval calculated for parameters in Bayesian statistics contain 95% of estimated

parameter values that are most plausible based on the observed data. In an approach analogous to equivalence testing in frequentist statistics, researchers using Bayesian methods can identify a Region of Practical Equivalence (ROPE) that contains the range of values for  $\theta$  we consider to be equivalent to null (Kruschke, 2011) and observe whether the credible interval within, outside, or partially within the ROPE. This approach therefore allows researcher to consider the *proportion* of  $\theta$  values equivalent to null as well as making categorical interpretations, and again can provide convincing evidence for the absence (entire interval falls within the ROPE) or presence (entire interval falls outside of the ROPE) of an effect in the context of replication research.

### **Final considerations and conclusions**

There is a need for addiction researchers to conduct replication studies to ensure the veracity of our understanding surrounding addiction and related phenomena. Historically, carrying out replications has not been incentivised by academic and scientific systems due the lack of “novelty” associated with such studies (Romero, 2018)<sup>15</sup>. However, a sea change appears to be occurring. Conceptual papers and meta-science studies have alerted us to the risks of observing statistical errors within any single study, highlighting the epistemic importance of replication studies (Cumming, 2014; Hartgerink et al., 2017; Ioannidis, 2005). Large scale, collaborative replication efforts have been, and are currently being, undertaken (Klein et al., 2018; Open Science Collaboration, 2015). Numerous scientific articles, many of them summarised here, have been published in recent years on the manifold considerations researchers must make when replicating (Wohl et al., 2019; Zwaan et al., 2018). With the advent of registered reports, researchers can now gain provisional approval from journals that their replication study will be published—subject to peer review of the introduction section and proposed methods—before they start data collection (Chambers, 2019), allaying concerns that their study may lack the “novelty” required for

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<sup>15</sup> For an excellent discussion of “who should replicate” how we can align incentives to encourage replication studies, see Romero (2018).

publication if it corroborates the original finding. Still, replicating studies can be a time-consuming endeavour and addictions researchers should carefully select target studies, taking into consideration their potential impact from theoretical, academic, societal, and clinical viewpoints.

Thorough consideration should be given to how the results of replication studies will be interpreted and compared with the original finding. A variety of approaches are available for this purpose, including meta-analysis, interval predictions, small telescopes, equivalence testing, and Bayesian methods. In line with the Open Science Collaboration's (2015) view that "*No single indicator sufficiently describes replication success*", using several of these methods together will allow for the most comprehensive interpretation of findings. In deciding which to use, addictions researchers will need to consider the feasibility of each (e.g., can the level of power required to use the small telescopes method be achieved?), the aims and purpose of their study (e.g., is the intention to determine whether an effect exists via direct replication? Or to contribute to the overall estimation of a well-established effect via conceptual replication?<sup>16</sup>), and their confidence in the outcomes of the original work (e.g., will meta-analysis be misleading if the original study's/studies' findings are under question?). This must be done prior to data collection, and the specific statistical approaches and inference criteria selected should be preregistered, including details of how researchers will deal with potential contingencies (e.g., what will the researchers conclude if their outcome is consistent with the original study's finding using one method but not another, say the effect lays within an interval prediction but using the Bayesian ROPE methods suggests the effect is equivalent to null?). Finally, when interpreting the findings of replications it is essential that addiction researchers recognise that multiple sources of variation can affect each study's outcome and therefore: [1] exact replication of an effect is unrealistic, and [2] one "failed" replication should not be cause to declare the original effect does not exist—the addictions field

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<sup>16</sup> These simply reflect hypothetical questions one may ask—determining whether an effect exists and estimating an effect are not the exclusive goals of direct and conceptual replication studies, respectively.

must adopt meta-scientific approach wherein the accumulation of evidence is prized and single findings are interpreted cautiously.

### **Supplemental materials**

Supplemental materials, including an annotated list of relevant articles and resources relating to the interpretation of replication outcomes, the R code used to produce the figures presented here and, and the code for calculating effect sizes when using the Small telescopes approach (i.e.  $d_{33\%}$ ) can be accessed via this project's Open Science Framework Project Page: <https://osf.io/5r7a9/>

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